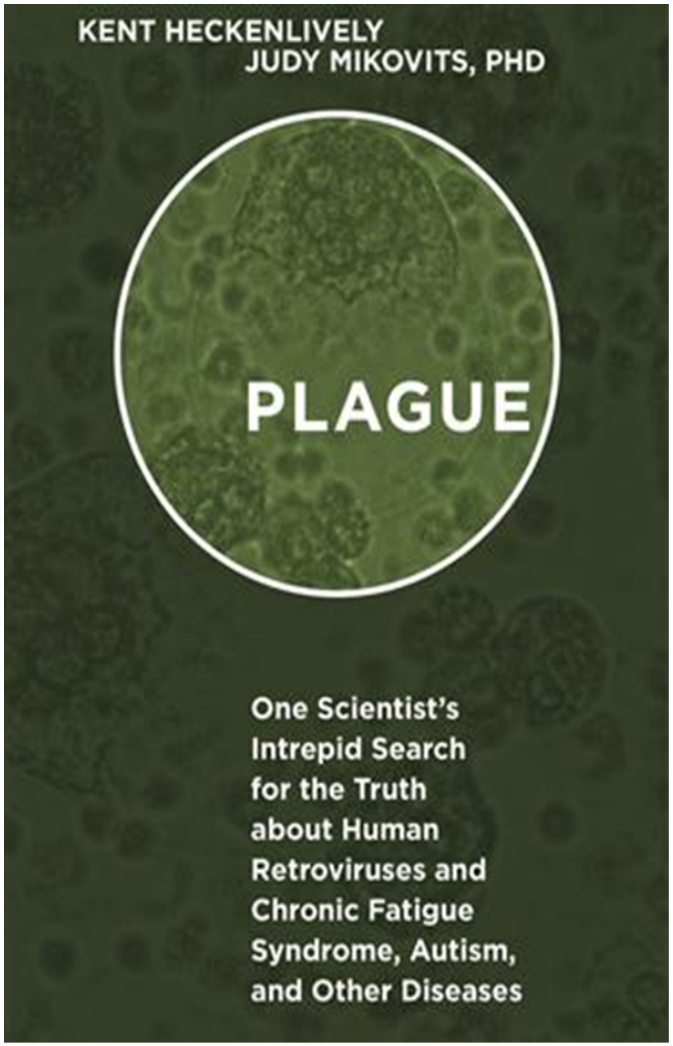
ASD : An Acquired Immune Deficiency!

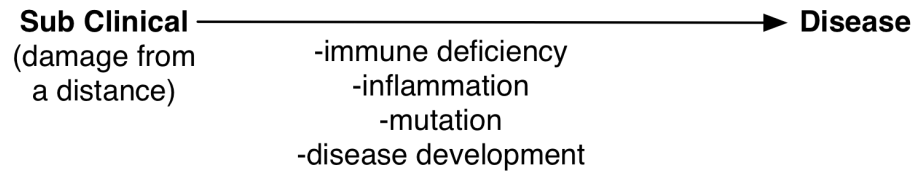


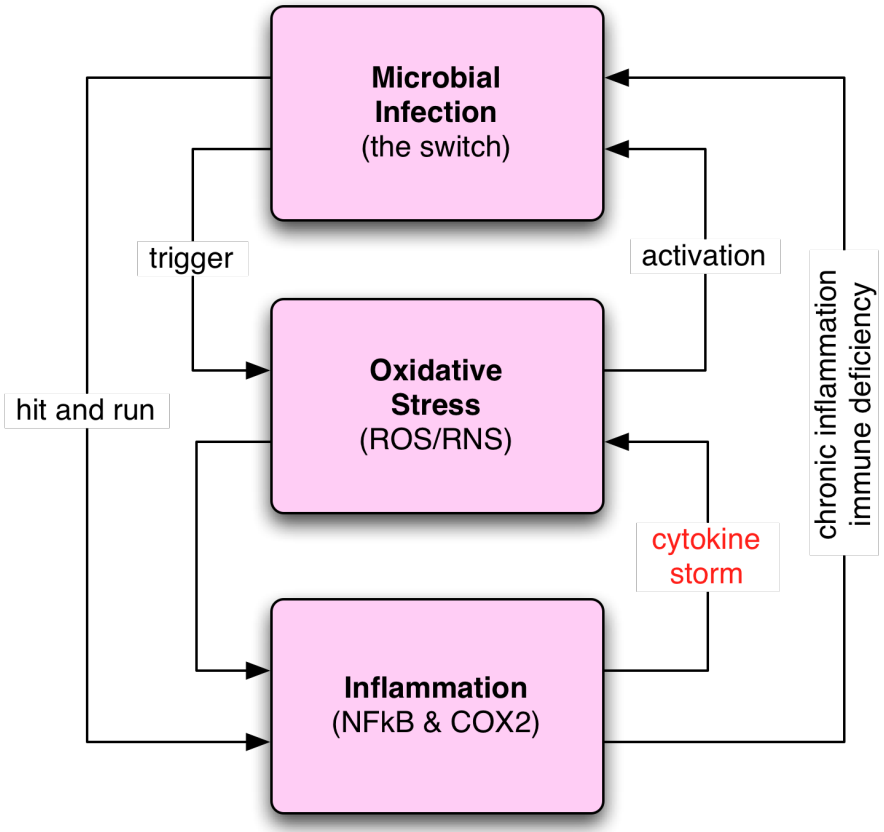
[T]he results suggest that xenograft approaches commonly used in the study of human cancer promote the evolution of novel retroviruses with pathogenic properties.
Retrovirology. 27 March 2013

Emerging Concepts

- Recombination events in animal and human cells can generate families of infectious related gamma retroviruses
- Greatest concern is that they have acquired the ability to infect humans as our data and that of others shows consistently 3-6% in control populations and Human cell lines
- Are XMRV-like sequences and proteins important in human disease pathogenesis?

Key Contributors to Chronic Diseases





Chronic Microbial infections lead to cellular stress and inflammation

Many scientists and doctors familiar with the disease had long suspected a retrovirus, an organism rife in nature that invades the immune system and central nervous system, as seen in AIDS. Once a retrovirus has infected an organism, it commandeers the organism's genetic machinery, turning a once-healthy cell into a retroviral powerhouse that spreads the infection to more cells in an irreversible cascade.

Hilary Johnson Discover Feb 2013 Chasing the Shadow Virus

Pathways of Retrovirus Elicited Pathogenesis

- Inflammation / hormone regulation
- Highly elevated ROS / RNS
- Immune deficiency
- Epigenetics — change in gene expression without a DNA change
- Insertional mutagenesis
- RVs can be vertically transmitted
- RVs can recombine with aberrantly expressed endogenous RVs creating RCRs

Clinical features of patients infected with human retroviruses:

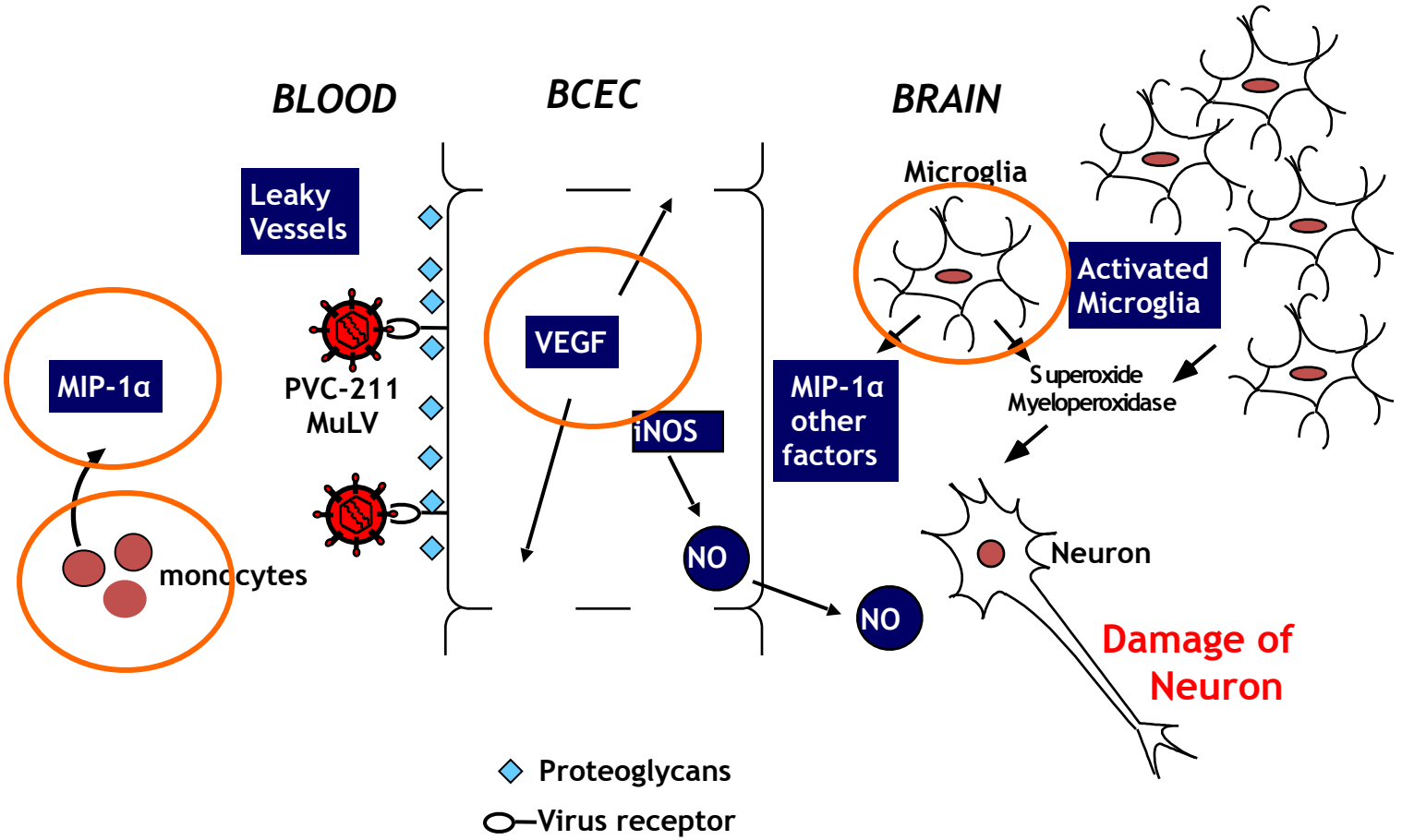
- Reduced Natural Killer (NK) cell numbers and NK cell cytotoxicity
- Abnormal $CD4^+$ and $CD8^+$ T cell numbers and ratio
- Reduced T cell response to antigens
- Presence of auto antibodies
- Alteration in cytokine profile
- Hyperactivation of T cells
- Decreased Antibody dependent cellular cytotoxicity (ADCC)

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*	
Colorectal		
Kidney*		
Ovarian*		

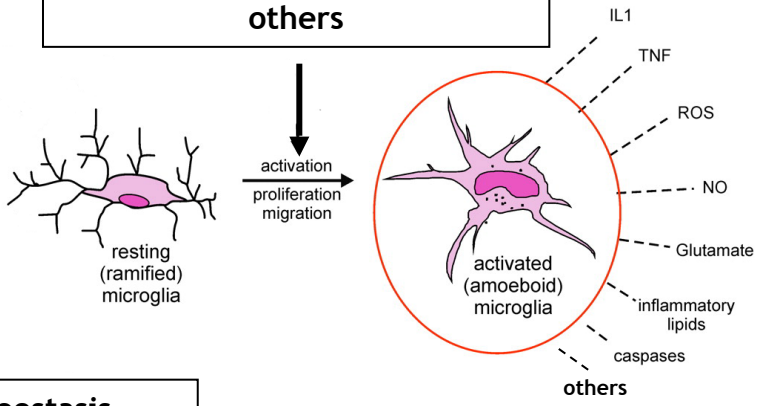
* RT Activity, RV sequences or proteins, antibodies to RV proteins

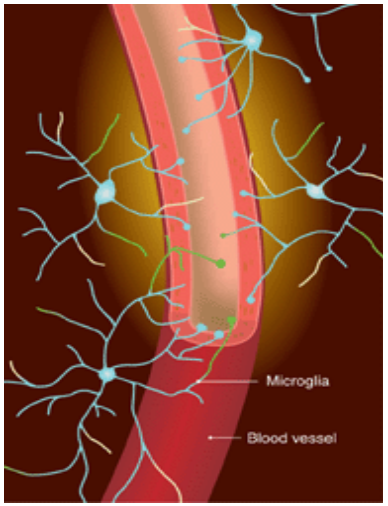
Putative Model for the Induction of Neurodegeneration by Gamma Retroviruses



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

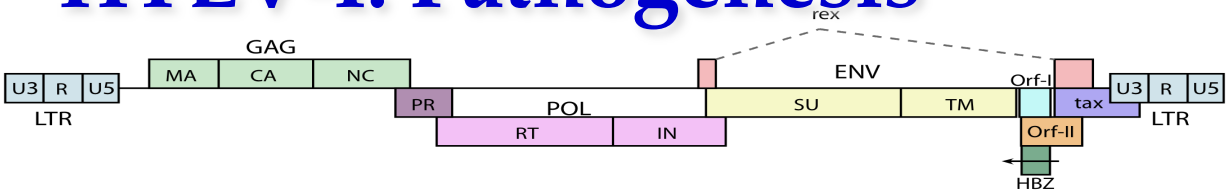
Damage
Neurotoxins, phagocytosis of normal neurons, apoptosis

Neurodegenerative disorders

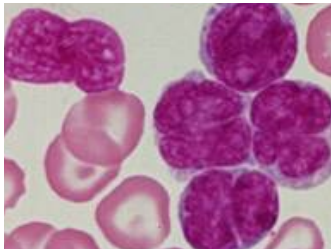
- Parkinson's disease
- Alzheimer's disease
- Multiple sclerosis

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

Detection of serum reverse transcriptase activity in patients with ALS and unaffected blood relatives

A.J. Steele, PhD; A. Al-Chalabi, PhD, FRCP; K. Ferrante, BA; M.E. Cudkowicz, MD, MSc; R.H. Brown, Jr., MD, DPhil; and J.A. Garson, MD, PhD

NEUROLOGY 2005;64:454–458

Quantification of reverse transcriptase in ALS and elimination of a novel retroviral candidate

Neurology® 2008;70:278-283

That Novel Candidate was XMRV

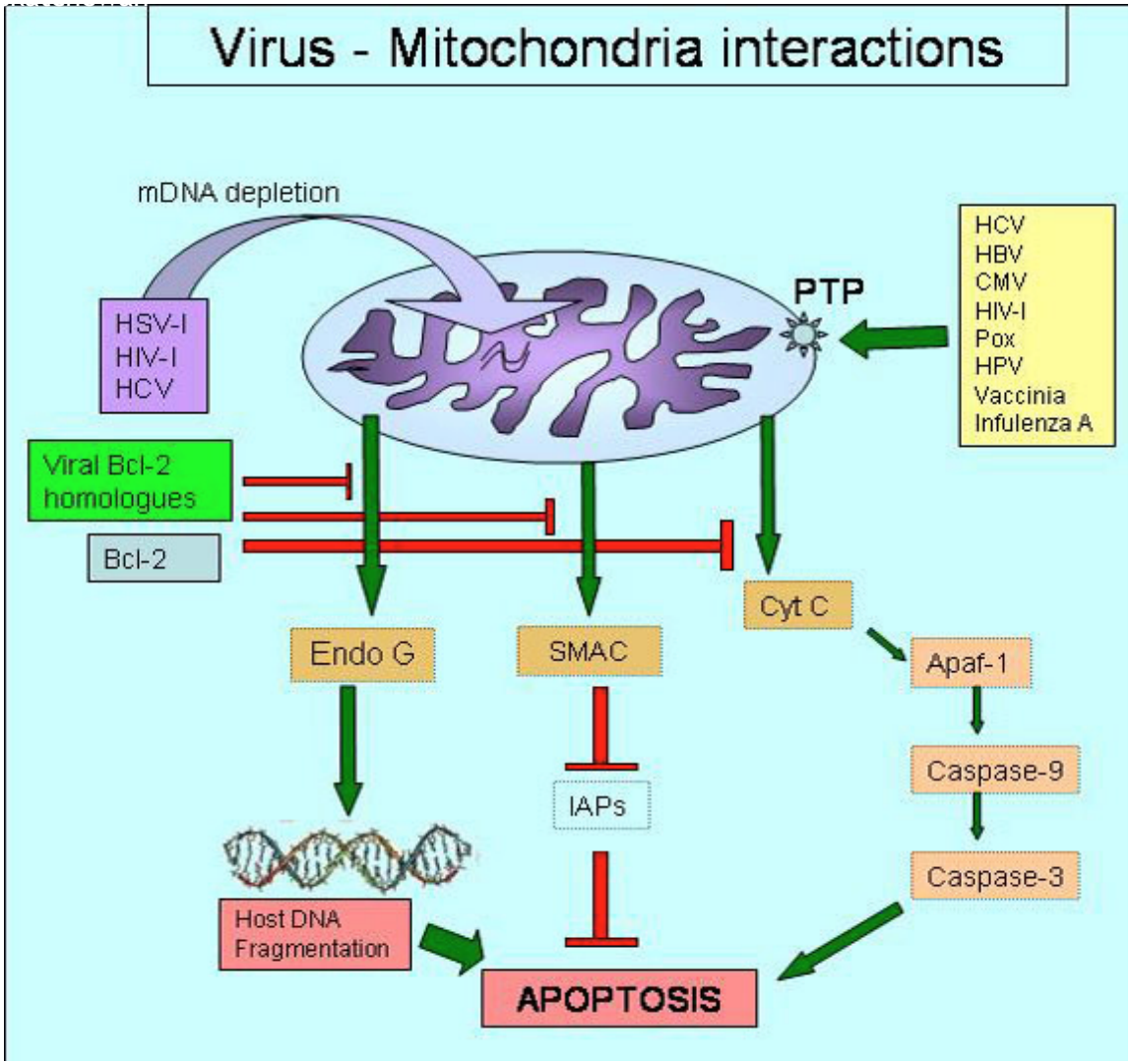
In Chronic Diseases Viruses Seldom Come Alone

Table 1. Mechanisms of Interactions between HIV-1 and Coinfecting Viruses

Mechanisms	Viruses
Immunoactivation	HCV, HSV-2, CMV, EBV, HTLV-2[a]
HIV-1 *trans*-activation	HSV-2, HTLV-1, JCV[a]
Abnormal production of chemokines	HTLV-1, HHV-6, HTLV-2, MV, GBV-C
CD4, CCR5, or CXCR4 downregulation	HHV-7, GBV-C
Expression of virokines and viroceptors	CMV, HHV-6, HHV-7
Blockage of CD4 T cell cycle	MV
Modulation of cytokine signaling	EBV, adenovirus
Inhibition of apoptosis	CMV, EBV
Aberrant activation of autologous complement	HHV-6, HHV-7
MHC downregulation	CMV, HHV-6, HHV-7

War and Peace between Microbes: HIV-1 Interactions with Coinfecting Viruses: Cell Host & Microbe 6, November 19, 2009 A. Lisco, C Vanpouille, & L Margolis

Recent advance in genomic technologies have identified ~1000 nuclear genes that regulate mitochondrial function …



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

PITFALL : ability of these viruses to spread to uninfected cells through

November 2012

LETTER

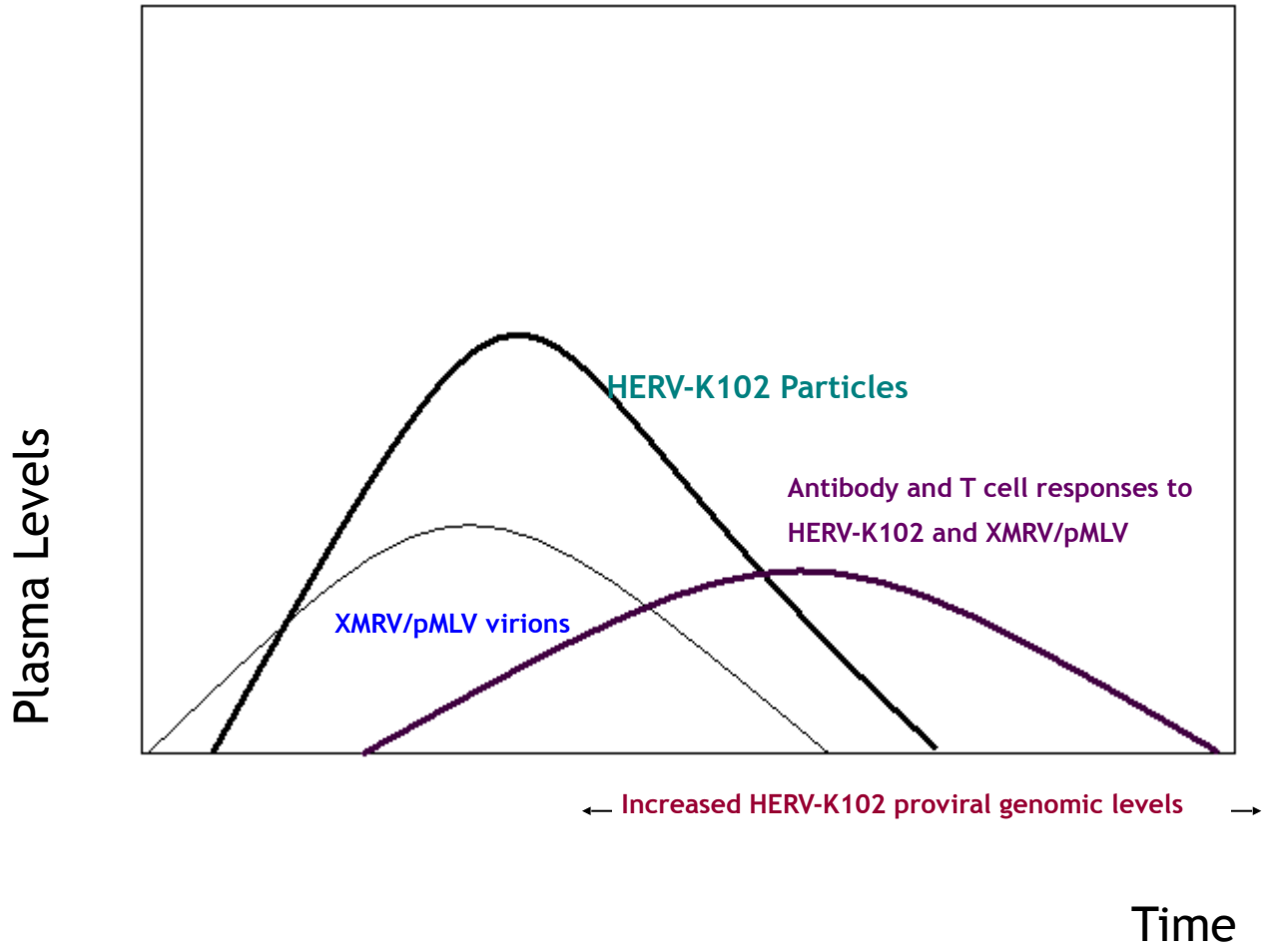
doi:10.1038/nature11599

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.

Presence of One Retrovirus Elicits expression of endogenous Retroviruses!!



With the exception of the initial lag (perhaps one or two days), there would not be an inverse correlation of HERV-K102 ddCt ratios with XMRV/pMLV viremia. Instead, as we found for HIV-1, if there is little or no detectable viremia, then HERV-K102 particles (inferred from the ddCt ratio) are not made. Thus, we expect to show a correlation of HERV-K102 particle production with active viremia with XMRV/pMLV, but not with levels of either.

- 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

ARE MLV Sequences and proteins in the US blood supply? (NYAS March 2011)

What are the detection rates in 1000 blood donors using these serological assays?

Analyzed to CA, TM and SU

SU is the most reactive antigen (subjects vs donors)

- 4% reactive
- 13% 'indeterminate'
  - Values above cut-off, but also reactive to neg ctrl antigen

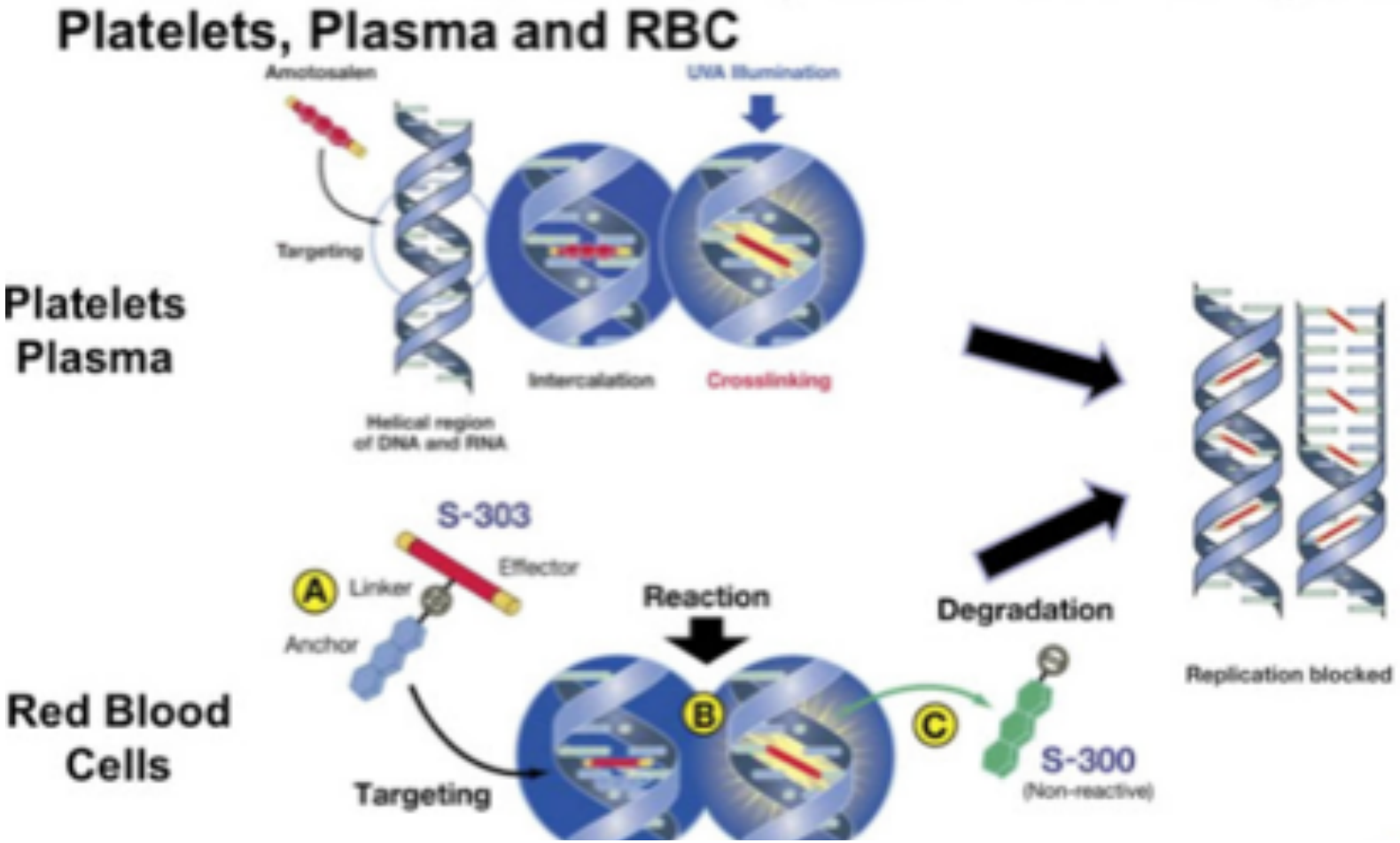
Technologies are available to decontaminate the blood supply and the vaccines: Cerus

by Intercept Blood System is consistent with results for other human Retroviruses

Organisms	Extent of Inactivation (log_{10} reduction)		
	Platelets[a]	Plasma[b]	RBC[c]
HIV-1 (cell associated)	>6.1	>6.1	>5.9[d]
HIV-1 (cell free)	>6.2	>6.8	NA
HTLV-I	4.7	≥4.5	>4.2[d]
HTLV-II	5.1	>5.7	> 5.1[d]
XMRV & MLV-Related virus	**>4.0[e]**	**NA**	**>4.0[e]**

NYAS March 29 2011, Mikovits

2014 December 1 FDA Approval



Summary/Conclusions

NYAS March 2011

- Data suggest there are different strains of Gamma Retroviruses that can infect humans
- Assays that capture the variation of these viruses in the blood supply are the best i.e. Serology and transmission
- Technologies can inactivate infectious strains of XMRV in Blood Components
- New Disease associations include leukemia, lymphoma and the platelet disorder, ITP

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 *

Review

CellPress

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

Agustín Estrada-Peña[1], Richard S. Ostfeld[2], A. Townsend Peterson[3], Robert Poulin[4], and Jo Trends in Parasitology, April 2014, Vol. 30, No. 4 205

[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

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

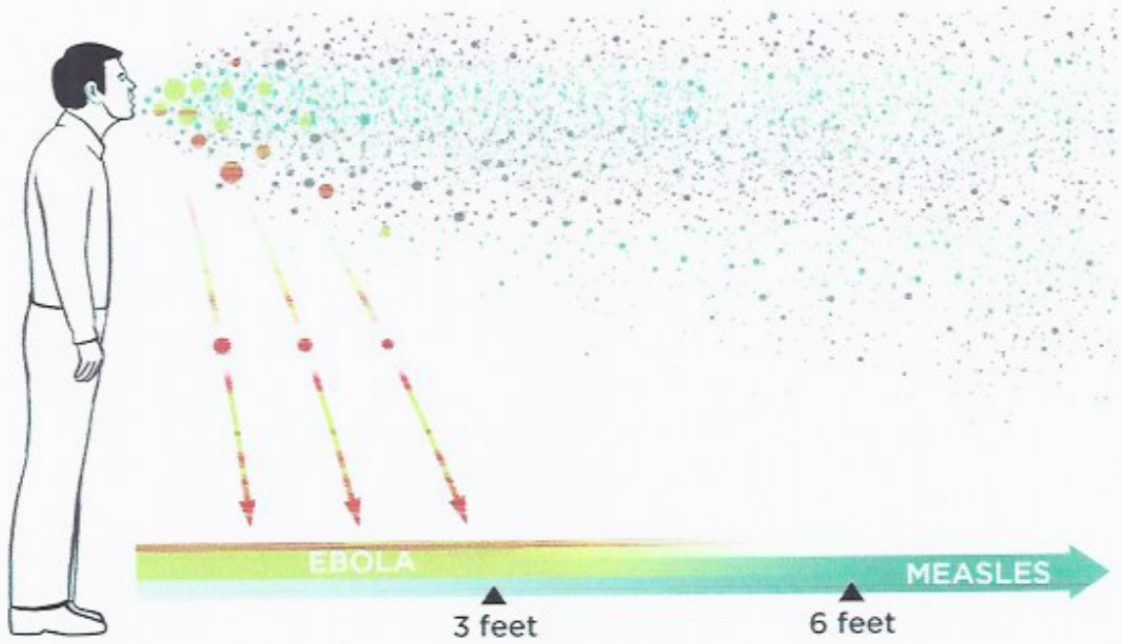
Vaccine schedules compound damage

- 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
- Toxic components exacerbate immune dysfunction resulting in aberrant expression of host endogenous RVs
- Reappearance of disease is BECAUSE of inappropriate vaccinations and the toxic components contained in them

Ebola In The Air: What Science Says About How The Virus Spreads

DECEMBER 01, 2014 12:29 PM ET

MICHAELEEN DOUCLEFF



Viruses can spread through the air in two ways: inside large droplets that fall quickly to the ground (red), or inside tiny droplets that float in the air (gray). In the first route, called droplet transmission, the virus can spread only about 3 to 6 feet from an infected person. In the second route, called airborne transmission, the virus can travel 30 feet or more.

Adam Cole/NPR

Here's an Ebola puzzle for you: If the virus isn't airborne, why do doctors and nurses need to wear full protective suits, with face masks, while treating patients?

Conclusions

Aberrant evolution of the human genome by:

- Replication competent retroviruses generated laboratories in current vaccines and cell cultures
- Increased zoonosis of novel retroviruses in human population from animal populations.
- That means GMOs and toxins in animals result in compromised immune systems and the xpression of endogenous viruses ..eg Bovine leukemia virus
- These retroviruses CAN and have been shown to infect human cells and like HTLV, HIV are passed in milk and other fluids
- The blood supply is contaminated and more than likely the vaccines are contaminated as is food supply (milk)

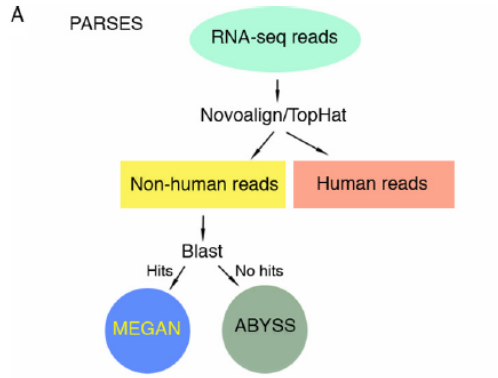
New Technologies Reveal the presence of Multiple Gamma retroviruses in a single Human Cell Line

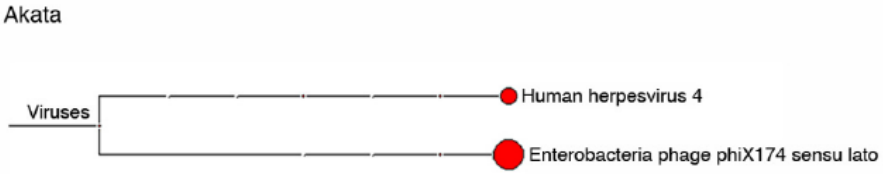
JVI
Journals.ASM.org

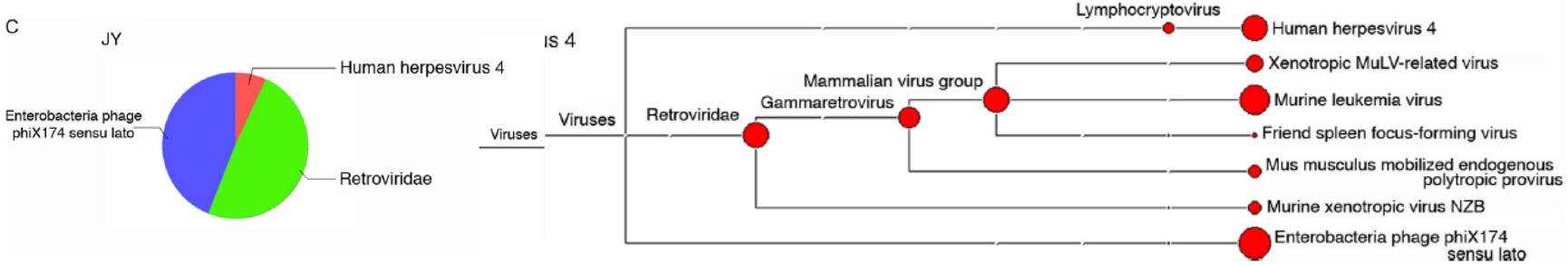
Detection of Murine Leukemia Virus in the Epstein-Barr Virus-Positive Human B-Cell Line JY, Using a Computational RNA-Seq-Based Exogenous Agent Detection Pipeline, PARSES

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]







RESEARCH ARTICLE

Prevalence and Characterization of Murine Leukemia Virus Contamination in Human Cell Lines

Cord C. Uphoff[1]*, Sandra Lange[1], Sabine A. Denkmann[1], Henk S. P. Garritsen[2], Hans G. Drexler[1]

1 Department of Human and Animal Cell Lines, Leibniz Institute DSMZ—German Collection of Microorganisms and Cell Cultures, Braunschweig, Germany, **2** Institute for Clinical Transfusion Medicine, Municipal Hospital, Braunschweig, Germany

New Technologies Opportunities for PREVENTION

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:

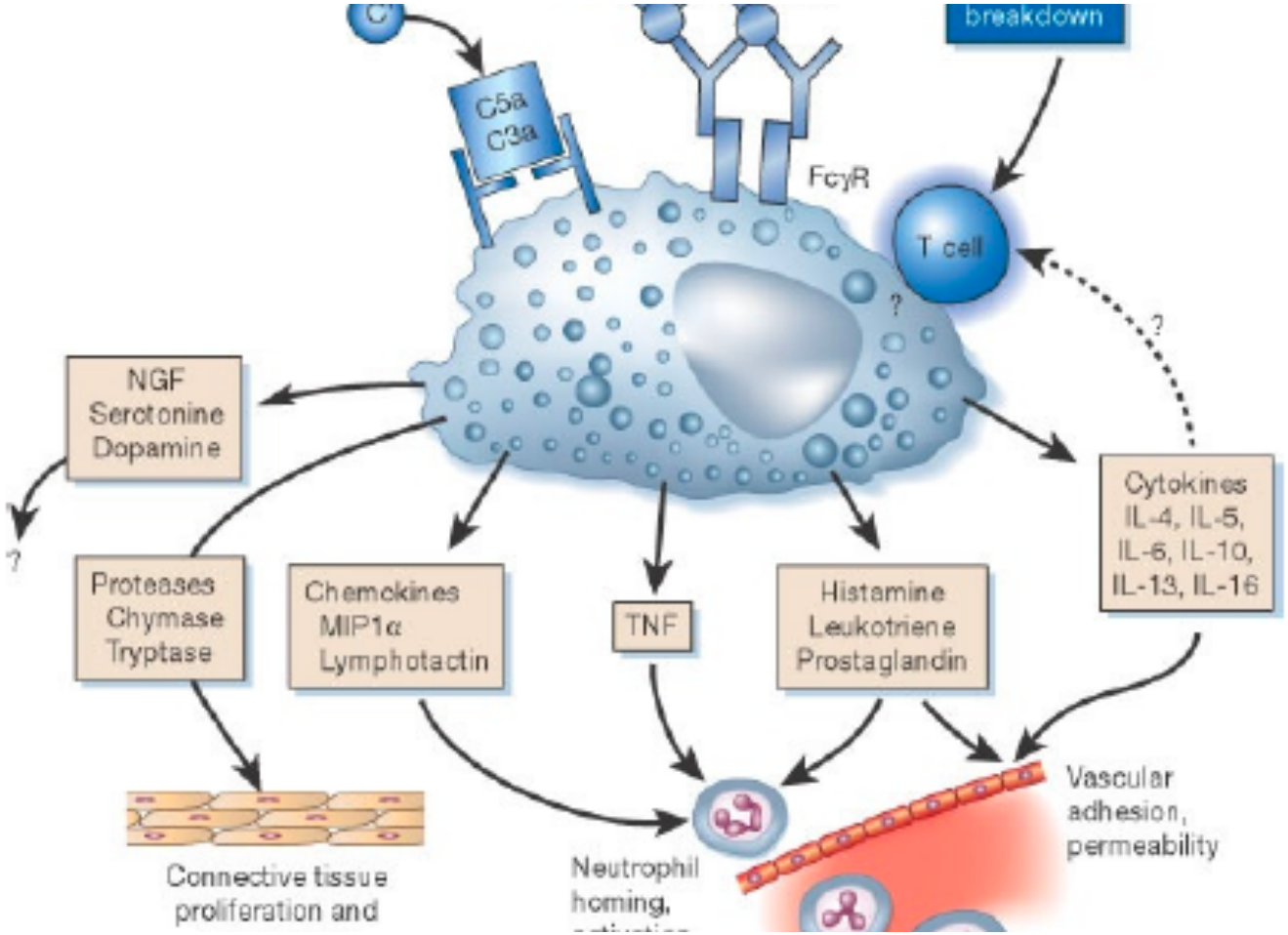
- Abnormal autosomal dominant Variant was found in SCN4A gene that is likely a pathological mutation
- Pathological mutations found in two other patients also with multiple functional conditions (ME/CFS)

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

Mast Cell as an Amplifier of Autoimmune Disease



ARVs provide therapeutic benefit in some patients with autoimmune, neuroimmune disease and Cancer

- Multiple Sclerosis
- Cancer

Retroviruses have been implicated in the etiology of various autoimmune diseases and cancer. Evidence for pathogenic role in triggering or maintenance of autoimmunity in these diseases includes: the presence of antibodies which are cross reactive with retroviral gag and envelope: detection of retroviral antigens in un-manipulated tissue/plasma; the occurrence of ME/CFS conditions in HIV patients and HTLV-1 associated disorders. Our knowledge of the overlap in these diseases of "acquired immune dysfunction, chronic inflammatory and autoimmune diseases was a basis for our original hypotheses and continue to be a basis for our research in ME/CFS.

Lessons Learned from HIV AIDS

Immune reconstitution inflammatory syndrome: the trouble with immunity when you had none

Daniel L. Barber, Bruno B. Andrade, Irini Sereti and Alan Sher

Abstract | Some individuals who are infected with HIV rapidly deteriorate shortly after starting antiretroviral therapy, despite effective viral suppression. This reaction, referred to as immune reconstitution inflammatory syndrome (IRIS), is characterized by tissue-destructive inflammation and arises as $CD4^+$ T cells re-emerge. It has been proposed that IRIS is caused by a dysregulation of the expanding population of $CD4^+$ T cells specific for a co-infecting opportunistic pathogen. Here, we argue that IRIS instead results from hyper-responsiveness of the innate immune system to T cell help, a mechanism that may be shared by the many manifestations of IRIS that occur following the reversal of other types of immunosuppression in pathogen-infected hosts.

Date: August 31, 2011 8:24:00 PM PDT
To: "Glynn, Simone (NIH/NHLBI) [E]" <glynnsa@nhlbi.nih.gov>
Cc: Frank Ruscetti <fwruscetti@gmail.com>
Subject: Re: SRWG-lab subgroup

That's impossible
I have IRB protected data that I cannot even access until the 6th. I told that to Graham yesterday and he indicated that was fine. Given the complexities and limitations of this study, many of which were not recognized at the time the (flawed) experimental design was agreed upon, to have one day to agree upon a manuscript, a holiday at that, is totally unacceptable. This is NOT good science or the appropriate process. What is the rush?
Afraid the truth??? how many of these viruses were introduced into the human population and are now threatening a lot more than the blood supply ??!because a few declared it "impossible" 40 years ago and JC himself was the most vociferous!
how many XMRVs??
I am sending this to only Simone and Frank because I will make this rush a public relations nightmare for the entire US govt..
I have integration data and variants of many new strains!! Did those arrogant SOBs introduce these into humans and now are trying to cover it up??
And then pedigree the negatives with a test with a cutoff so high it would not find a willing roman in a whore house???
Wonder if anyone will listen to a press conference from me??Asking how many new recombinants from Vaccines? From lab workers?? doctors?
The first ever contagious Human retrovirus???? Spread like mycoplasma?? Are you kidding me???
It happened once!!! How many xenograft cell lines were created?? How many vaccines contained mouse tissue??
These sick people lost their entire lives and this travesty of justice will not be carried out at their expense.. Not again
If we have to write and publish online a dissenting opinion, we will and I will not coauthor any paper that misrepresents our findings..
Not will our data be included .. You can simply say we all found nothing ..totally expected ANC we'll prove them all wrong.
Our assays may not be sensitive or reproducible given the complexity and lack of knowledge of reservoirs etc
Nothing about these data say anything about Lombardi et al of Lo et al
Except that their are likely many strains of XMRVs and only God knows the impact on chronic disease but nothing about this study says anything about our original discoveries
And if this is rushed to print without a fair and balanced discussion of its limitations, I will spend every minute of my life exposing the fraud that has been perpetrated against this patient population.
Judy Mikovits

Thank You

May 23, 2015

Judy A. Mikovits, PhD
MAR Consulting Inc., Carlsbad CA
www.marconsultinginc.com

jamikovits@me.com

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

An Unlikely Driver of Evolution: Arsenic

Around 11,000 years ago, humans first set foot in **the driest place on Earth**. **The Atacama Desert** straddles the Andes Mountains, reaching into parts of Chile, Peru, Bolivia and Argentina. Little rain falls on the desert — some spots haven't received a single drop in recorded history.

But the people who arrived at the Atacama managed to turn it into a home. Some Atacameños, as they are known today, fished the Pacific. Others hunted game and herded livestock in the highlands. They mummified their dead, decorating them with ceremonial wigs before leaving them in the mountains. Those mummies reveal a hidden threat in the Atacama. When **scientists analyzed the hair in 7,000-year-old mummy wigs, they discovered high levels of arsenic**. Through their lives, the Atacameños were gradually poisoned.

Arsenic can poison people today through exposure to pesticides and pollution. But arsenic is also naturally present in the water and soil in some parts of the world. The Atacama Desert, sitting on top of arsenic-rich volcanic rock, is one of them. The concentration of arsenic in Atacama drinking water can be 20 times higher than the level considered safe for human consumption.

Now a team of scientists has discovered that the arsenic of the Atacama Desert didn't just make people sick. It also spurred their evolution.

In a new study in the journal ***Molecular Biology and Evolution***, researchers report that, over the years, the Atacameños became more resistant to arsenic, thanks to natural selection. It is the **first documented case of natural selection in humans for a defense against an environmental toxicant**.