ME/CFS and Retroviruses

In October 2009, a team of researchers from the Whittemore Peterson Institute (WPI) published in the prestigious journal *Science* a paper linking ME/CFS with the retrovirus XMRV. The article stated that the blood of 67% of a sample of 101 ME/CFS patients tested positive for the virus, compared to only 3.7% of a control sample.

XMRV, a murine leukemia virus (MLV), was the third retrovirus that had been found to be associated with human disease. The others, HIV and HTLV, were known to cause complex lifelong systemic health disorders. While an association previously had been found between XMRV and prostate cancer, its being linked with a complex neuro-immune disease suggested that it was especially worthy of study.

Thus, the publication generated a great deal of interest amongst virologists and others in the scientific community, as well as substantial media coverage. ME/CFS patients and others interested in the disease were especially excited, both because of the attention that the finding brought to the disease and because of the hope that it might lead to effective treatment.

The idea that a retrovirus might be at the heart of ME/CFS is one that had been considered since the mid-1980s, when the illness first emerged in the U.S. As with AIDS, ME/CFS patients were observed to have immune systems unable to control a wide variety of infections that normal people keep in check. Although one research finding in the early 1990s of a possible retroviral link was eventually dismissed, many people interested in the disease continued to believe that an HIV-like pathogen eventually would be found.

Subsequent to the publication of this paper, millions of dollars were spent following up on its findings. Eventually, the results were determined to have been a laboratory contaminant and the original WPI paper was retracted.

At present, there are no papers linking ME or CFS with a retrovirus currently in the medical literature.

-Lisa Petrison, Ph.D.
ME/CFS AND RETROVIRUSES

MEDICAL LITERATURE, 2009-2012 (XMRV and MLV’s):


The original investigators who found XMRV and pMLV (polytropic murine leukemia virus) in blood of subjects with CFS report that this association is not confirmed in a blinded analysis of samples from rigorously characterized subjects.


Science is fully retracting the report "Detection of an infectious retrovirus, XMRV, in blood cells of patients with chronic fatigue syndrome" (V. C. Lombardi et al., Science 326, 585 (2009)).


A paper showing an association between a retrovirus and CFS is retracted.


The authors failed to find XMRV or other MLV’s in a group of Canadian patients.


This group recently showed that XMRV was created through recombination between two endogenous murine retroviruses, PreXMRV-1 and PreXMRV-2, during the passaging of a prostate tumor xenograft in nude mice. Here, multiple approaches that led to the identification of PreXMRV-2, as well as the distribution of both parental proviruses among different mouse species are described.


The authors failed to find XMRV or related retroviruses in a population of Swedish CFS patients.


The authors discuss findings related to XMRV and CFS.


Coded replicate samples of blood from 15 subjects previously reported to be XMRV/MLV-positive (14 with CFS) and from 15 healthy donors previously determined to be negative for the viruses were distributed in a blinded fashion to nine laboratories, which performed assays designed to detect XMRV/MLV nucleic acid, virus replication, and antibody. Only two laboratories reported evidence of XMRV/MLVs; however, replicate sample results showed disagreement, and reactivity was similar among CFS subjects and negative controls. These results indicate that current assays do not reproducibly detect XMRV/MLV in blood samples.


In our 23 October 2009 Report, "Detection of an infectious retrovirus, XMRV, in blood cells of patients with chronic fatigue syndrome," two of the coauthors, Silverman and Das Gupta, analyzed DNA samples from chronic fatigue syndrome (CFS) patients and healthy controls. A reexamination by Silverman and Das Gupta of the samples they used shows that some of the CFS peripheral blood mononuclear cell (PBMC) DNA preparations are contaminated with XMRV plasmid DNA.


The authors found no definitive evidence for XMRV DNA sequences or antibody in a cohort of CFS patients, which included patients from diverse regions of the United States. In addition, XMRV was not detected in a cohort of patients with chronic inflammatory disorders.

The authors failed to find XMRV in a group of CFS patients from Quebec.


A study looking for XMRV in CFS patients and unaffected identical twins did not find the virus in blood samples from any of the participants.


This authors suggested that XMRV has clear ancestors in mice and described a possible source of laboratory contamination for the virus.


The authors looked at primers that might be used to attempt to detect XMRV and suggest that those reagents may be contaminated with the virus.

gammaretroviruses in CFS patients previously identified as XMRV-infected. Science. 2011 Jul 1;333(6038):94-7. PMID: 21628393

The authors failed to find XMRV or other MLVs in blood of a sample of 61 patients with CFS. MLV sequences were detected in commercial laboratory reagents.


The authors found genetic sequences very similar to those in XMRV in specific mice, and speculate that this was the source of laboratory contamination with the virus.


The editors of Science express their belief that the association between XMRV and CFS “likely reflects contamination of laboratories and research reagents with the virus.”


CFS patients testing positive for XMRV display a specific pattern of cytokines and chemokines.

The authors failed to find XMRV or related MLVs in the blood of 100 CFS patients and 200 healthy volunteers from the Salt Lake City, Utah, area.


The authors vaccinated mice with XMRV, observing transiently high levels of antibodies.


The authors failed to find XMRV or other common viruses in the cerebrospinal fluid of 43 CFS patients.


The authors failed to find “solid evidence” of XMRV sequences or antibodies in any of the blood samples from 500 healthy donors, 67 prostate cancer patients or 100 CFS patients.

The authors failed to find XMRV in a population of CFS patients in the UK, using techniques that they say were similar to those used in the original Science study showing an association.

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The authors report that 2 of 14 patient-derived XMRV integration sites are identical to ones from experimentally infected cells, and they thus conclude that XMRV is a contamination rather than a genuine human pathogen.

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The authors failed to find XMRV or MuLV in any blood samples of a group of 45 CFS sufferers demonstrating a high degree of disability from across the U.S. and 42 matched controls.

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The authors inoculated five macaque monkeys with XMRV intravenously. The virus established a persistent, chronic disseminated infection, with low transient viremia and provirus in blood lymphocytes during acute infection. Although undetectable in blood after about a month, XMRV viremia was reactivated at 9 months, confirming the chronicity of the infection. Viral sequences were disseminated through the body,
infecting CD4 T cells in lymphoid organs including the gastrointestinal lamina propria, alveolar macrophages in lung, and epithelial/interstitial cells in other organs, including the reproductive tract.


The authors studied how XMRV might spread in the body, reporting that human peripheral blood mononuclear cells (PBMCs) can potentially act as a source of infectious XMRV for spread to cells that express low levels of host restriction factors. They state that hypermutation of XMRV in human PBMCs constitutes one of the blocks to replication and spread of XMRV and that hypermutation of XMRV proviruses at GG dinucleotides may be a useful and reliable indicator of human PBMC infection.


The authors report that NF-kB activation can markedly increase XMRV production, and suggest that EBV infection or other sources of inflammation may promote XMRV spread in humans.


The authors failed to find XMRV sequences or XMRV specific antibodies in blood samples of 39 CFS patients, 112 MS patients or 40 healthy donors, all from Germany. The authors were able to experimentally infect samples from healthy donors and CFS patients with XMRV, resulting in the release of low levels of transmittable virus.

The WPI group that initially found XMRV in the blood of CFS patients details the techniques that they used to detect it, suggesting that more than one type of assay be used.


The authors of Retrovirology counsel the need to take extreme care to make sure that laboratory contamination is not responsible for detection of XMRV in human samples.


The authors demonstrate that primers used as laboratory agents can be contaminated and express concern that patient sequences of XMRV are not more diverse, thus proposing that XMRV might not be a genuine human pathogen.

Sato E, Furuta RA, Miyazawa T. An endogenous murine leukemia viral genome contaminant in a commercial RT-PCR kit is amplified using standard primers for XMRV. Retrovirology. 2010 Dec 20;7:110. PMID: 21171978

The authors found evidence of contamination of endogenous MLV and XMRV genomes in laboratory test kits.

The authors tested blood of 112 CFS patients and 36 healthy controls for XMRV, finding it only when other evidence of contamination was present.


The authors summarize what has been learned about XMRV in human illness.


The authors failed to find XMRV in blood samples of 32 CFS patients, 43 HIV patients, 97 rheumatoid arthritis patients, 26 hematopoietic stem-cell or solid organ transplant patients, or 96 patients presenting for various types of medical care.


The authors screened rodents and other mammals for sequence variation in the Xpr1 receptor for the mouse xenotropic or polytropic mouse leukemia viruses (X-MLVs or P-MLVs, respectively) of the gammaretrovirus family and for susceptibility to mouse-
derived X/P-MLVs and to XMRV, identifying multiple distinct susceptibility of phenotypes.


The authors of the original paper Science finding an association between XMRV and CFS discuss issues in laboratory techniques used to attempt to find the virus.


Information and hypotheses about XMRV and its possible role in CFS are summarized.


The authors failed to find XMRV in peripheral blood mononuclear cells (PBMCs) and plasma of Chinese patients with CFS.


The findings of sequences related to XMRV in CFS patients and reported in the Lo et al 2010 paper are discussed.

Commentary on Lo et al 2010, from the same publication issue.

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The authors found murine leukemia virus (MLV) like virus sequences in 32 of 37 (86.5%) CFS patient samples compared with only 3 of 44 (6.8%) of samples from healthy volunteer blood donors. No evidence of mouse DNA contamination was detected in the PCR assay system or the clinical samples. Seven of 8 gag-positive patients tested again positive in a sample obtained nearly 15 y later. A genetically diverse group of MLV-related viruses were found, with sequences from CFS patients more closely related to those of polytropic mouse endogenous retroviruses than to those of XMRVs.

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The authors summarize what is known about XMRV thus far.

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The authors infect three rhesus macaque monkeys with XMRV to determine dynamics of the antibody responses.

The authors study the Apobec3 (A3) proteins, a family of cytidine deaminases that serve as one important group of host proteins to control primary infection and efficient viral spread of XMRV.


The authors discuss the importance of finding XMRV in CFS patients.


Current knowledge about the role of XMRV in CFS is discussed.


The authors failed to find XMRV using multiple molecular or serologic assays in archived blood samples of 51 people with CFS and 56 healthy people from populations in Kansas and Georgia.

The authors discuss what is known about XMRV in CFS and prostate cancer.


The authors detected XMRV-specific sequences in 2%-3% of samples from 168 immunocompetent carriers and approximately 10% of samples from 161 immunocompromised patients.


The authors discuss what is known about the role of XMRV in prostate cancer and CFS.


The authors studied the fidelity of XMRV integration DNA into cells, concluding that it involves a coordinating joining of two viral DNA ends space 4 bp apart on the target DNA and that it proceeds with high fidelity.

The article discusses the risk that vaccines may be contaminated with retroviruses such as XMRV.


The authors evaluated 45 different drugs to see if any were capable of inhibiting XMRV replication. Those that were effective included the retroviral integrase inhibitor raltegravir; an integrase inhibitor, L-000870812; and two nucleoside reverse transcriptase inhibitors, zidovudine (ZDV) and tenofovir disoproxil fumarate (TDF). Combinations of the substances displayed particular effectiveness.


The establishment of XMRV infection in patients may be dependent on infection of A3G/A3F-deficient cells, and cells expressing low levels of A3G/A3F, such as prostate cancer cells, may be ideal producers of infectious XMRV. Furthermore, the anti-HIV-1 drugs AZT, tenofovir, and raltegravir may be useful for treatment of XMRV infection.


The authors look at blood cell restriction factors that might have the capability of preventing replication of XMRV. They found that human APOBEC3 and tetherin proteins are able to block XMRV replication. Expression of human TRIM5alpha had no effect on viral infectivity. The virus was inhibited by factors from nonhuman species, including mouse Apobec3, tetherin, and Fv1 proteins.

The authors failed to find XMRV in the blood of 76 CFS patients or 69 matched controls from the Netherlands.


The authors failed to find XMRV DNA in a sample of 170 CFS patients and 295 controls from the UK.


The authors failed to find XMRV or MLV sequences in the blood of a group of 186 CFS patients in the UK.


The authors look at how XMRV might spread, showing that it can infect various human cell types.

The authors found DNA from XMRV in the blood of 67% of a sample of 101 CFS patients vs. 3.7% of healthy controls. Cell culture experiments suggested that the virus is infectious.

MEDICAL LITERATURE, 1991-2000


The endogenous retrovirus p15E does not seem to be associated with CFS.


Authors tested human T-lymphotropic viruses types I and II; human spuma retrovirus; simian T-lymphotropic virus type I; simian retroviruses types 1, 2, and 3; bovine leukemia virus; feline leukemia virus; and gibbon ape leukemia virus on CFS patients’ blood and concluded that none of them was playing a role in the disease.

The HTLV-II gag gene sequence was not a marker for CFS in a study of well-defined patients.


The authors did not find a variety of retroviruses (HTLV-II, simian T cell leukaemia virus, human spumavirus, bovine leukaemia virus and simian retrovirus) to be associated with CFS.


HTLV-II was not found in a group of CFS patients in Japan.


Researchers were not able to distinguish CFS from healthy controls with three retroviral tests.


Researchers did not find evidence of HTLV-II in a group of CFS patients.

The authors found an association between an HTLV-II like virus and CFS.
ME/CFS AND RETROVIRUSES

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PNAS Paper on Virus-Chronic Fatigue Syndrome Link Has Its Own Story  
By Amy Dockser Marcus  
  

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FDA Advisory Committee to Hear About XMRV Working Group’s Research
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CDC Team’s XMRV-Chronic Fatigue Syndrome Paper Is Out
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By Amy Dockser Marcus


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The AABB Makes It Official: CFS Patients Shouldn’t Give Blood
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Chronic Fatigue Sufferers May Be Asked To Avoid Donating Blood
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By Lawrence K. Altman


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