Environmental Causes of Autism

May 23, 2014

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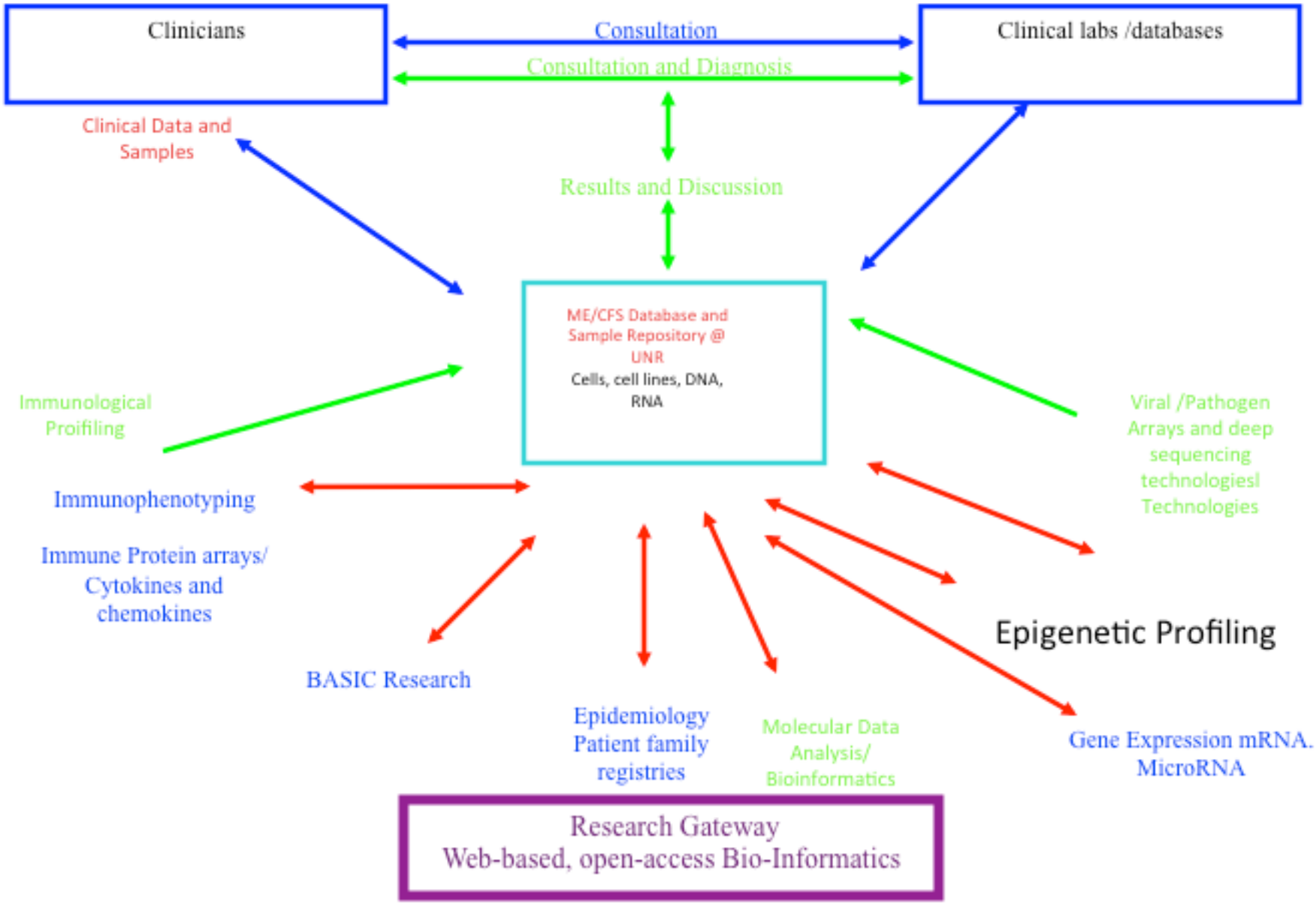
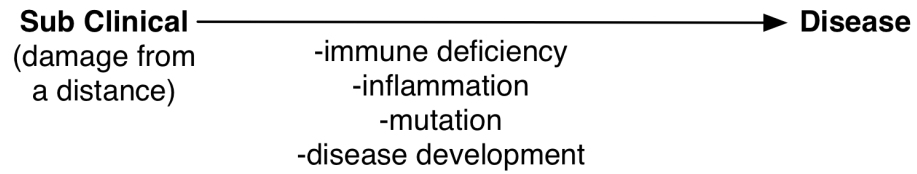
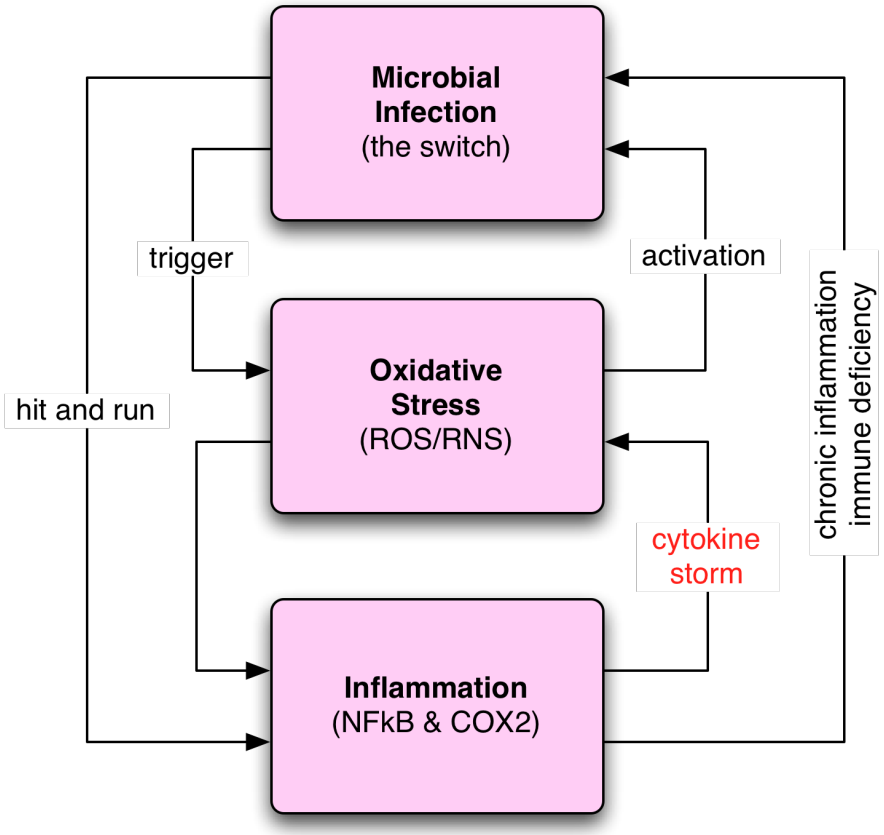
Here we are not afraid to follow the truth wherever it may lead,
Nor tolerate error so long as freedom is left to combat it
Thomas Jefferson

Systems Biology Approach to Chronic Disease.. 2007



Key Contributors to Chronic Diseases



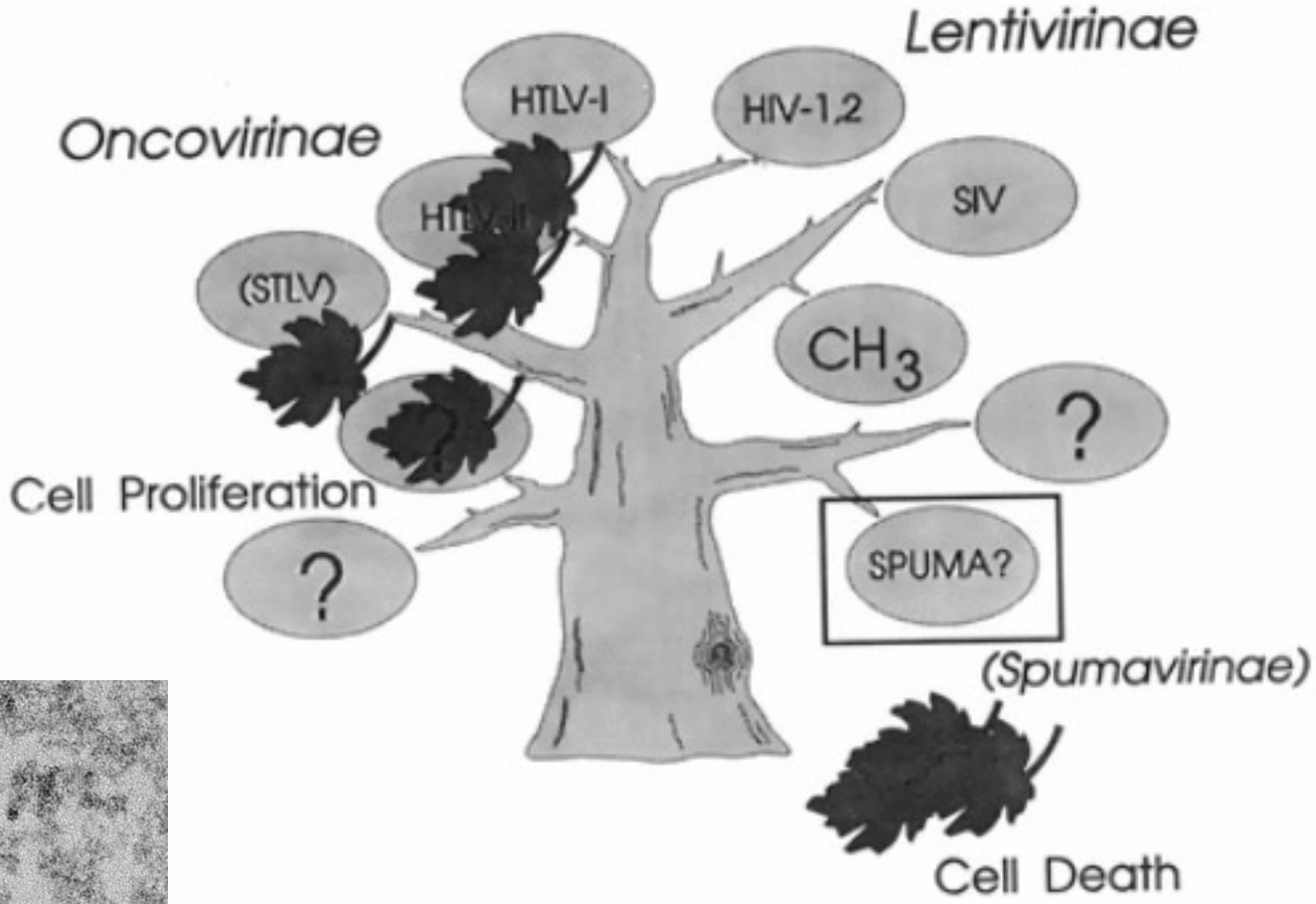


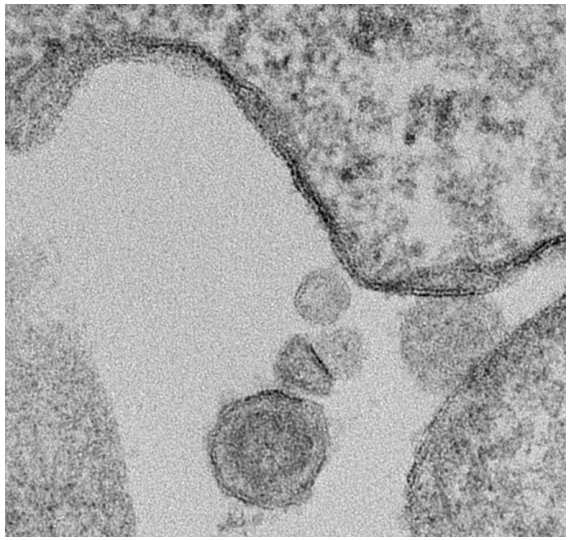
The Environment and ASD
All Chronic Disease?

- More than 200 genes associated with Autism
- Many subtypes
- Pesticides
- Toxins
- EMF
- **Lessons learned from Other human retroviral Infections**
- **Zoonotic transmission exposures**
- **Heavy metals in water-Example from the Silver state**
- **GMO**
- **Vaccinations-The Anti-hygiene Theory**
- Microbiome.

ALL ON TOP OF THE GENETICS

Retrovirus Phylogeny

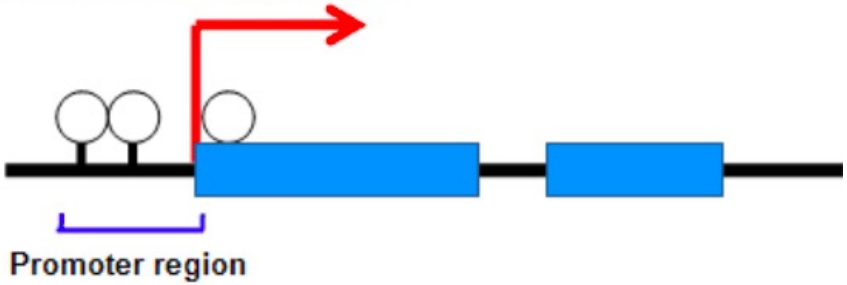




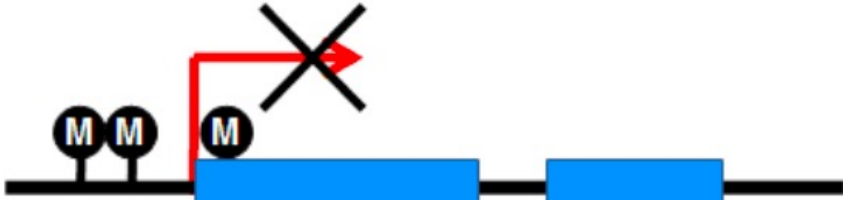
Retroviruses Integrate into genome..forever part of DNA of host

As much as 15% of human genome is made up of endogenous Retroviruses that have been crippled by the immune system That is they are not replication competent

Genes that can be expressed



Genes inactivated by DNA methylation



Molecular and Cellular Biology

Infection with Human Immunodeficiency Virus Type 1 Upregulates DNA Methyltransferase, Resulting in De Novo Methylation of the Gamma Interferon (IFN-γ) Promoter and Subsequent Downregulation of IFN- γ Production

Judy A. Mikovits, Howard A. Young, Paula Vertino, Jean-Pierre J. Issa, Paula M. Pitha, Susan Turcoski-Corrales, Dennis D. Taub, Cari L. Petrow, Stephen B. Baylin and Francis W. Ruscetti

Mol. Cell. Biol. 1998, 18(9):5166.

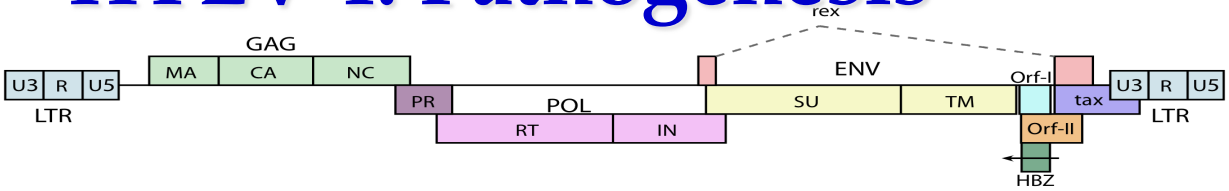
Mechanisms of Pathogenesis:

- Lessons learned from 30 years of Human retrovirus study

- Lessons learned from 40 years of MLV study

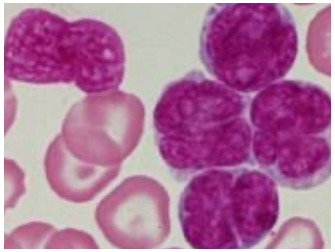
HTLV-I: Pathogenesis

HTLV-I



Genus: Deltaretrovirus (complex)

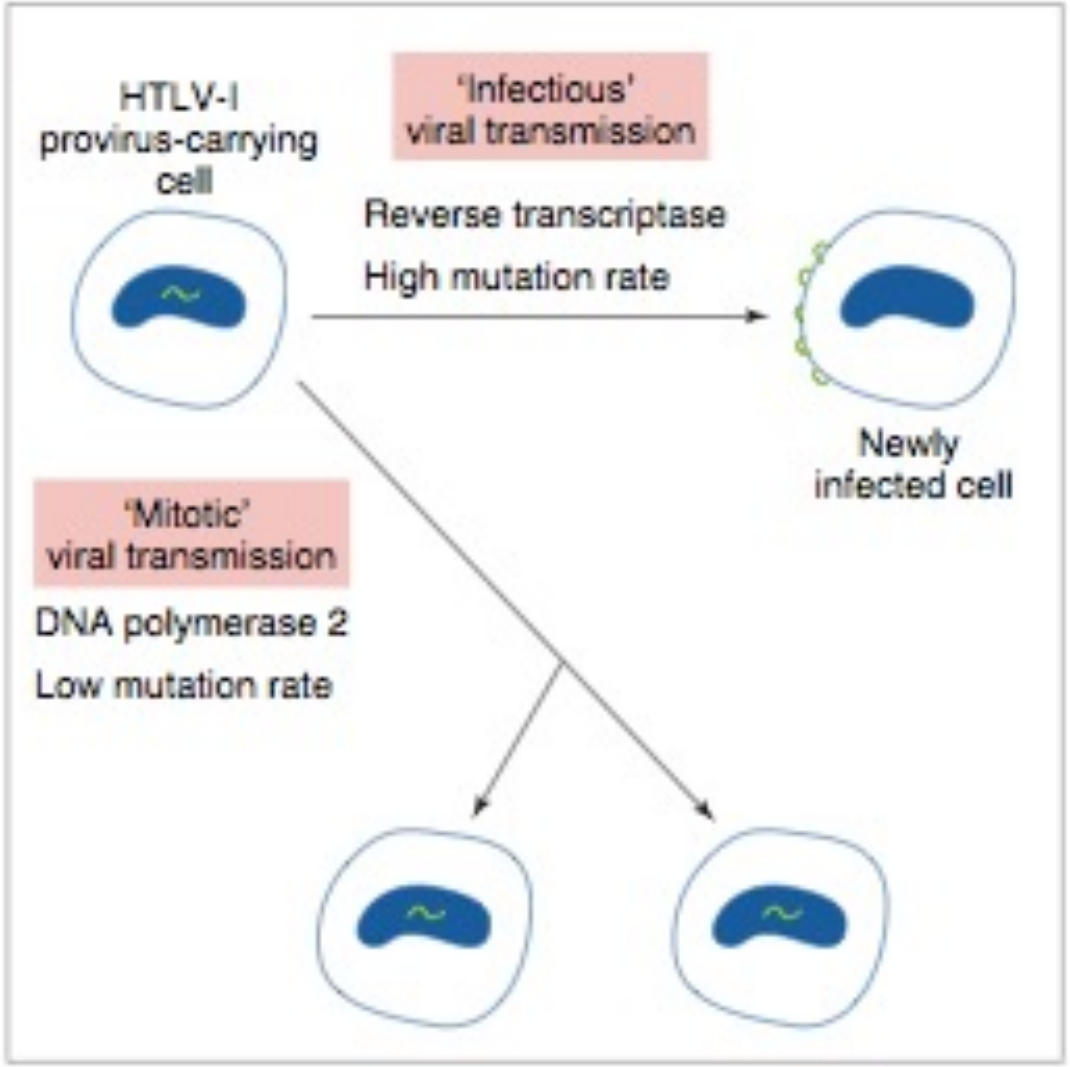
Genome: Multiple spliced RNAs for regulatory and accessory proteins



Pathogenesis:

- **Asymptomatic in majority of individuals**
- **5-8% lifetime risk of developing types of disease:**
  - **Adult T cell leukemia**
    - Clonal malignancy of $CD4^+$ T cells.
    - Long latency; Immune deficiency
    - Tax and HBZ needed for transformation
  - **Inflammatory syndromes**
    - HTLV-I associated myelopathy/Tropical spastic paraparesis
    - Uveitis
    - Arthropathy

Infectious vs. Mitotic transmission of HTLV-1



Increased Cytokine/Chemokine Production in plasma from ATL patients

Concentration in culture supernatant (pg/ml)	**ATL Patient**	**Uninfected**
IL-12p40	130	36
IL-6	2800	17
IL-1β	162	---
TNF-α	600	---
IP10	130	---
MCP-1	770	150
MIP-1α	450	90
IL-8	8500	420

- **Many cytokines such as IL-4, IL-5, IL-7 and type 1 interferons are not expressed in blood of infected patients**

Dysregulated Cytokine/Chemokine Production plasma from ME/CFS patients

CYTOKINES/ CHEMOKINES	Patient N = 156	Control N=140	P value	FUNCTION IN INFLAMMATION
IL-8	1067	11.1	<0.0001	RNase L and CMV activated
IL-13	28	86	<0.0001	Inhibits inflammatory cytokine production
MIP-1β	1840	157	<0.0001	Elevated in Neurodegenerative disease
TNF-α	109	12.8	<0.0001	Stimulates chronic inflammation
MCP-1	468	421	0.003	Elevated in chronic inflammatory diseases
IL-7	21.1	82	<0.0001	Stimulates proliferation of B and T lymphocytes and NK cells
IFN-α	35	60	<0.0001	Stimulates macrophages and NK cells to elicit an anti-viral response
IL-6	271	29	<0.0001	Stimulates chronic inflammation
MIP-1α	673	91	0.0062	Elevated in Neurodegenerative disease
GM-CSF	108	166	<0.0001	Stimulates proliferation of B and T lymphocytes and NK cells

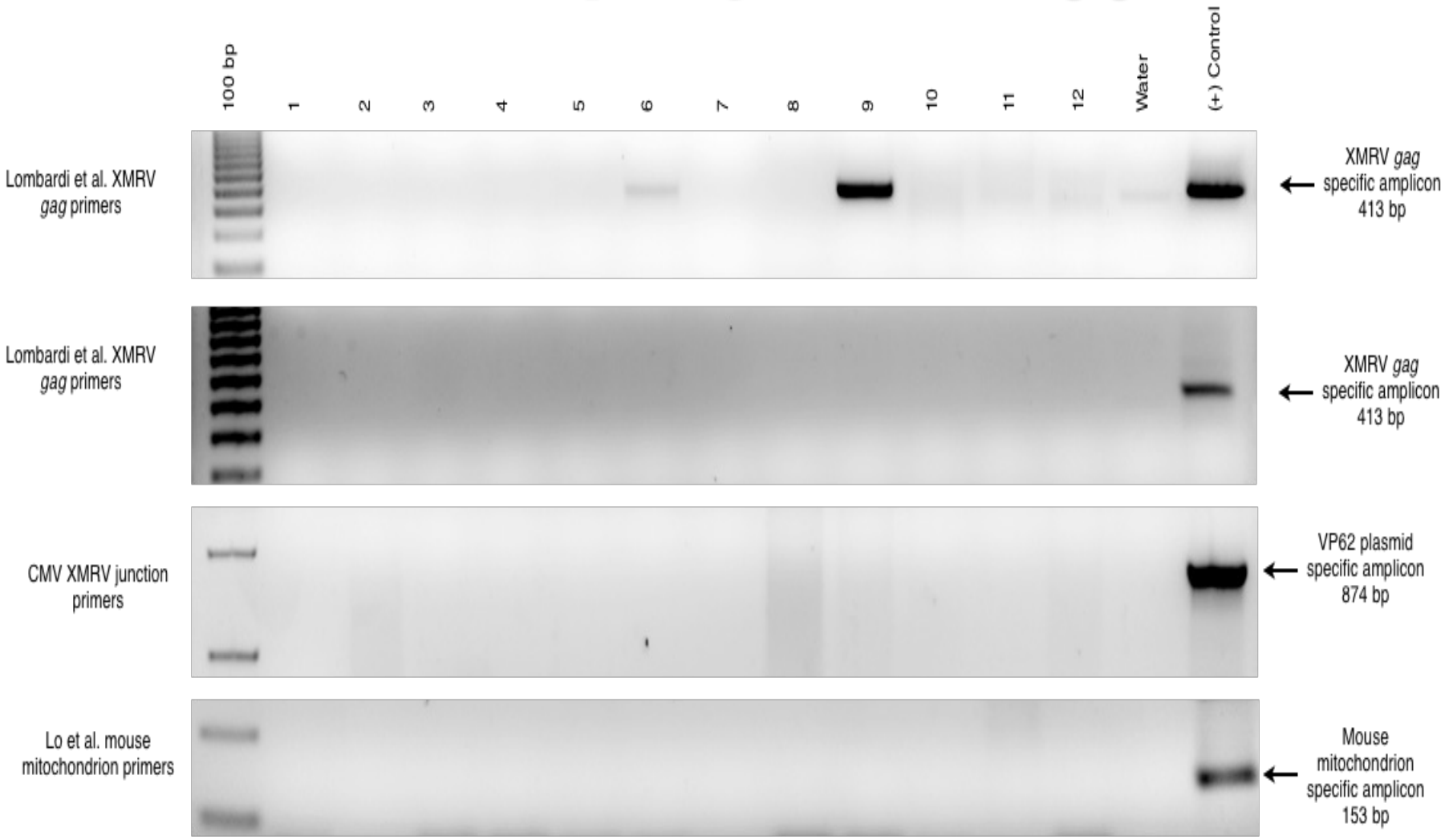
3 B-Cell Lines Derived Directly From CFS Patients' PBMCs

- **CFS patient PBMCs were cultured; 3 samples developed into immortalized cell lines**
- **All three showed high CD20+ expression and two showed high CD23+ expression.**
- **All three showed strong similarity to B cells seen in patients.**

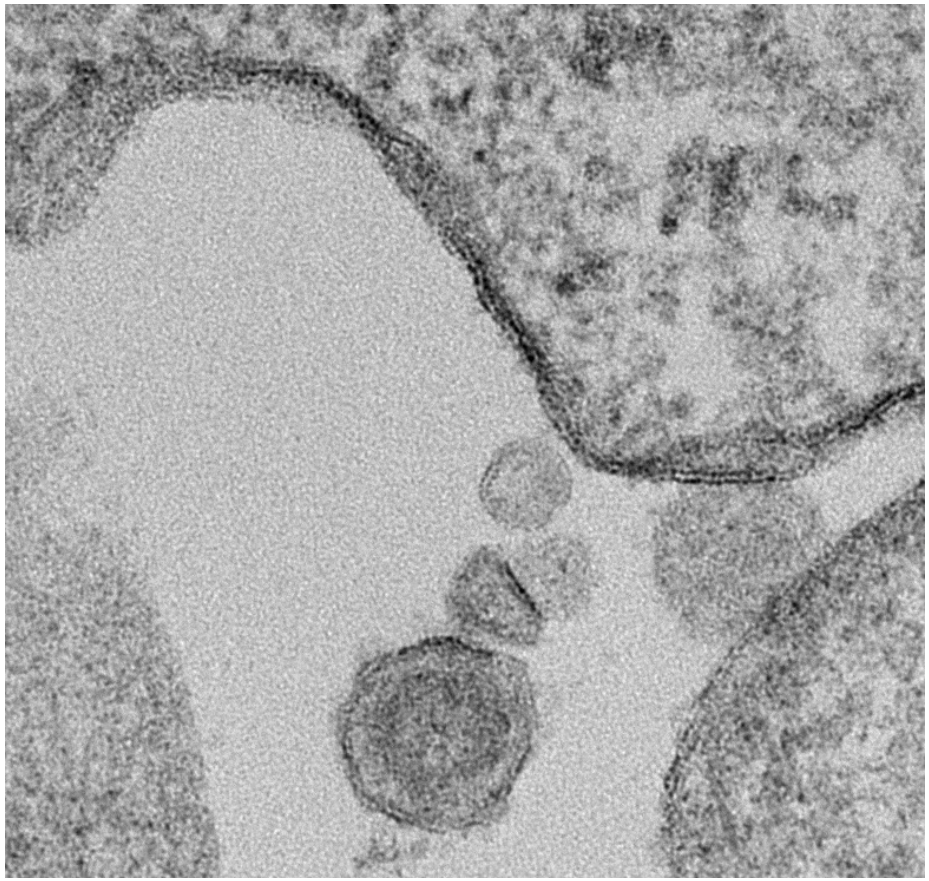
Marker	MCL	WPI 1125	WPI 1186	WPI 1143
CD5	+	+	+	+
CD23	-	-	+	+
CD19	+	+	+	+
CD20	+	+	+	+
FMC7	+	+	-	-
CD3	-	-	-	-
CD4	-	-	-	-
CD7	+	-	-	-
CD8	-	-	-	-
CD10	-	-	-	-
CD38	+	+	+	+
CD45	+	+	+	+
CD56	-	-	-	-
CD122	-	-	-	-
HLA-DR	+	+	+	+
Lambda	+	+	-	-
Kappa	+	+	+	+

These Cell lines were developed from CFS patients. One, (1125) developed MCL; one (1186) was developed from a bone marrow biopsy, 3rd a CLL

Extended PBMC cell culture without manipulation shows XMRV *gag* infection in samples negative for XMRV *gag* RNA



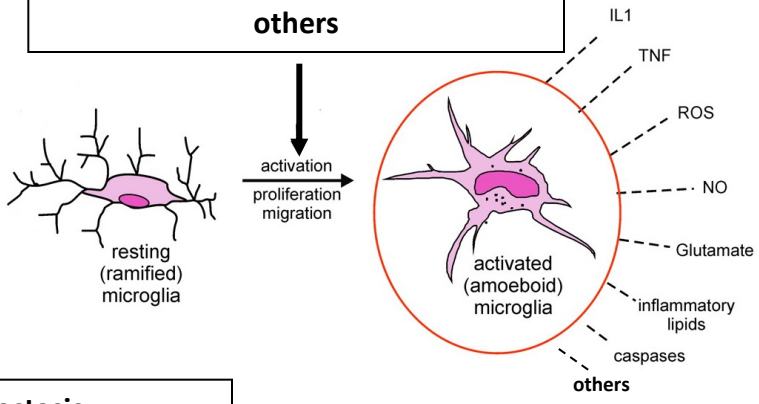
Electron Micrograph of gamma retrovirus isolated from ME/CFS patients blood cells

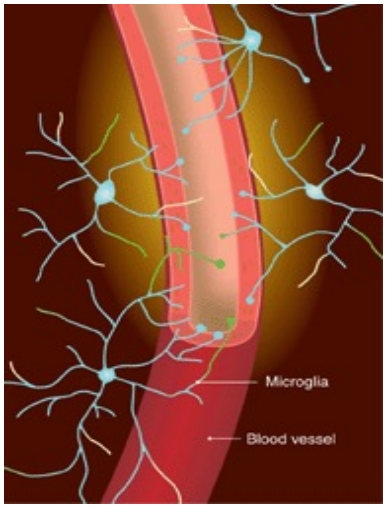


March 2009

Microglia Activation in Neurodegeneration

Stimuli
Loss of cell:cell communication, matrix breakdown, infections, vascular damage, others





Fetler, L and S Amigorena, Science 2005, 309:392

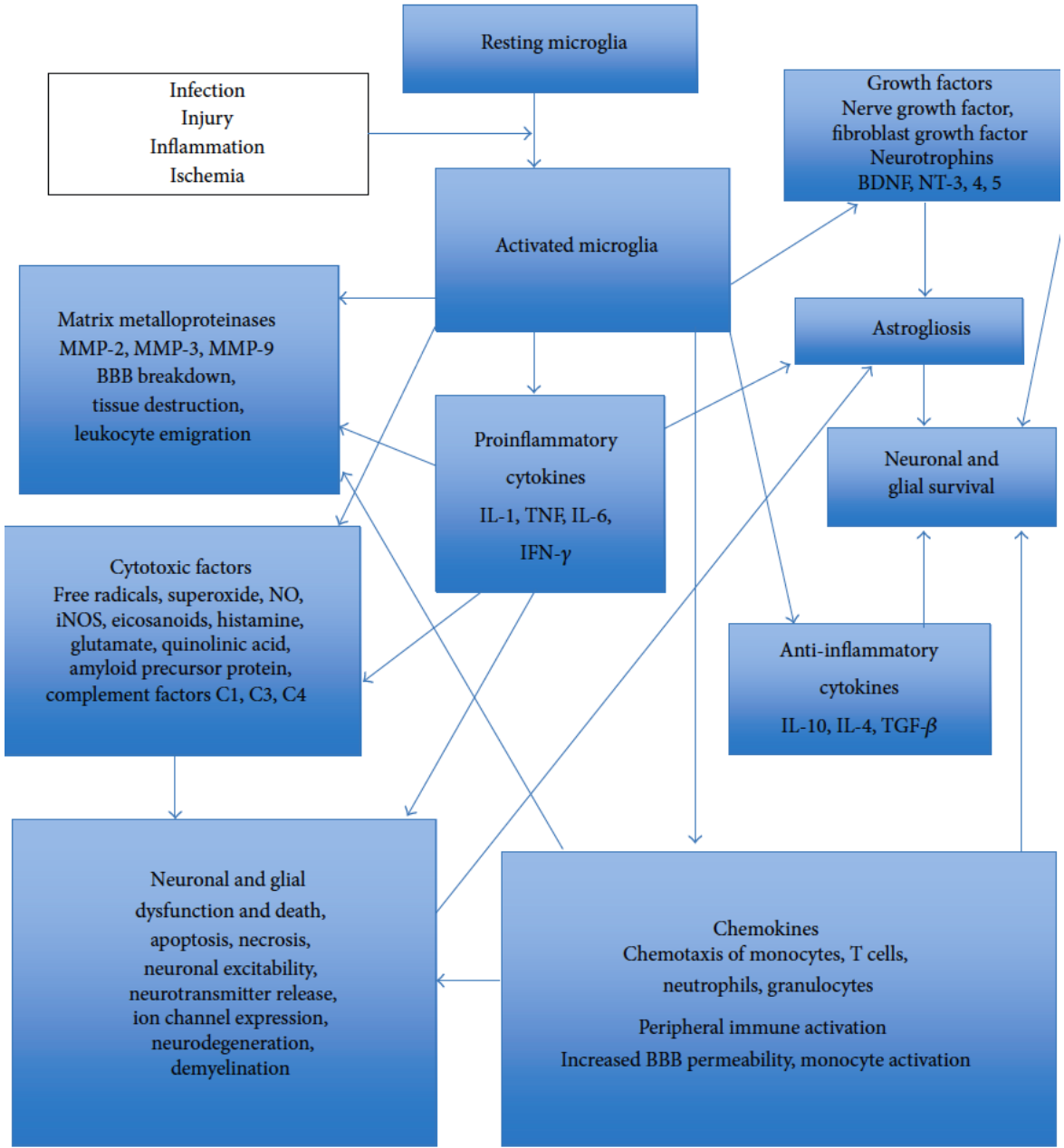
Homeostasis
Phagocytosis of debris, neurotrophins

Damage
Neurotoxins, phagocytosis of normal neurons, apoptosis

Neurodegenerative disorders

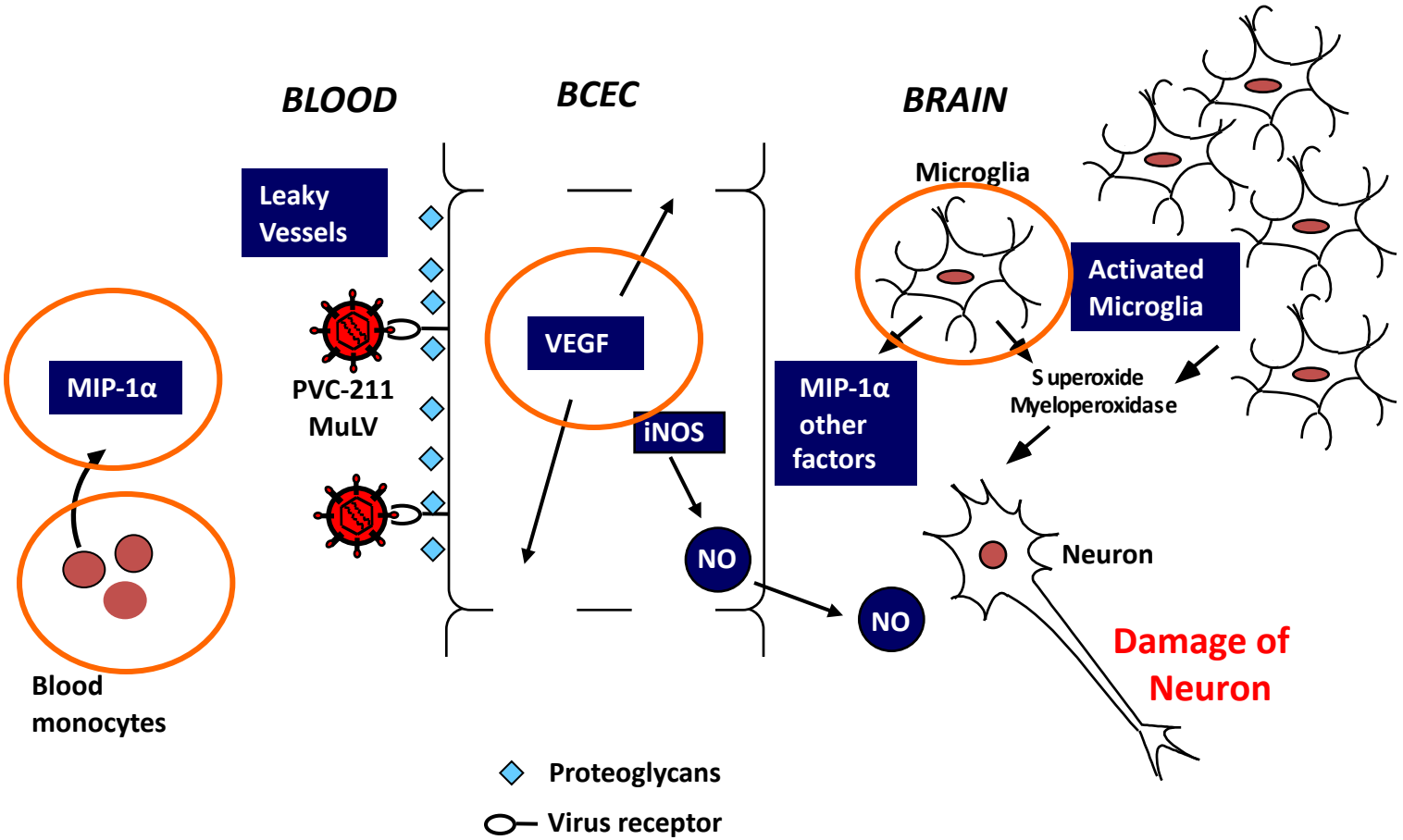
- **Parkinson's disease**
- **Alzheimer's disease**
- **Multiple sclerosis**

Central role of micgroglia in Neuroinflammation



Rameshe Et. Al. 2013
Mediators of inflammation

Model for the Induction of Neurodegeneration by one strain of MLV in an animal model



these immune pathways see in ASD and Other Chronic neurological diseases

Two important lessons learned from studying MuLVs

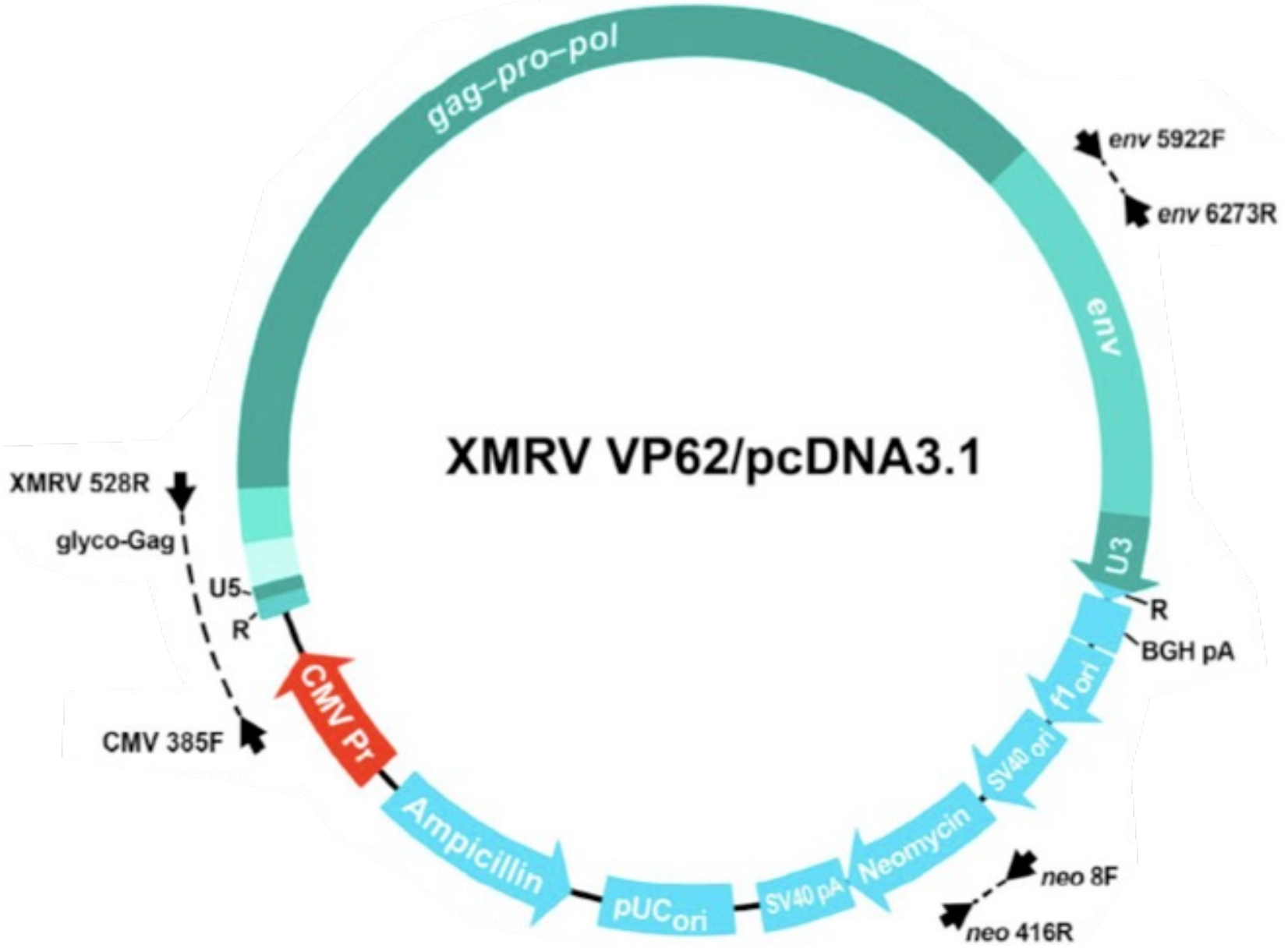
- **While insertional mutagenesis by MuLVs can result in transformation of cells and the development of leukemias and lymphomas, the envelope proteins encoded by these viruses can also have profound biological effects.**
  So it's important to study the biological effects of the XMRV envelope protein.

- **MuLVs can be expressed in the CNS, triggering an inflammatory response that can cause severe neurological damage. Since similar inflammatory responses are associated with ME/CFS, XMRV could be playing a role.**

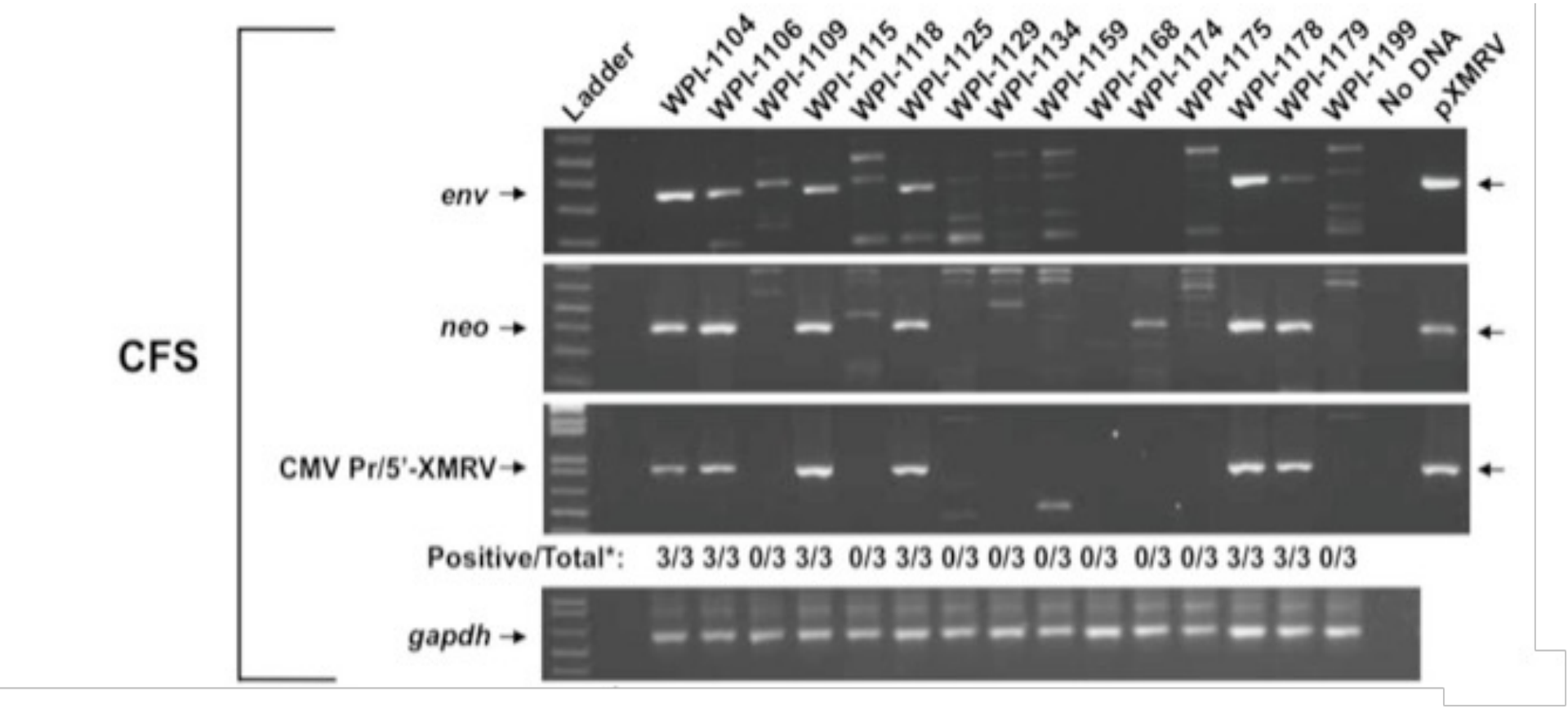
XMRV Controversy

- What happened?
- What did we learn?
- Where do we go from here?

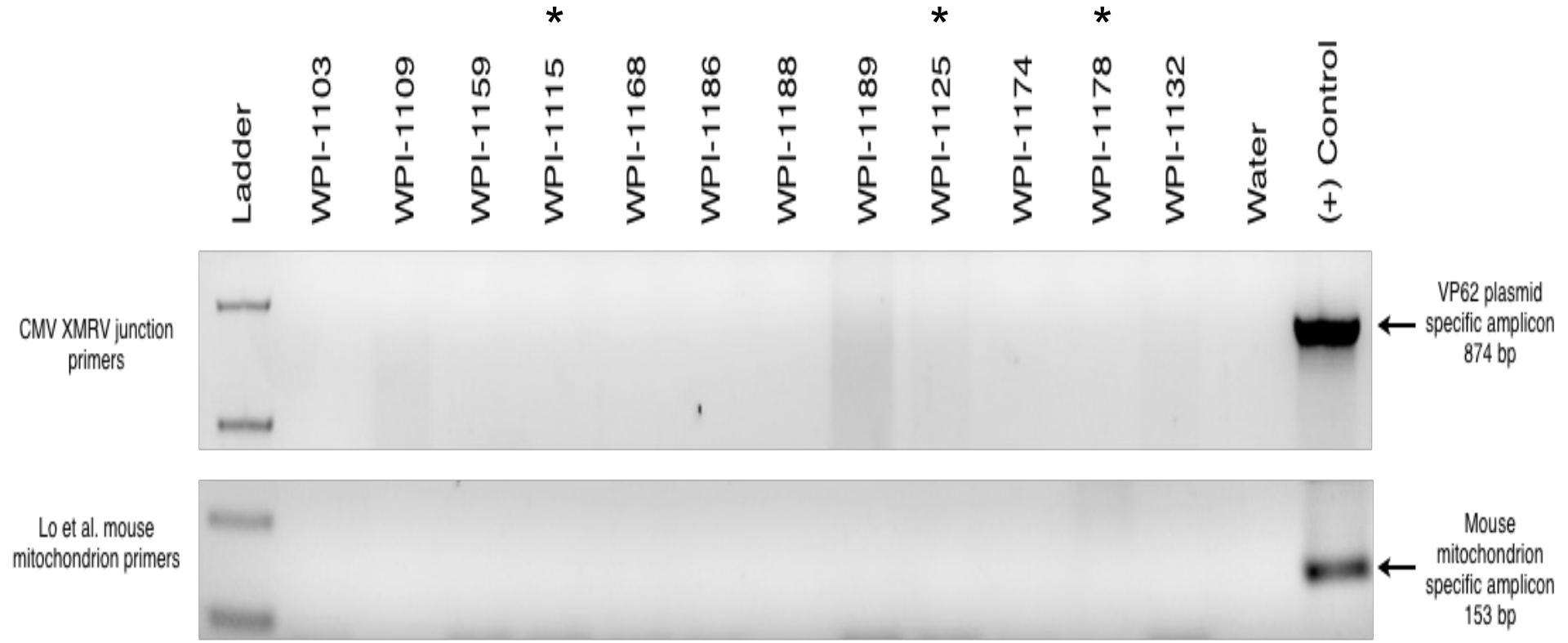
Schematic of Plasmid containing XMRV/VP62



Six WPI DNA Samples shown in Fig. 1 of the original study analyzed by the Silverman Lab in 2009 contained VP-62 plasmid

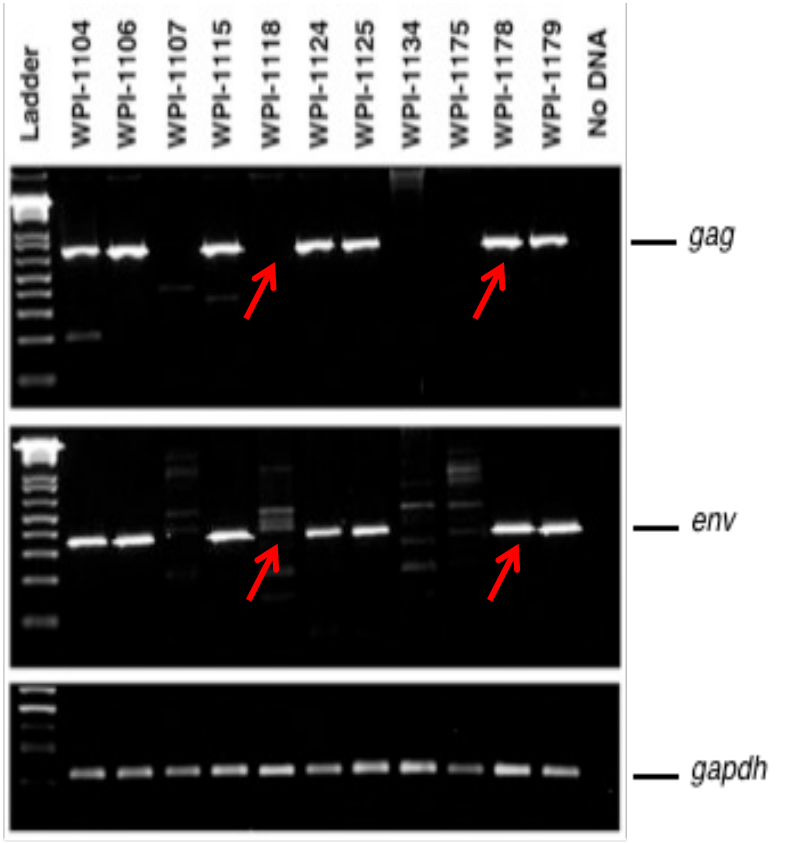


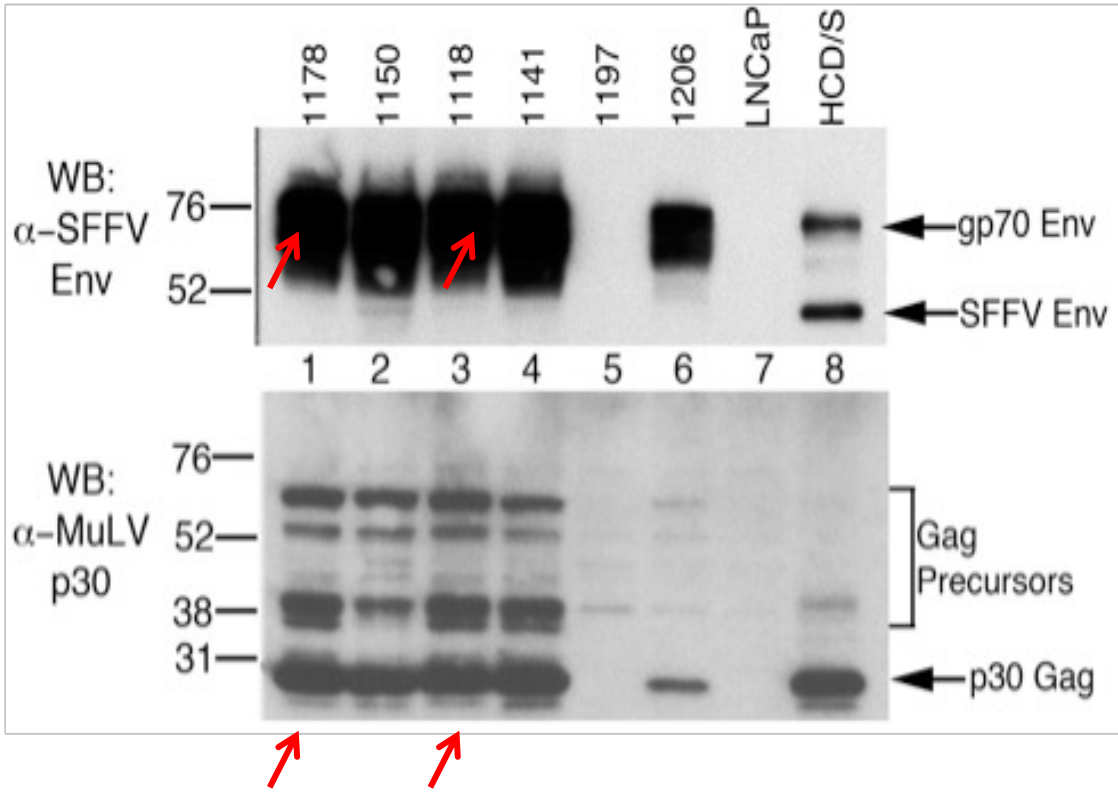
Original DNA Samples were negative for XMRV plasmid



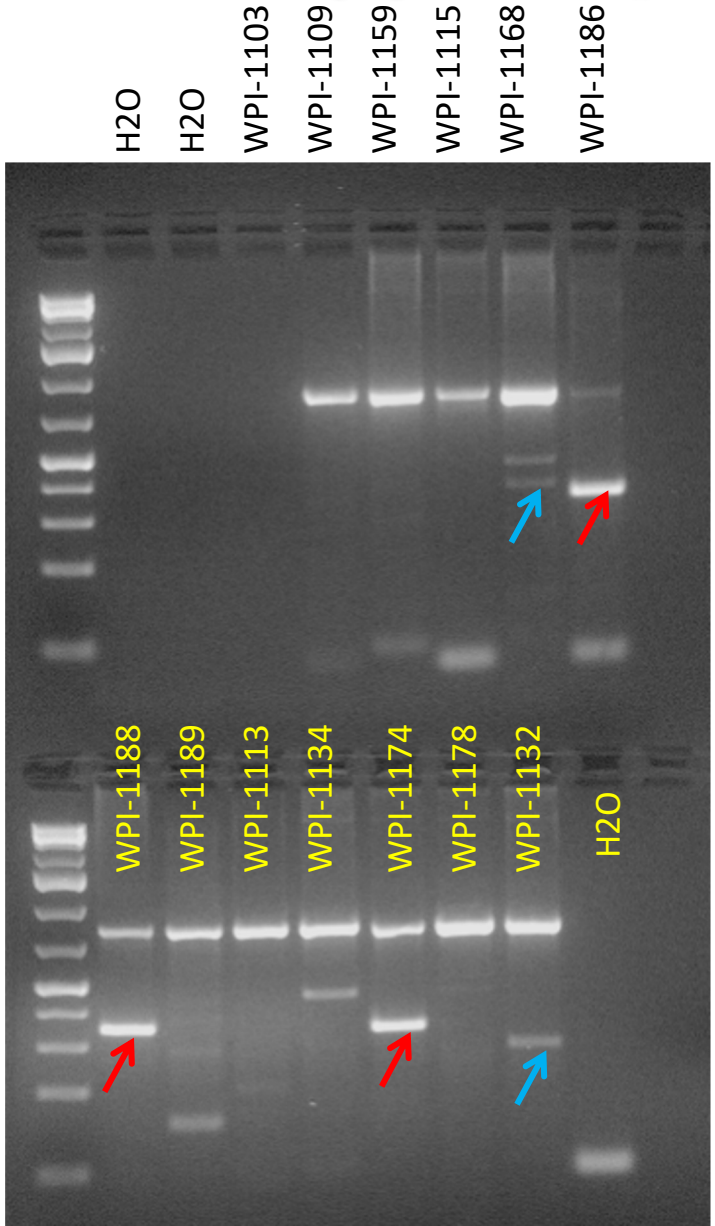
- **Pitfall: Choose your collaborators wisely!!**

Cell-Free Transmission of XMRV from PCR-negative CFS Patients' Plasma to LNCaP cells





Independent Reanalysis of archival samples used in Original Study Detected XMRV gag without plasmid or mouse contamination



PCR performed with USB HotStart-IT FideliTaq Master Mix

94°C 2 min
45 cycles:
94°C 30 sec, 54.8°C 30 sec, 72°C , 30 sec
72°C 3 min.

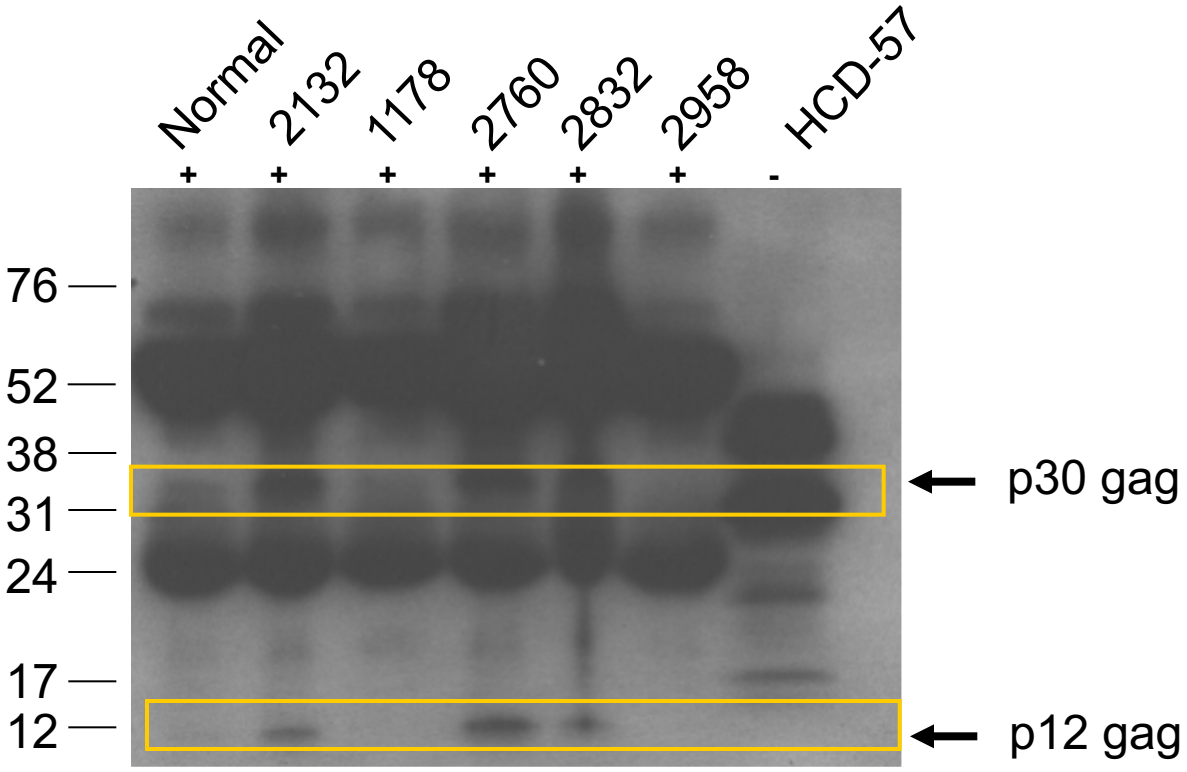
All three are negative for IAP and negative for CMV385F/XMRV528R primers for VP62 junction fragment

Sequencing of bands:

↗ Non-specific (Human DNA)
↗ XMRV Gag

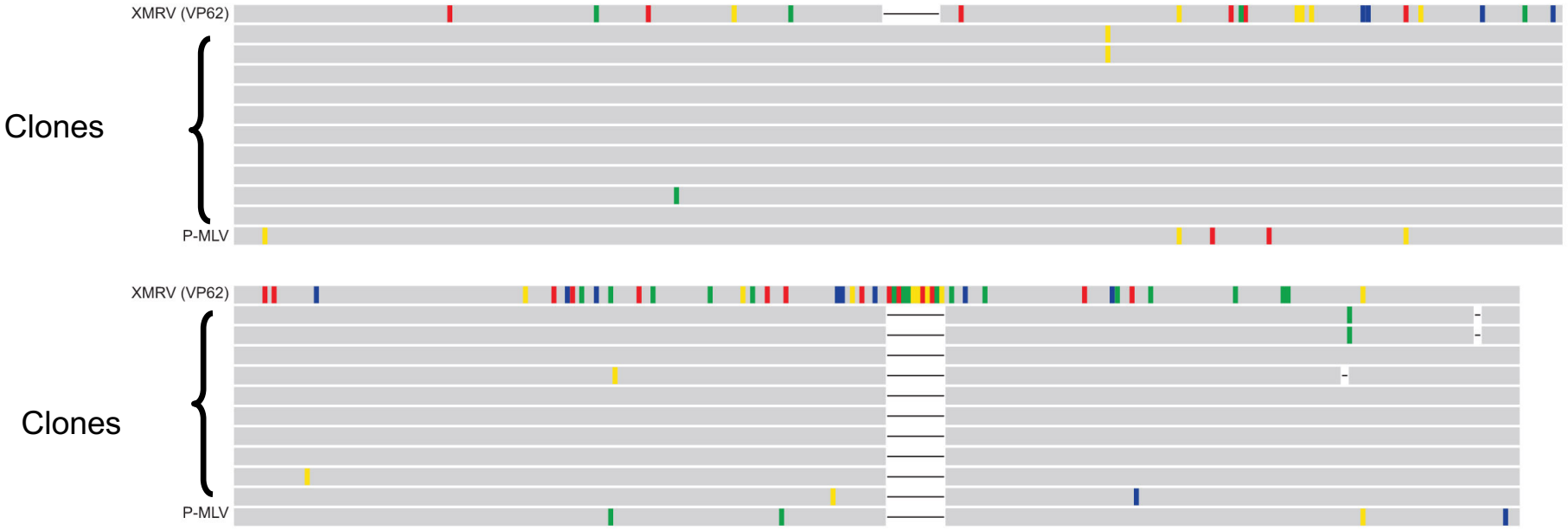
Direct Isolation of XMRV Protein From Plasma of CFS Patients By Immunoprecipitation with Anti-X-MLV Antibodies

IP: Goat anti-X-MLV (BALB-V2)



Blot: Goat anti-R-MuLV Gag

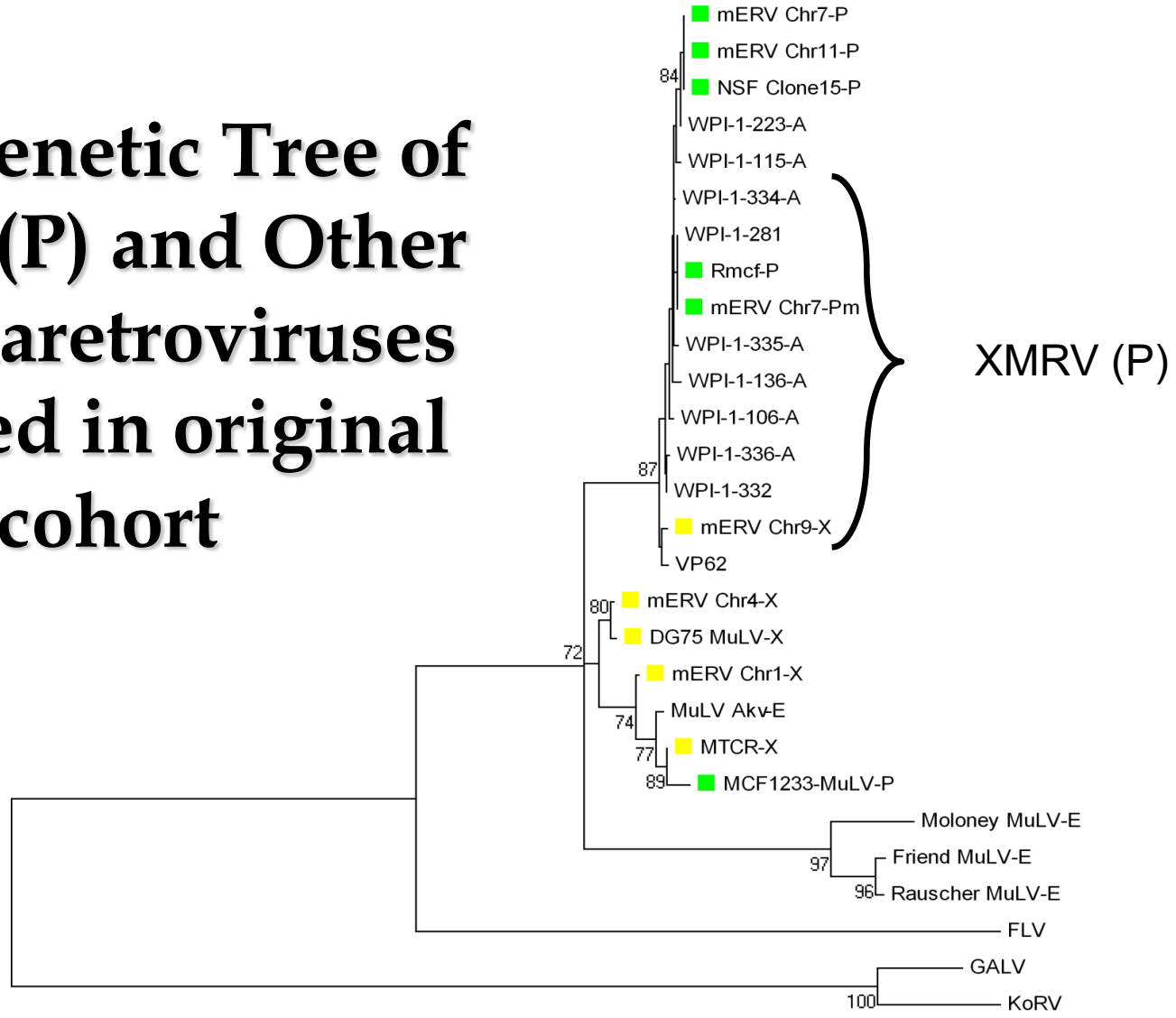
Clones of XMRV Env SU Similar to Polytropic XMRVs



- The main XMRV/ in this patient is unlikely to be VP-62

Pitfall: Extraordinary measures are required to rule out contamination!

Phylogenetic Tree of XMRV(P) and Other Gammaretroviruses detected in original cohort



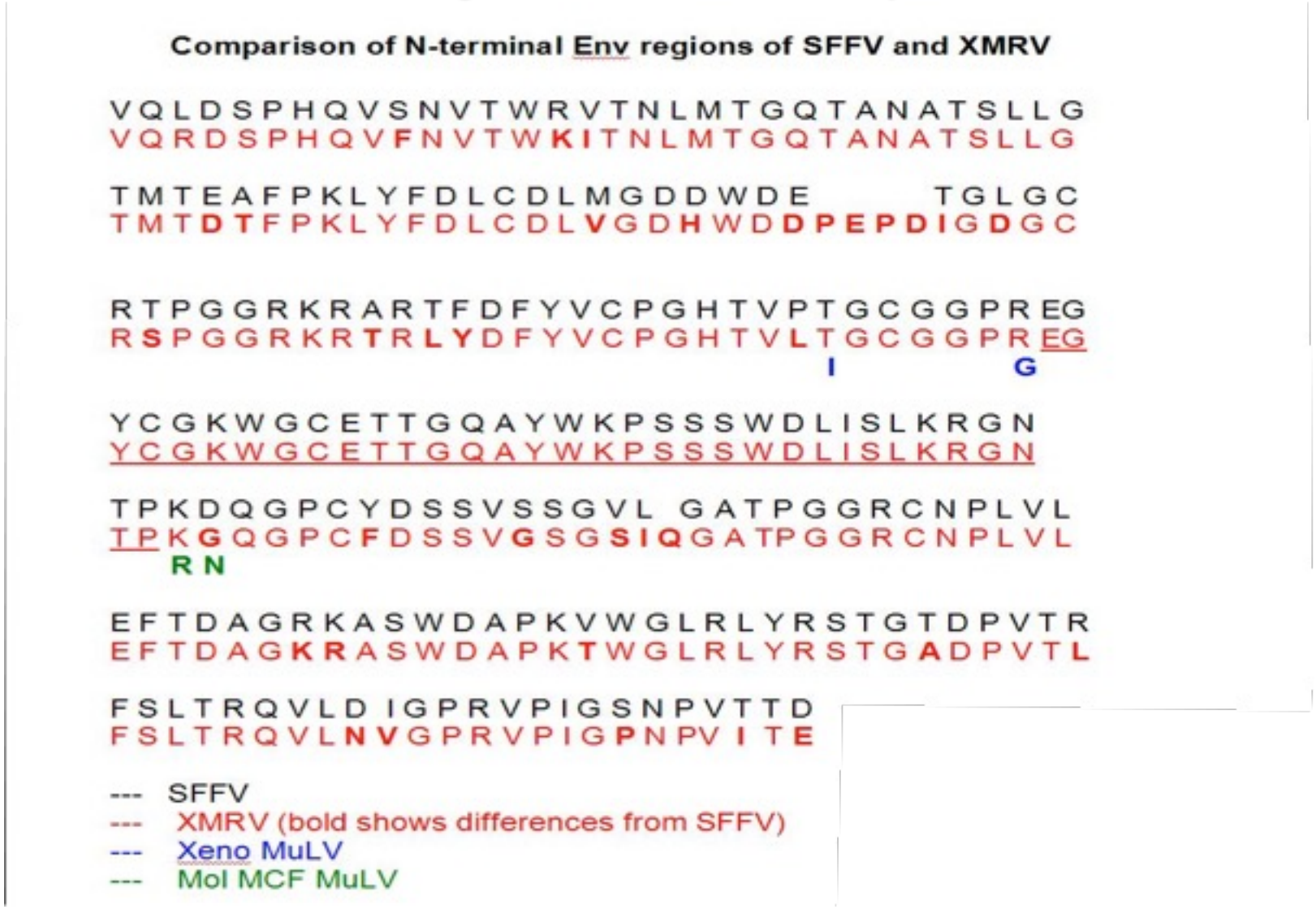
Detection of MLV-related virus gene sequences in blood of patients with chronic fatigue syndrome and healthy blood donors

Shyh-Ching Lo[a,1], Natalia Pripuzova[a], Bingjie Li[a], Anthony L. Komaroff[b], Guo-Chiuan Hung[a], Richard Wang[c], and Harvey J. Alter[c,1]

[a]Tissue Microbiology Laboratory, Division of Cellular and Gene Therapies and Division of Human Tissues, Office of Cellular, Tissue and Gene Therapy, Center for Biologics Evaluation and Research, Food and Drug Administration, Bethesda, MD 20892; [b]Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115; and [c]Department of Transfusion Medicine, The Warren Grant Magnuson Clinical Center, National Institutes of Health, Bethesda, MD 20892

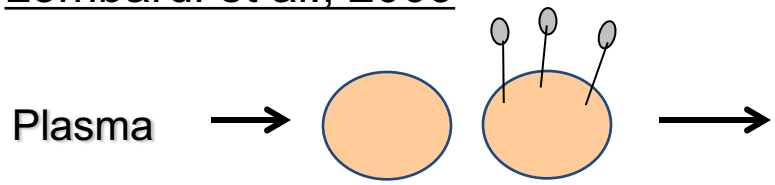
- Showed gag sequences of MRV more closely related to polytropic MLV related by nested PCR replication of Lombardi et. al. nested PCR
- PMRV DNA in 86.5 of CFS and 6.8% of healthy from samples drawn in 1991-4
- Rigorously ruled out contamination
- 8/9 CFS patients showed same gag sequences upon fresh draw 15 years later

N-Terminus of SFFV ENV allows recognition of most potential XMRVs using monoclonal antibody 7C10



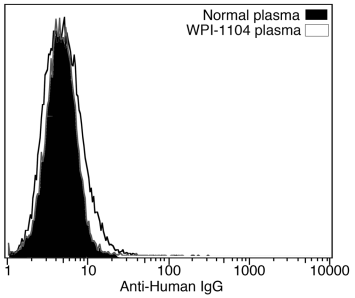
Assay used to Detect Anti-XMRV/HGRV Antibodies

Lombardi et al., 2009

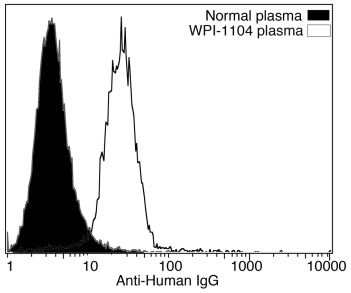


FACS analysis of Murine Cell Line
+/- SFFV Env

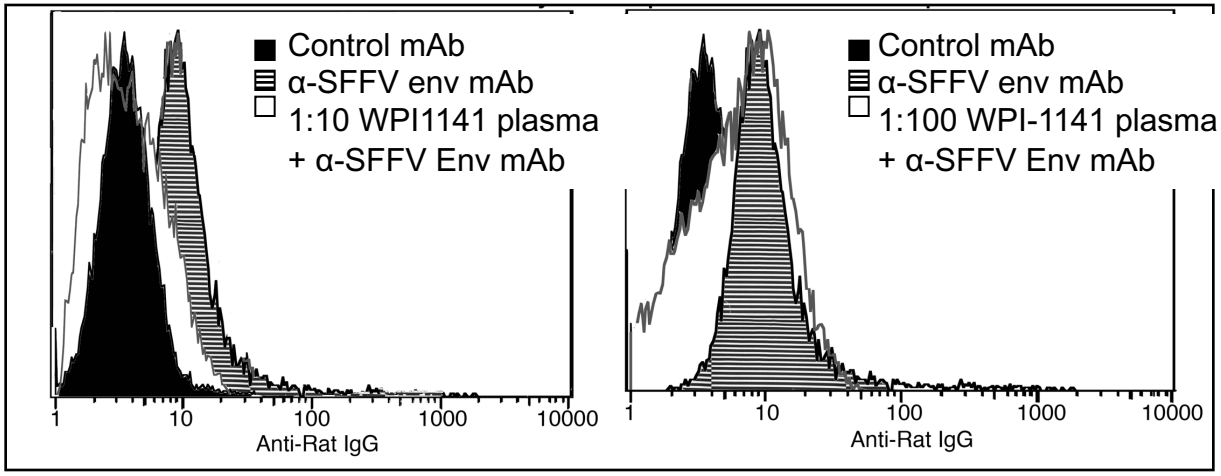
- SFFV Env



+ SFFV Env



Plasma from CFS patients block binding of SFFV Env rat mAb to the B cell line expressing SFFV Env, demonstrating specificity



An ANTIBODY POSITVE RESULT DOES NOT NECESSARILY SHOW THE PRESENCE OF A REPLCIATION COMPETENT RETROVIRUS

Antibodies to XMRV ENV Reproducibly Detected in Human Population

TABLE 3 Equivalent levels of XMRV sequences and anti-XMRV antibodies in CFS (chronic fatigue syndrome) patients and matched controls

			CFS/ME cases ($n = 147$)		Controls ($n = 146$)	
Lab site	Analysis	Sample	Total studied	No. positive (%)	Total studied	No. positive (%)
CDC	RT-PCR	Plasma	147	0 (0.0)	146	0 (0.0)
FDA	RT-PCR	Plasma	121[a]	0 (0.0)	110[a]	0 (0.0)
	PCR	PBMC	121[a]	0 (0.0)	111[a]	0 (0.0)
Mikovits, Ruscetti, and Hanson	PCR of cultured PBMC	PBMC	117[b]	0 (0.0)	126[b]	0 (0.0)
Mikovits and Ruscetti	Serology	Plasma	147	9 (6.1)	146	9 (6.2)

[a] Numbers represent all samples available for analysis at that site.

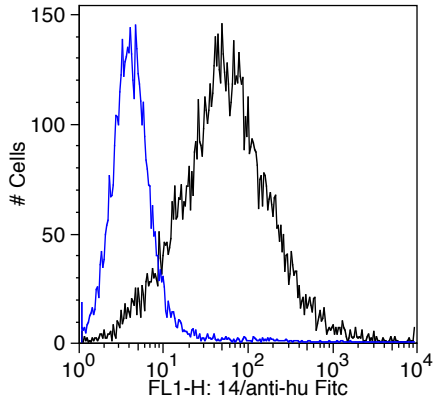
[b] Fifty samples (30 cases; 20 controls) were unable to be assayed because at least one of two aliquots from each set of subject PBMC did not grow in tissue culture.

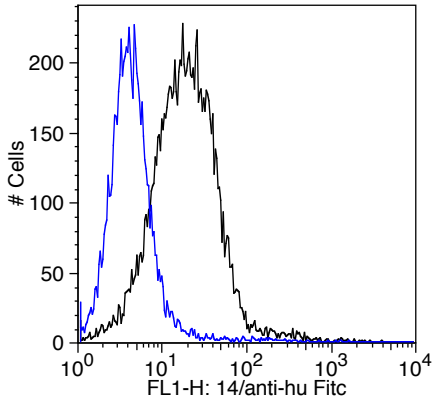
CONCLUSION

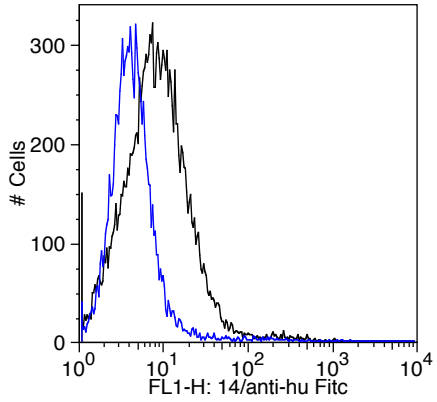
Taken together these data suggest there are
additional human gamma retroviruses which may be involved in the
Pathogenesis of neuroimmune diseases?

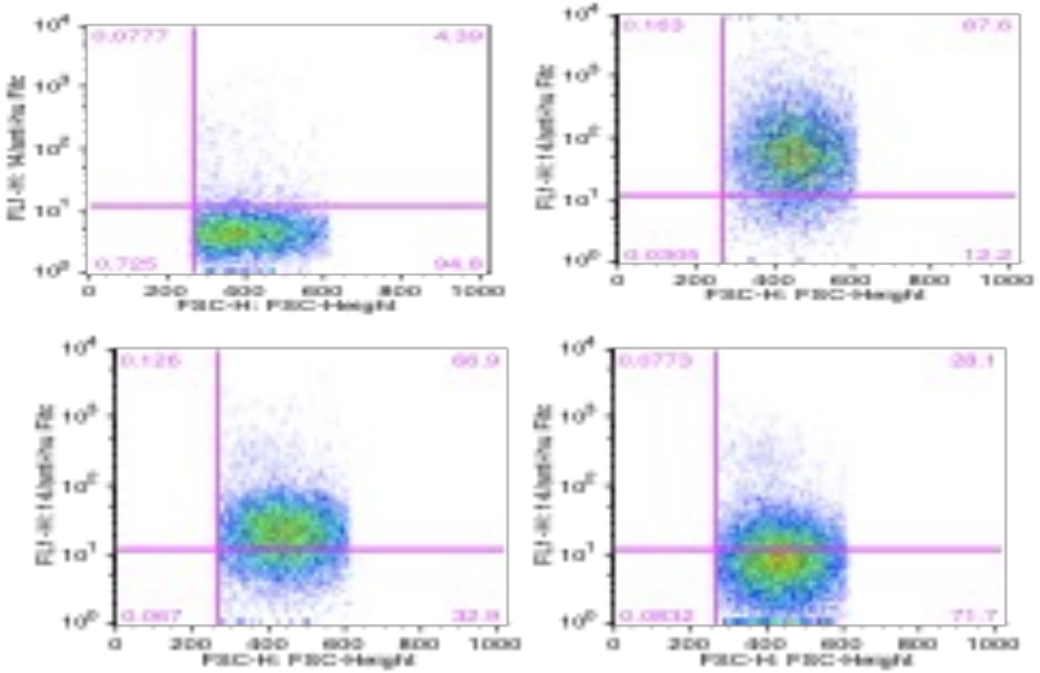
Is there evidence beyond these data to support this conclusion?

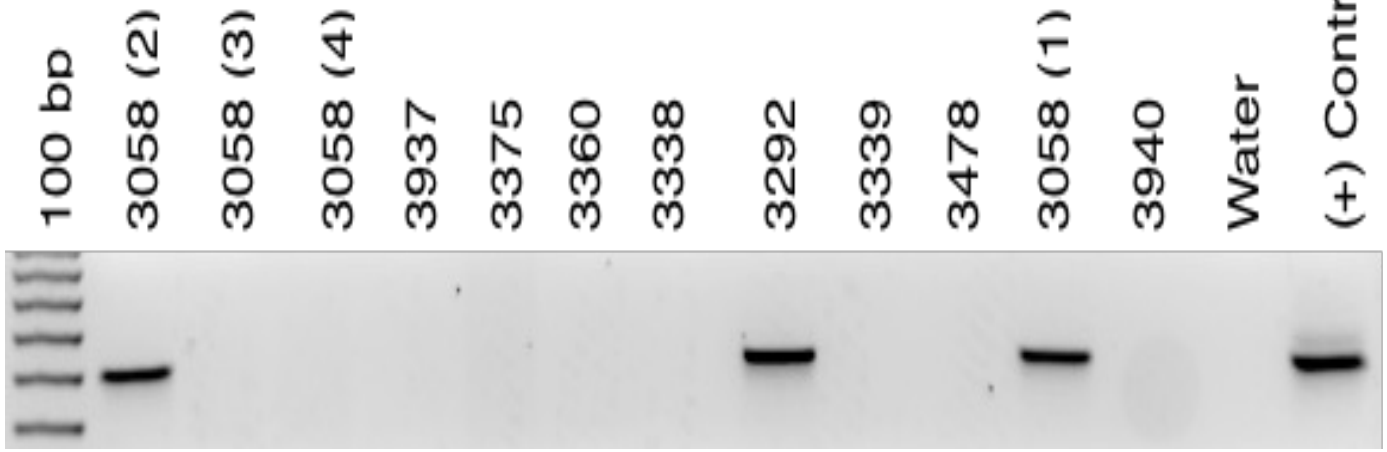
Case report 3058 : plasma Gag RNA + and seropositive CFS/CLL











ARVs provide therapeutic benefit in some patients with autoimmune, Neuroimmune Disease and Cancer

Beneficial Effects could be against:

- An exogenous Replication Competent Retroviruses
- An expressed endogenous virus in an immune compromised individual
- A defective virus expressing only viral proteins
- Aberrantly expressed cellular RNA including microRNA (regulatory)

Identification of additional retrovirus isolated 15 years ago in ME/CFS

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

Brian D Halligan[1], Hai-Yuan Sun[2], Vladimir M Kushnaryov[2] & Sidney E Grossberg*[2]

[1]Biotechnology & Bioengineering Center, Medical College of Wisconsin, 8701 Watertown Plank Road, Milwaukee, WI 53226, USA

[2]Department of Microbiology & Molecular Genetics, Medical College of Wisconsin, 8701 Watertown Plank Road, Milwaukee, WI 53226, USA

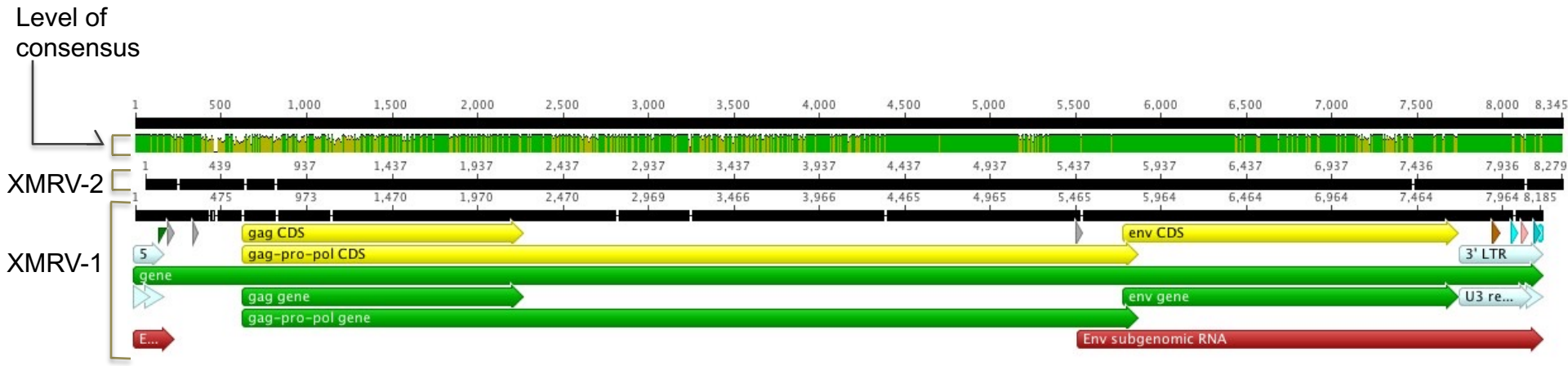
*Author for correspondence: Tel.: +1 414 276 8194 ▪ segrossb@gmail.com

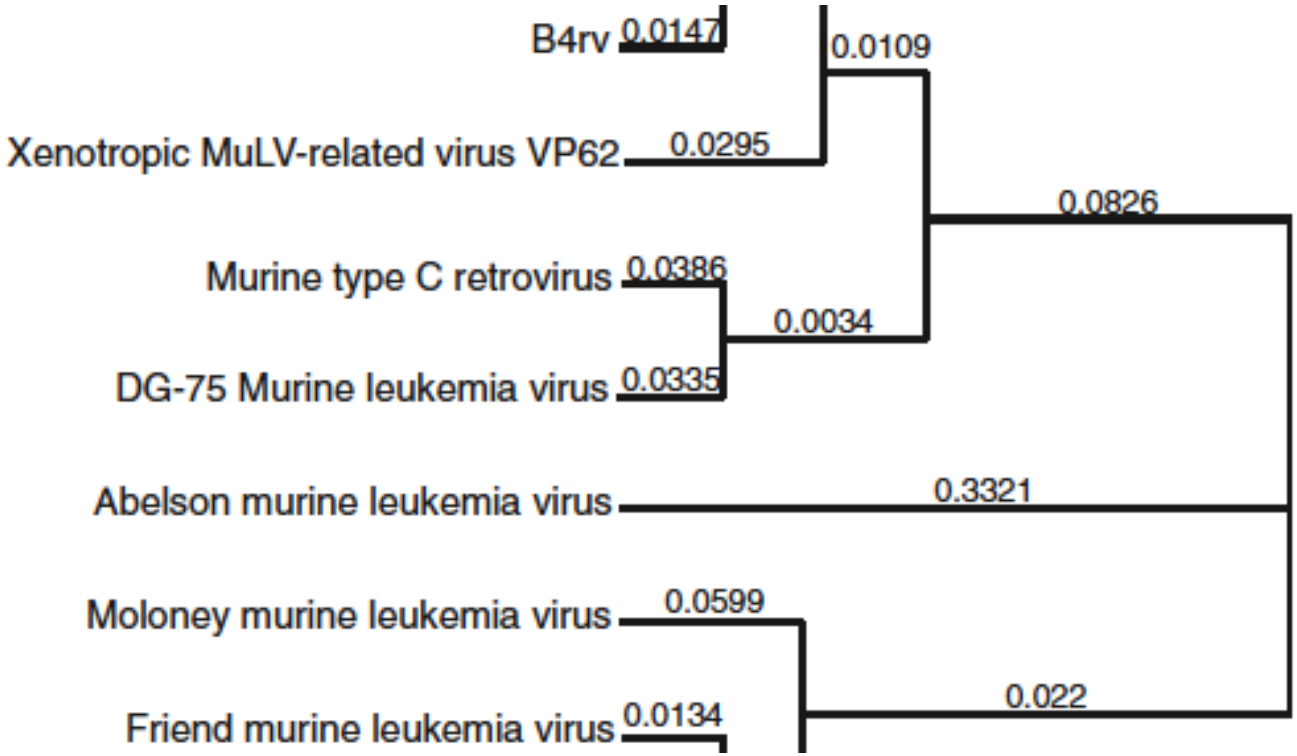
Future Virology

The JHK virus (JHKV) was previously described as a type C retrovirus that has some distinctive ultrastructural features and replicates constitutively in a human B-lymphoblastoid cell line, JHK-3. In order to facilitate the cloning of sequences

August 2013

Sequence and phylogenetic analysis of a novel xenotropic XMRV-like MLV B4rv,



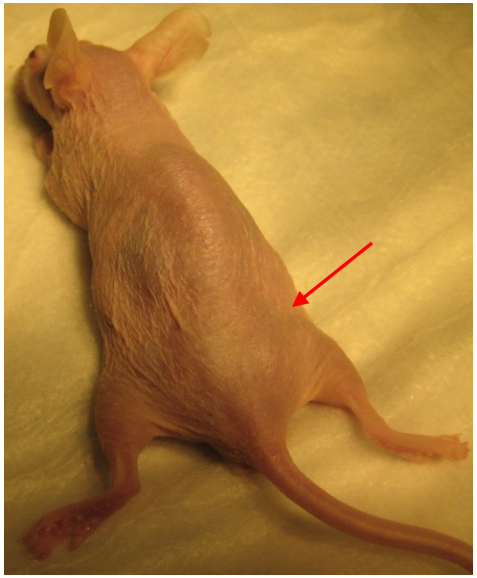


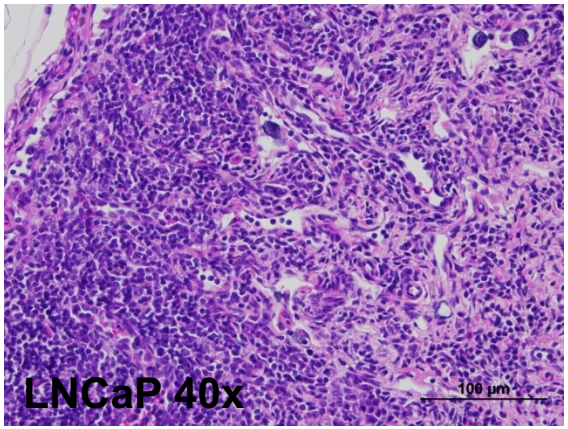
93% overall homology

Gary Owens lab
November 10, 2009
Published 2013

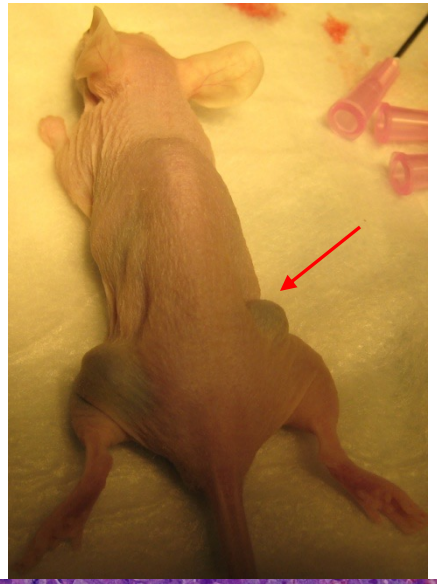
XMRV2 (B4RV) Infected Tumors were Hemorrhagic

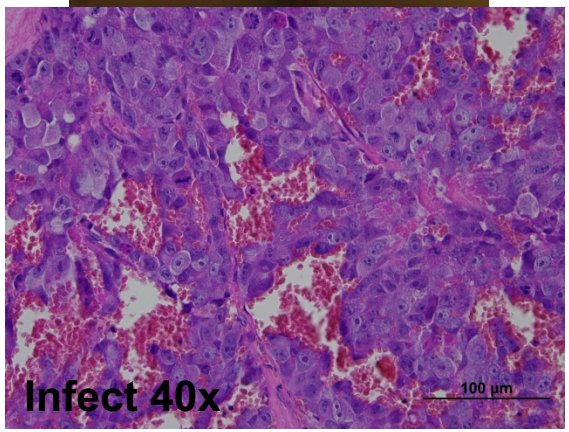
LNCaP





LNCaP-XMRV2 Infect





Murgai *et al. Retrovirology* 2013, **10**:34
http://www.retrovirology.com/content/10/1/34

RETROVIROLOGY

RESEARCH Open Access

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

Meera Murgai[1], James Thomas[2], Olga Cherepanova[1], Krista Delviks-Frankenberry[4], Paul Deeble[3], Vinay K Pathak[4], David Rekosh[5] and Gary Owens[1*]

ENV proteins from both viruses impact tumor pathogenesis (change microvasculature)

Similarities to Vascular Pathologies seen in ME/CFS

These Microvasculature aberrations caused solely by XMRV ENV protein

Although it is highly unlikely that either XMRV VP62 or B4Rv themselves infect humans and are pathogenic, the results suggest that xenograft approaches commonly used in these studies of human cancer promote the evolution of novel retroviruses with pathogenic properties.
Similar retroviruses may have evolved to infect humans!

What did we learn?

- Human Retroviruses could be transmitted through aerosolization
- Additional gamma retroviruses in humans with chronic Disease?
- Recombinants of RVs which were replication competent (that is could be infectious new virus!)

Horizontal Spread of Gammaretroviruses in Tissue Culture

Table 4. Characterization of murine leukemia viruses (MLV) detected in human non-xenograft cultures in xenograft culture laboratories[1]

Cell line type	MLV positive cell lines[1]	MLV sequence homology[2]	RT Enzyme (nU/μl)	Mouse DNA[3]	Other sources or passages[4]	Source: Lab PI
NSCLC	NCI-H460	ND	Negative	-	Negative	C. Rudin
NSCLC	NCI-H1155	MLV N417	ND	-	ND	A. Gazdar (NCI)[6]
SCLC	NCI-H60	MLV N417	3.6×10^6	-	Negative	A. Gazdar (NCI)[6]
SCLC	NCI-H82	MLV NZB	1.3×10^6	-	Negative	C. Rudin
SCLC	NCI-H1092	MLV N417	8.0×10^3	-	Negative	A. Gazdar (NCI)[6]
SCLC	NCI-H182	MLV N417	ND	-	ND	A. Gazdar (NCI)[6]
SCLC	NCI-H289	MLV N417	ND	-	Negative	A. Gazdar (NCI)[6]
SCLC	NCI-H1514	MLV N417	ND	-	ND	A. Gazdar (NCI)[6]
Colon	RKO	XMRV	2.9×10^3	-	Negative	A. Maitra
Prostate	PrEC2	ND	ND	-	ND	J.T. Hsieh
Prostate	LNCaP	Multiple MLV strains[5]	ND	++++	Negative	J.T. Hsieh
Prostate	PC3	ND	ND	-/+	Negative	J.T. Hsieh
SCLC	NCI-H146	MLV NZB likely	7.2×10^5	-/+	Negative	C. Rudin

Zhang et al., Cancer, Biol. Ther. 2011, 12:617

Learned : ability of these viruses to spread to uninfected cells through aerosolization

Replication competent Retroviruses in 10 Days!

JVI
Journals.ASM.org

Generation of Multiple Replication-Competent Retroviruses through Recombination between PreXMRV-1 and PreXMRV-2

Krista Delviks-Frankenberry,[a] Tobias Paprotka,[a]* Oya Cingöz,[c]* Sheryl Wildt,[d] Wei-Shau Hu,[b] John M. Coffin,[c] Vinay K. Pathak[a]

Viral Mutation Section[a] and Viral Recombination Section,[b] HIV Drug Resistance Program, National Cancer Institute—Frederick, Frederick, Maryland, USA; Program in Genetics, Sackler School of Graduate Biomedical Sciences, Tufts University, Boston, Massachusetts, USA[c]; Harlan Laboratories, Indianapolis, Indiana, USA[d]

- Are two RCRs made by passing human prostate tissue through mouse; XMRV, BRV4 (second recombinant infectious virus occurring in human cells)
- Additional XMRV-like viruses may exist

- They do not have to be the exact sequence of XMRV (VP62)

Emerging Concepts

- Recombination events in animal and human cells can generate families of infectious related gamma retroviruses
- Greatest concern is that they may acquire the ability to infect humans
- XMRV-like sequences and proteins important in human disease pathogenesis

New Technologies: Comprehensive Sequence Analysis of Nuclear mitochondrial genes

- NGS for variants in the nuclear mitochondrial exome that contribute to neurological disorders whose symptoms resemble mitochondrial disease.

Case Report Results:

- Abnormal autosomal dominant Variant was found in SCN4A gene that is likely a pathological mutation
- Pathological mutations found in two other patients also with multiple functional conditions (ME/CFS)

Incidental finding:

This patient has three variants in *RNASEL*. Mutations in this gene have been associated with predisposition to prostate cancer and this gene is a candidate for the hereditary prostate cancer 1 (HPC1) allele. One of these variants, p.E265*, has been reported in the literature in 4 brothers with prostate cancer.

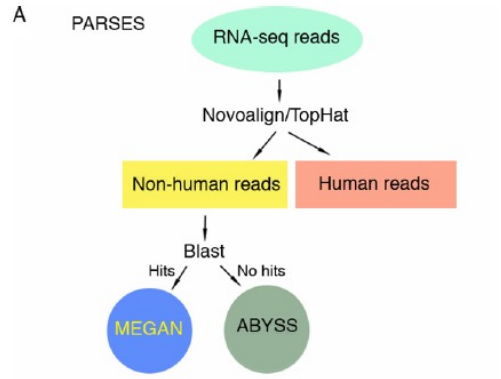
New Concepts: drugs targeting channelopathies (Diamox) and key mitochondrial targets mTOR

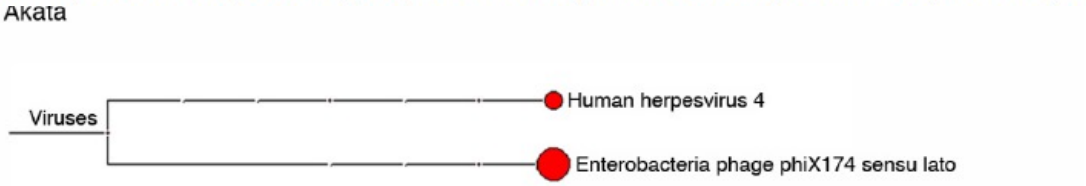
JVI
Journals.ASM.org

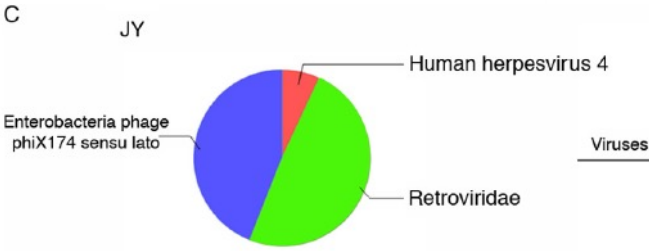
Detection of Murine Leukemia Virus in the Epstein-Barr Virus-Positive Human B-Cell Line JY, Using a Computational RNA-Seq-Based Exogenous Agent Detection Pipeline, PARSES

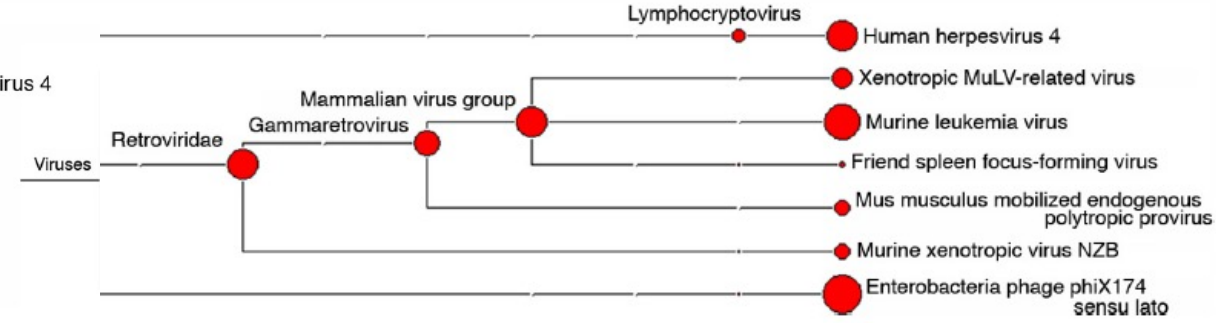
Zhen Lin,[a] Adriane Puetter,[a] Joseph Coco,[b] Guorong Xu,[b] Michael J. Strong,[a] Xia Wang,[a] Claire Fewell,[a] Melody Baddoo,[a] Christopher Taylor,[b] and Erik K. Flemington[a]

Tulane University Health Sciences Center and Tulane Cancer Center, New Orleans, Louisiana, USA,[a] and University of New Orleans, New Orleans, Louisiana, USA[b]









- Endogenous retroviruses (sleeping giants) are reactivated in immune deficient individuals (ME/CFS, CLD, CLL, ASD, HIVAIDS), likely because of dysregulated DNA methylation

- Co-infections, reactivated viruses, GMOs, genetic susceptibilities can create perfect storm of aberrant methylation immune activation (including microglia and inflammation seen in ASD, ME/CFS, other neuroimmune disease and cancer

November 2012

LETTER

doi:10.1038/nature11599

Resurrection of endogenous retroviruses in antibody-deficient mice

George R. Young[1], Urszula Eksmond[1], Rosalba Salcedo[2], Lena Alexopoulou[3], Jonathan P. Stoye[4] & George Kassiotis[1]

Our results shed light onto a previously unappreciated role for immunity in the control of ERVs and provide a potential mechanistic link between immune activation by microbial triggers and a range of pathologies associated with ERVs, including cancer.

Plasmacytoid Dendritic Cells in the Duodenum of Individuals Diagnosed with Myalgic Encephalomyelitis Are Uniquely Immunoreactive to Antibodies to Human Endogenous Retroviral Proteins

KENNY L. DE MEIRLEIR[3] et al., In Vivo, 27:177 (2013

This manuscript claims that SFFV antibodies cross react with human endogenous retroviral proteins like HERV-K env

Monoclonal Antibody 7C10 Does Not recognize primary HERVs (HERV-K)

Are signifcant amounts of protein recognized by SFFV ENV monoclonal antibody used in all of our studies

Could This ENV protein be toxic to PDC Independent of presence of RCR?

NP-7 - mouse line expressing lots of SFFV gp55
MCF-7 human breast line expressing HervK env
7C10 rat monoclonal Antibody against SFFV gp55
6H5 mouse moAB against HERV K env
MCF lysate western with 7c10 did not give same band (ie non specific) as 6H5

The replicative activity of human endogenous retrovirus K102 (HERV-K102) with HIV viremia

Marian P. Laderoute[a,b], Antonio Giulivi[a,b], Louise Larocque[a], Deana Bellfoy[a], Yangxun Hou[a], Hong-Xing Wu[a], Keith Fowke[c], Jun Wu[a] and Francisco Diaz-Mitoma[d]

Results: Both the peptide serology and ddCt qPCR excess ratio methods suggested the activation of HERV-K102 in about 70–80% of HIV viremic cases whereas only 2–3% of normal healthy adults had marginally activated HERV-K102 ($P < 0.0001$). Moreover, by

Conclusions: Our work uniquely suggests the common activation of HERV-K102 with HIV viremia and may be first to directly demonstrate HERV-K102 cDNA production *in vivo*. The potential implications of the induction of HERV-K102 activation and replication for the prevention and control of HIV are discussed.

AIDS 2007, **21**:2417–2424

Hypothesis

Aberrant evolution of the human genome by:

- RCRs generated in current vaccines
- Increased zoonosis of novel retroviruses in human population

Vaccine Schedules=The anti-Hygiene hypothesis

- Sterile environments result lack of educated immune systems

- Vaccination schedules result in anergic immune systems that is the inability to mount an immune response to the antigen
- Reappearance of disease is BECAUSE of inappropriate vaccinations

Review

CellPress

Effects of environmental change on zoonotic disease risk: an ecological primer

Trends in Parasitology, April 2014, Vol. 30, No. 4 205

Agustín Estrada-Peña[1], Richard S. Ostfeld[2], A. Townsend Peterson[3], Robert Poulin[4], and José de la Fuente[5,6]

[1] Department of Animal Pathology, Faculty of Veterinary Medicine, Miguel Servet, 177, 50013-Zaragoza, Spain
[2] Cary Institute of Ecosystem Studies, Millbrook, NY 12545-0129, USA
[3] The University of Kansas Biodiversity Institute, Lawrence, KS 66045-7593, USA
[4] Department of Zoology, University of Otago, Dunedin 9016, New Zealand
[5] SaBio, IREC, Ronda de Toledo s/n, 13071 Ciudad Real, Spain
[6] Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK 74078, USA

Entropy **2013**, *15*, 4748-4781; doi:10.3390/e15114748

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Review

The New Genetics and Natural *versus* Artificial Genetic Modification

Mae-Wan Ho

Institute of Science in Society, 29 Tytherton Road, London N19 4PZ, UK;

Hazards of GMOs

1. Uncontrollable, unpredictable impacts on safety due to the genetic modification process *
   - Scrambling the host genome *
   - Widespread mutations *
   - Inactivating genes *
   - Activating genes *
   - Creating new transcripts (RNAs) including those with regulatory functions *
   - Creating new proteins *
   - Creating new metabolites or increasing metabolite to toxic levels *
   - Activating dormant viruses *
   - Creating new viruses by recombination of viral genes in GM insert with those in the host genome *
2. Toxicity of transgene protein(s) introduced (intentionally or otherwise)
   - Transgene protein toxic *
   - Transgene protein allergenic or immunogenic *
   - Trangenic protein becoming allergenic or immunogenic due to processing *
   - Unintended protein created by sequence inserted may be toxic or immunogenic
3. Effects due to the GM insert and its instability *
   - Genetic rearrangement with further unpredictable effects *
   - Horizontal gene transfer and recombination *
     - Spreading antibiotic and drug resistance *
     - Creating new viruses and bacteria that cause diseases
     - Creating mutations in genomes of cells to which the GM insert integrate including those associated with cancer *
4. Toxicity of herbicides used with herbicide tolerant GM crops *

Chronic Diseases Potentially Associated with Human Retroviral Infection

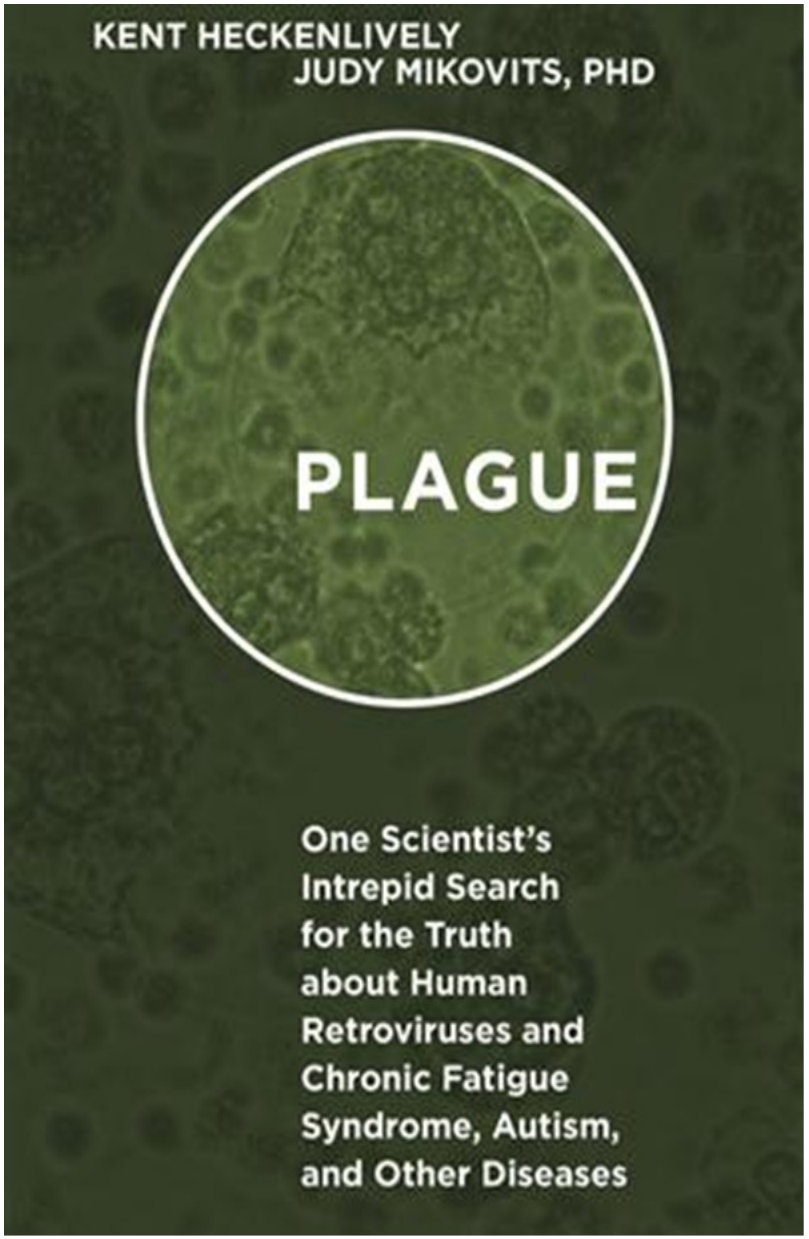
Cancer	Auto-Immune Diseases	CNS
Prostate*	Lupus	ME/CFS*
Breast*	Crohn's*	Gulf War Syndrome*
Non Hodgkin's Lymphoma*	Hashimoto'sThyroiditis*	Autism*
Chronic Lymphocytic Leukemia*	Polymyositis	MS*
Mantle Cell Lymphoma*	Sjogren's syndrome	Parkinson's*
Hairy Cell Leukemia	Bechet's Disease*	ALS*
Bladder*	Primary Billary Cirrhosis*	Lyme Borreliosis Complex (LBC)*
Colorectal		HAND*
Kidney*		
Ovarian*		

* RT Activity, RV sequences or proteins, antibodies to RV proteins

CONCLUSION

Therapies to counteract environmentally induced aberrant gene RCR expression, inflammation immune dysregulation urgently need to be addressed

The best scientist in jail story since Galileo.



How politics corrupts scientific research

Particularly research involving human subjects

"they find what they want to find"

No funding = no associations

Difference between associations found in research associations found in MEDIA