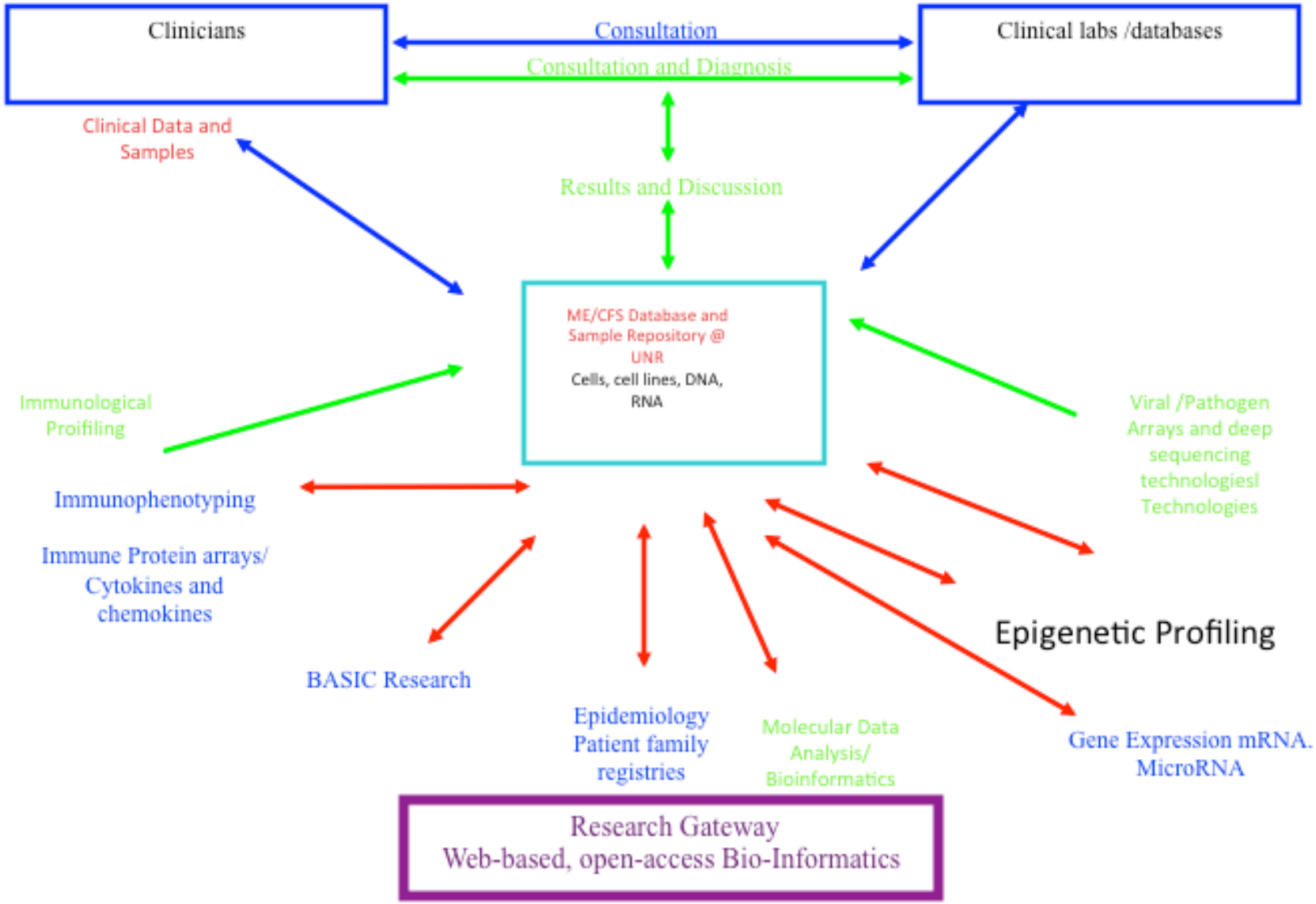
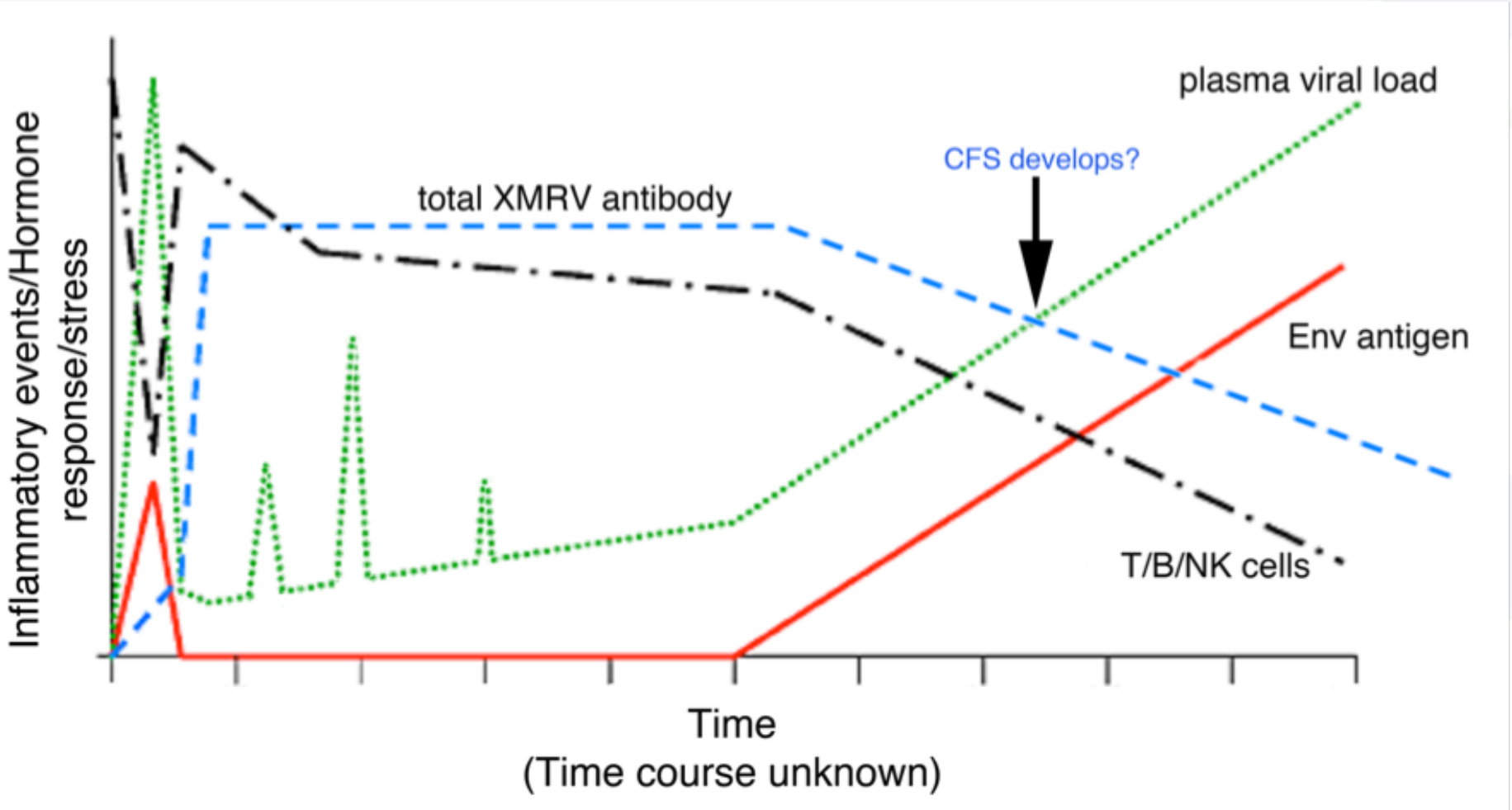
Systems Biology Approach to Chronic Disease.. 2007

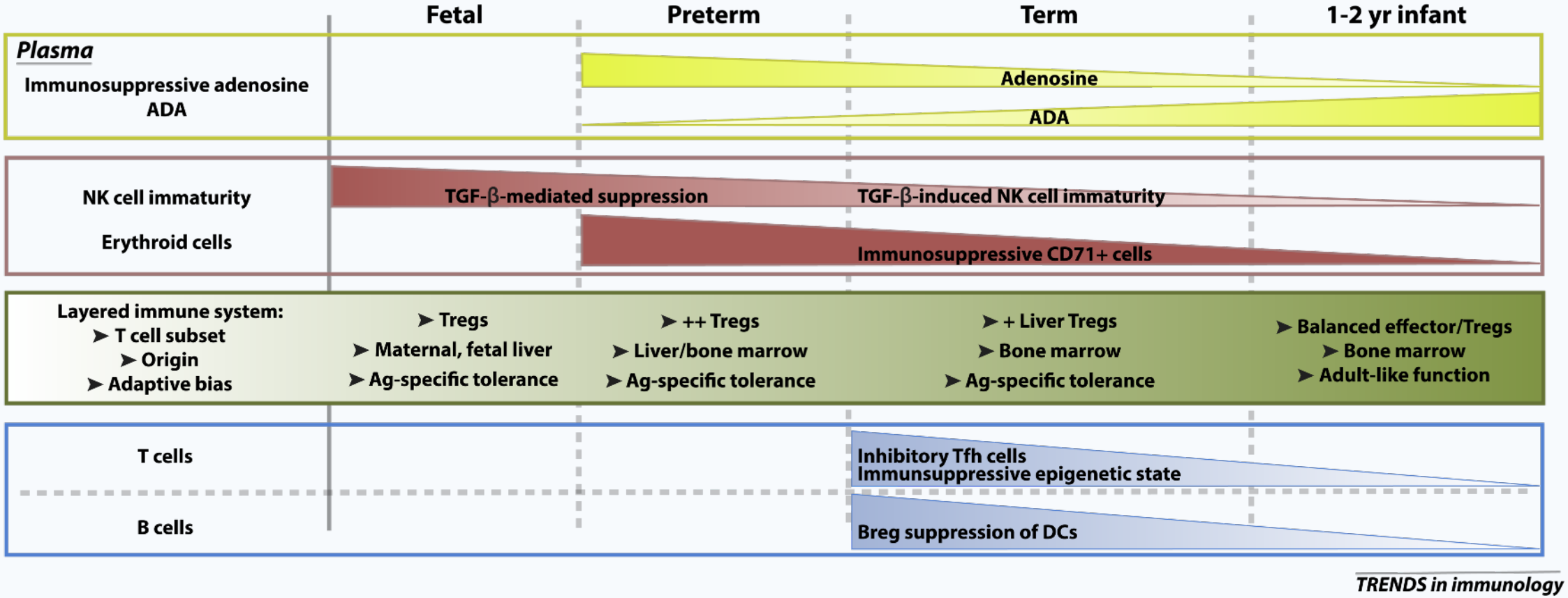


HYPOTHESIS of XMRV INDUCED PATHOGENESIS:
Chronic infection with XMRV may lead to an Immune Deficiency



Xenotropic Murine Leukemia Virus Related Virus (XMRV): Current Research, Disease Associations, Therapeutic Opportunities (Future Medicine, Therapy, Sept 2010)

Immunity is not static: it changes with age; many unique features in early life



The Brain and The Immune System are inextricably linked from Conception

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

enyous Biomed

GENETIC SUSCEPTIBILITIES

Genetics are extremely important in Vaccine Injury

Multifactorial: subsets of genes

- Diagnostics: Multiplex technologies genetic, epigenetic and protein signatures of Disease

    Ex: Courtagen, Oncotype DX

Channelopathies: SCN4A..SCN1A

Immunity Genes: RNASEL

Methylation: MTHFR, MeCP2, IGF-1

Detox: CYP p450

2008: Footprints of retroviral infection!

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

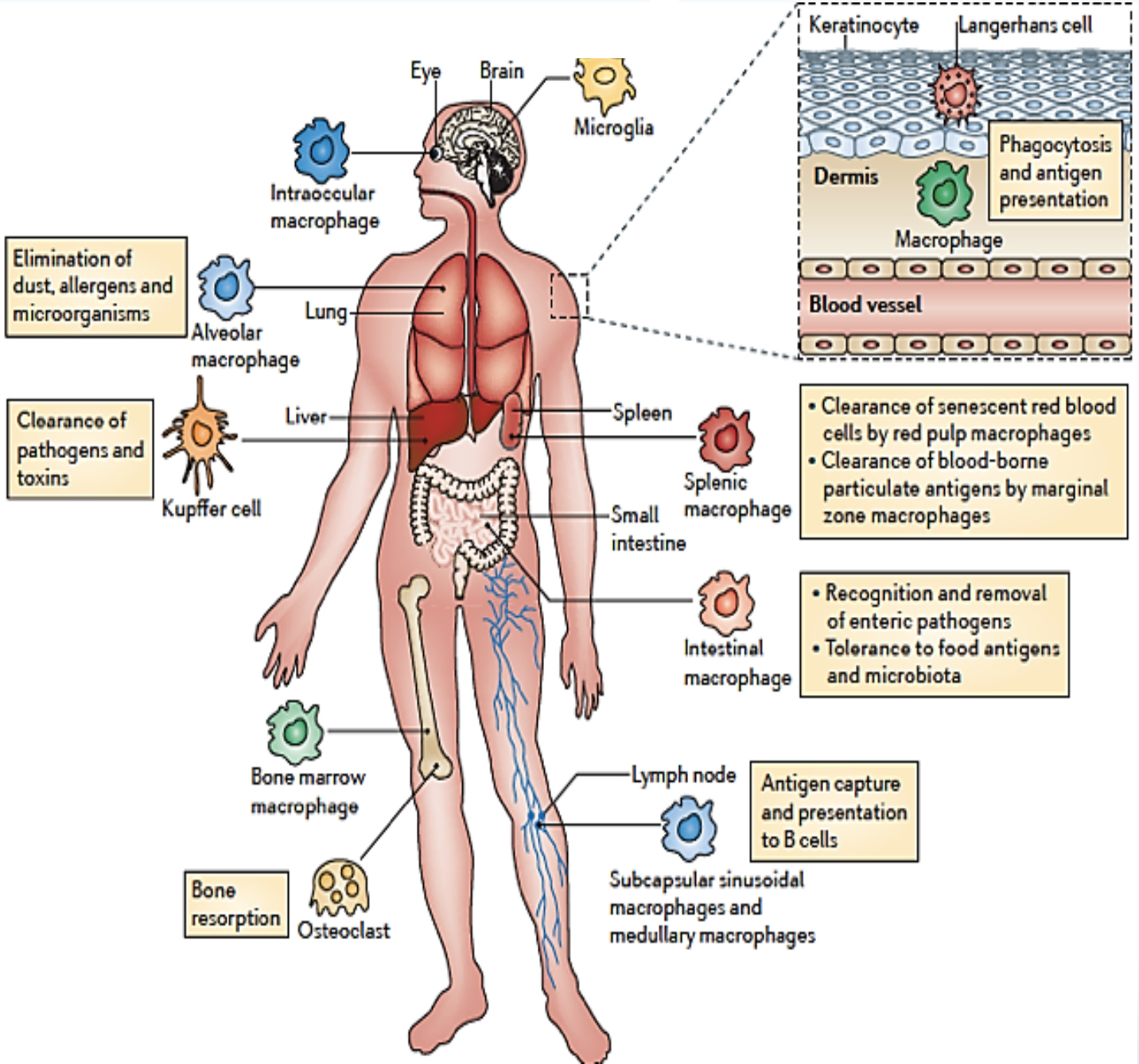
Dysregulated Cytokine/Chemokine Production plasma from ME/CFS patients

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

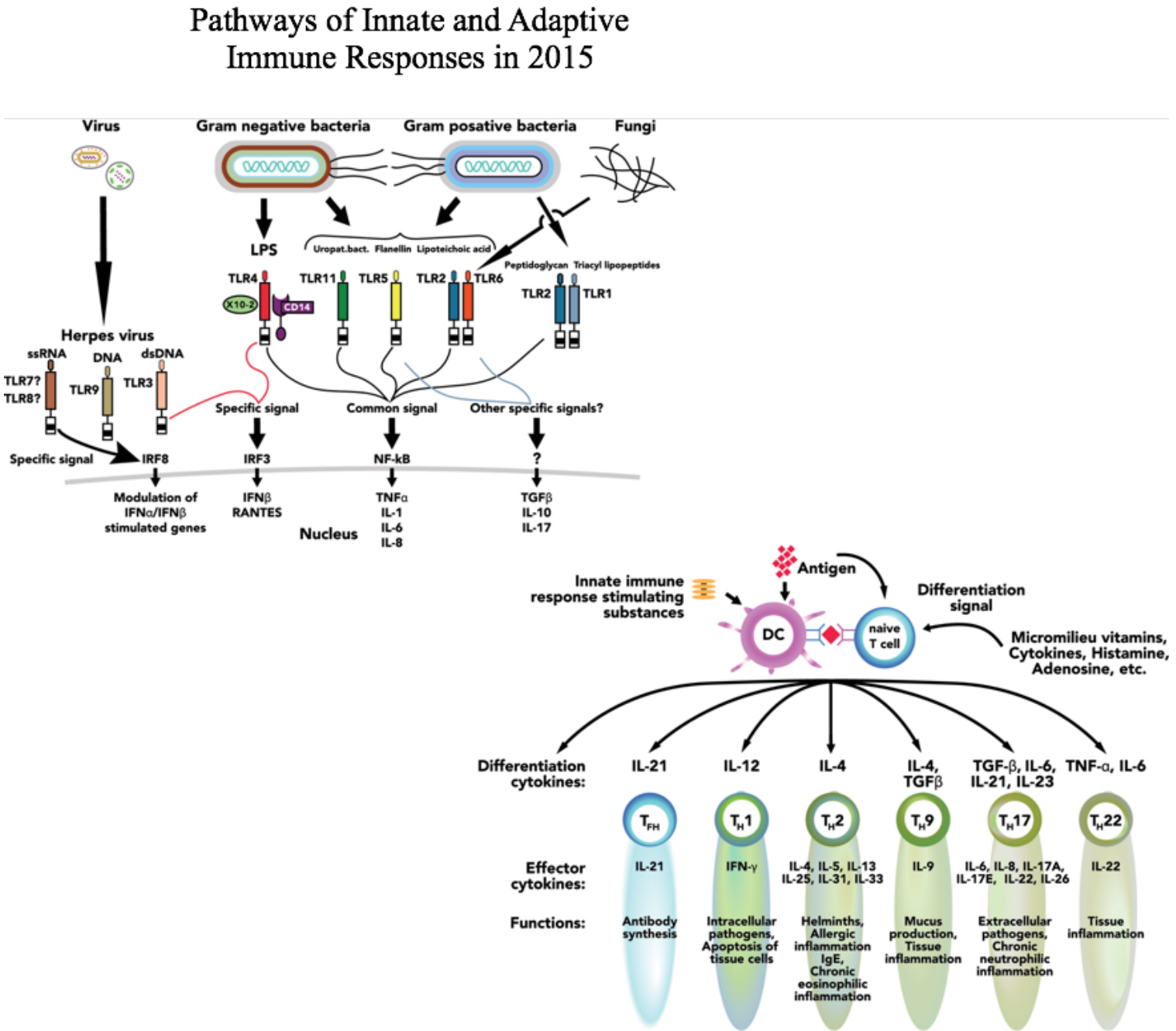
- **Express** Purinergic Receptors:
- **P2XR and P2YR**.
- **Express Cannabinoid Receptors**
- **CB1 & CB2**

Tissue Macrophages perform Key Homeostatic Functions Modulated by

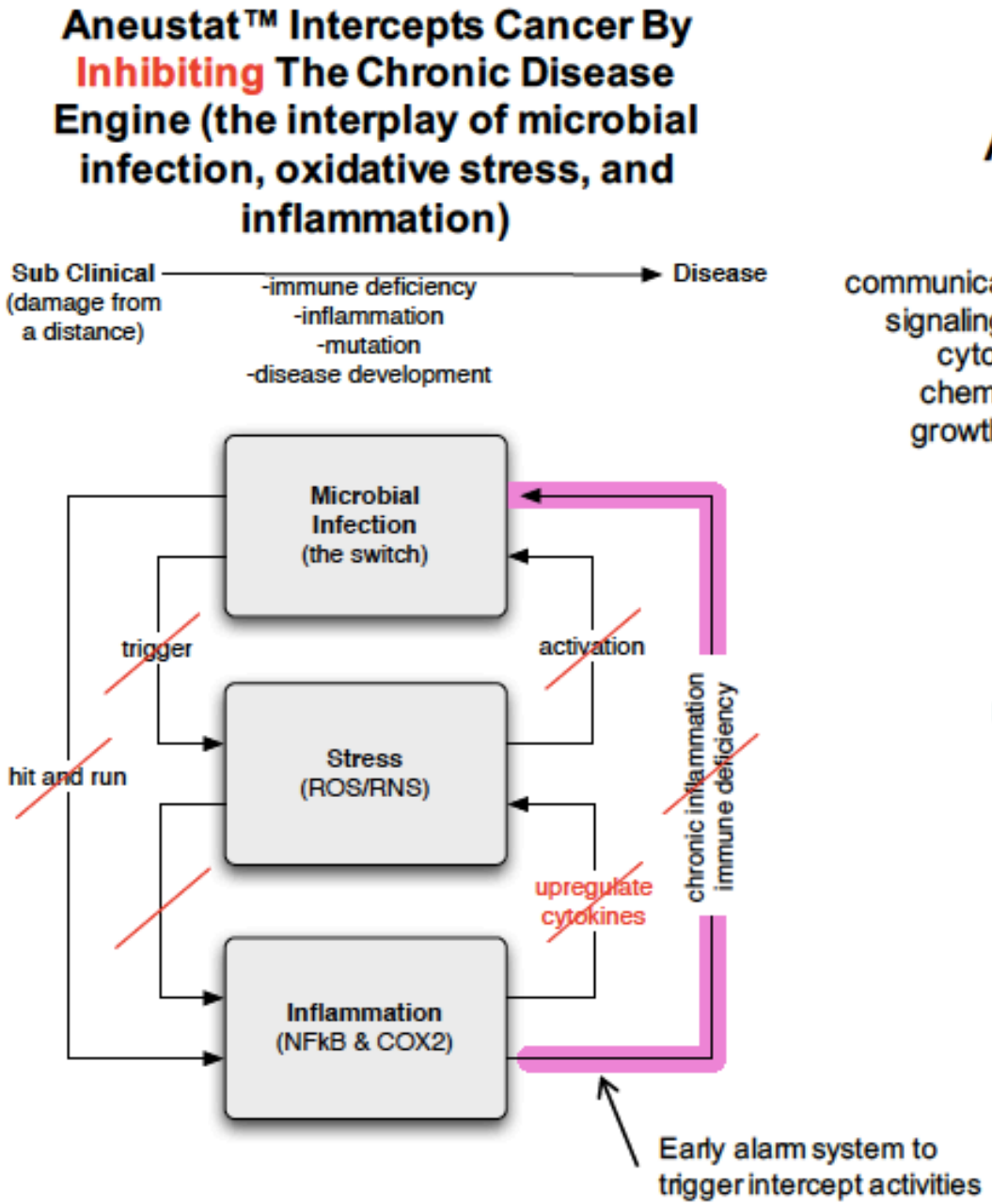
- **Cannabinoids**
- **GcMAF**
- **Suramin**
- **Ivermectin**
- **Vitamin C**
- **DMG**
- **Decitibine (Vidaza)**
- **Peptide T**

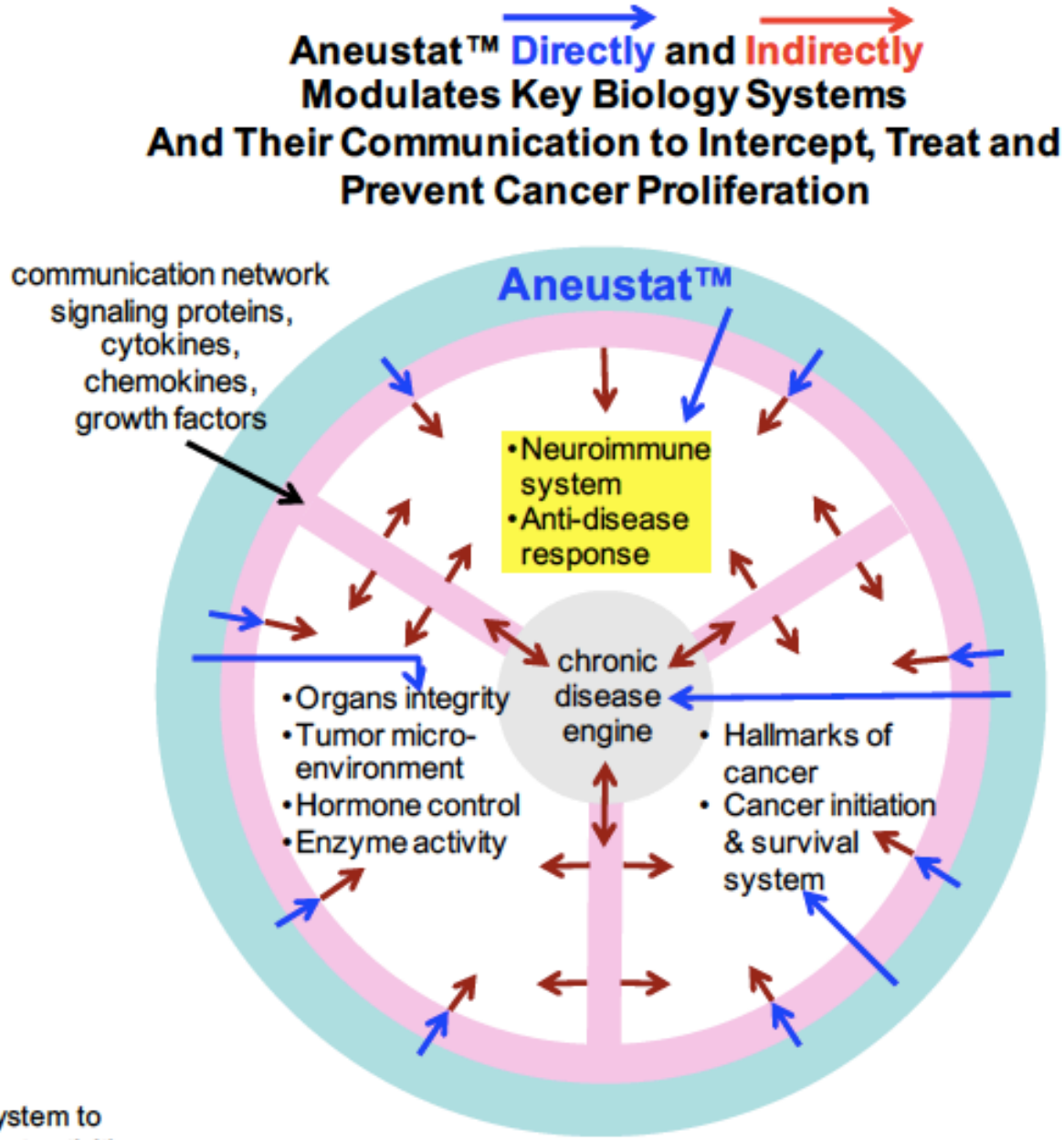


In 1978 Frank Ruscetti discovered T-cell growth factor (IL2) enabling the isolation of HTLV1

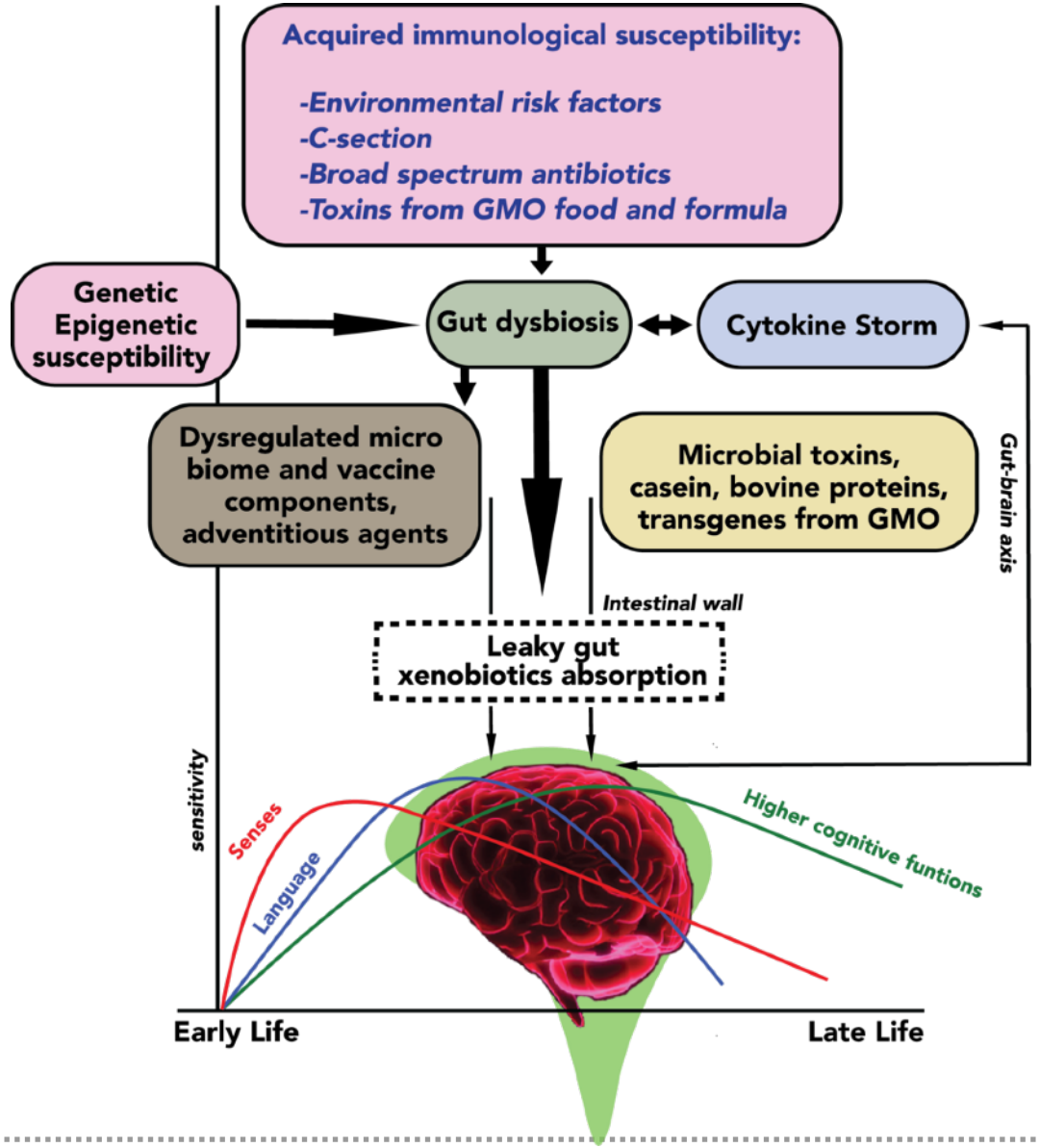


FOOD AS MEDICINE





Inflammatory Insults on Genetic and Epigenetic Susceptible Individuals Results in Chronic Disease



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.

EPIGENETICS: ALL ON TOP OF THE GENES

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

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 *

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

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ISSN 1099-4300

www.mdpi.com/journal/entropy

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;

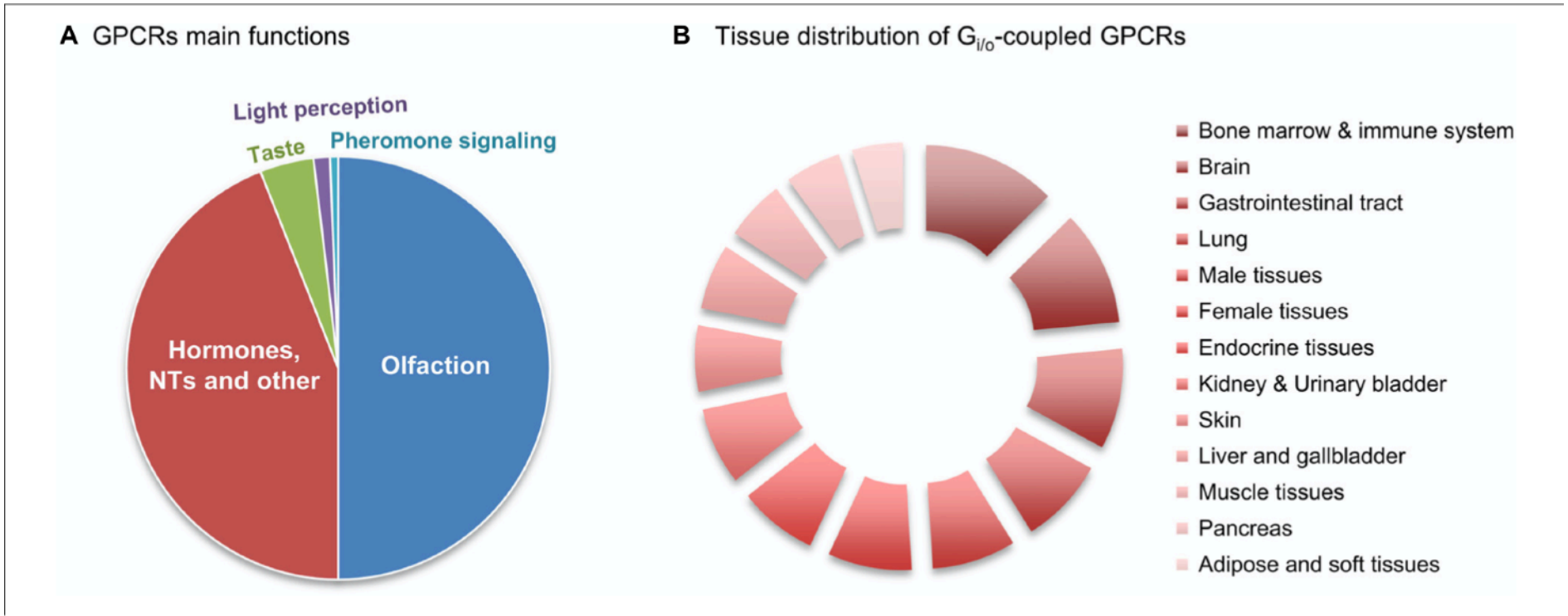
VIRUSES/POSIONS
Lack of Minerals, Essential Amino acids, Phytocannabinoids

frontiers in Aging Neuroscience

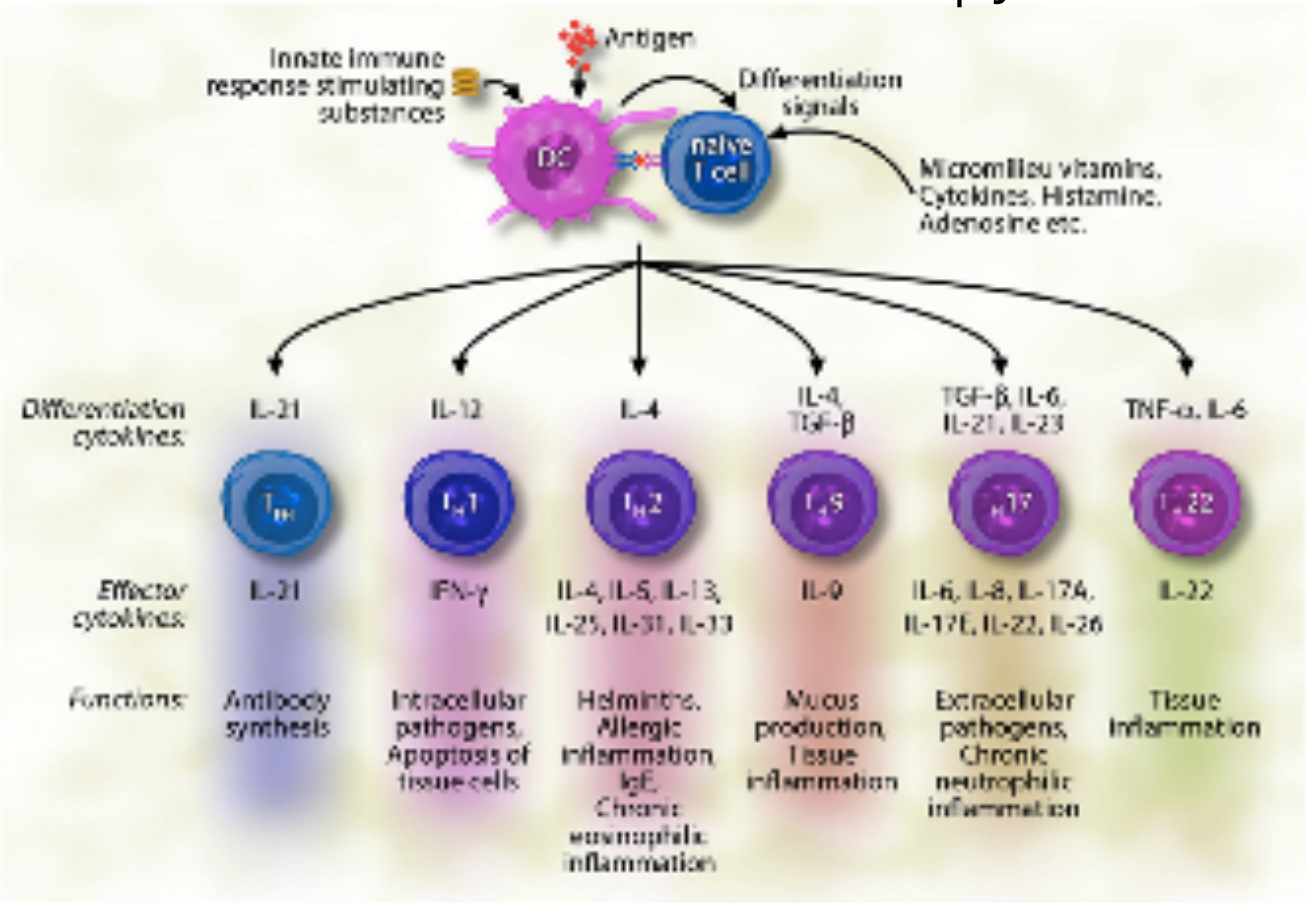
REVIEW
published: 24 April 2019
doi: 10.3389/fnagi.2019.00089

$G_{i/o}$-Protein Coupled Receptors in the Aging Brain

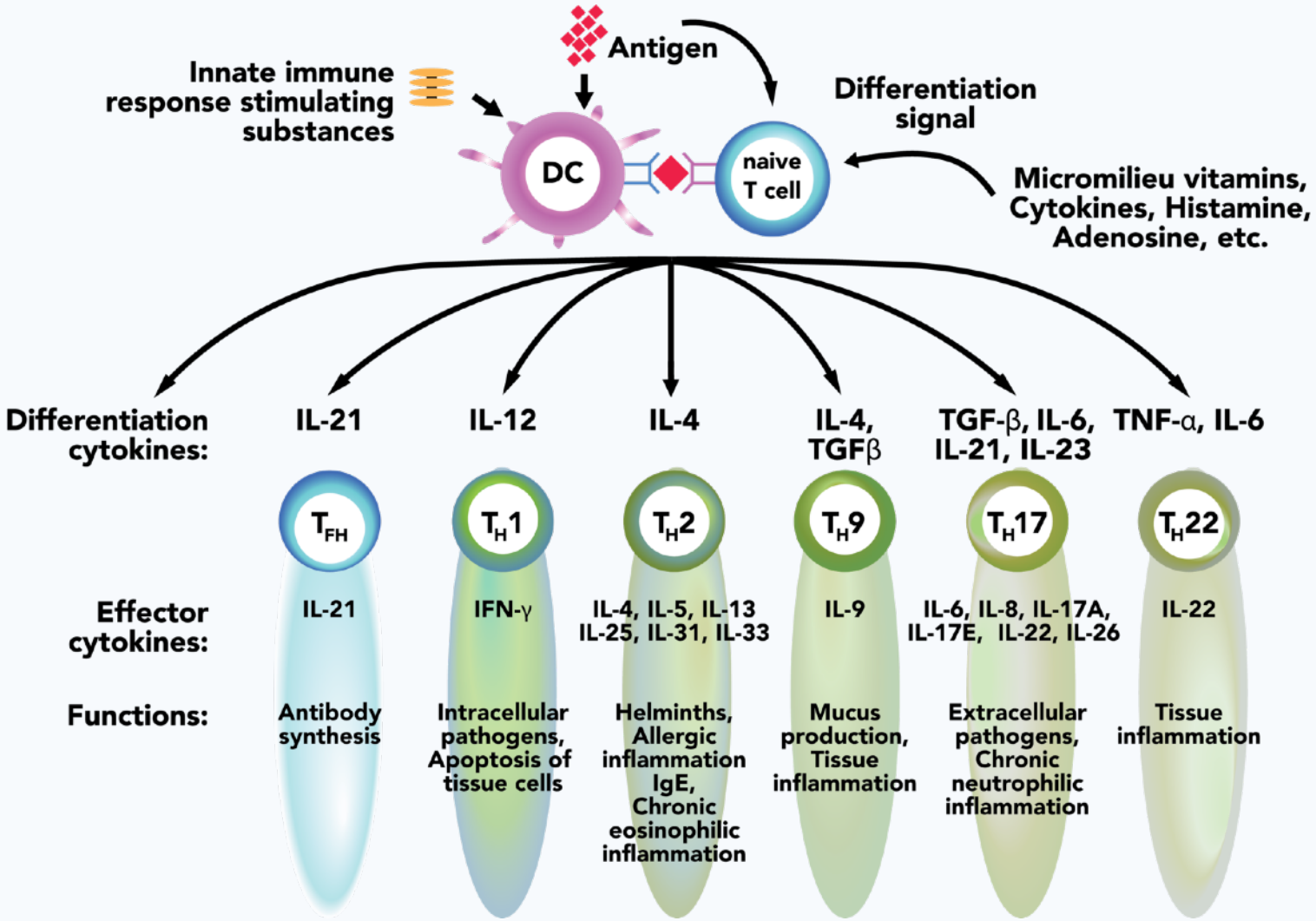
Patrícia G. de Oliveira[1†], Marta L. S. Ramos[1†], António J. Amaro[2], Roberto A. Dias[1†‡] and Sandra I. Vieira[1†]*



Cytokine signatures can serve as a diagnostic fingerprint of pathogens and Biomarkers for therapy



Inappropriate Activation of the cellular Immune system is important in the pathogenesis of human Retrovirus Associated Disease



Every Experimental injection Bypasses The Innate Immune System

Omnitüra Therapeutics

Combination therapy for prostate cancer using botanical compositions and bicalutamide

WO 2012061790 A1

ABSTRACT

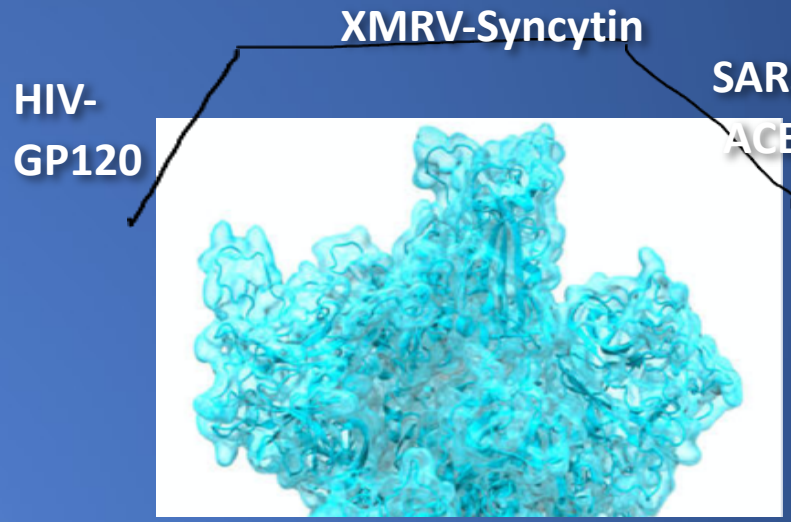
Botanical compositions comprising non-alcoholic organic extracts of Ganoderma lucidum, Salvia miltiorrhiza, and Scutellaria barbata for use in conjunction with bicalutamide therapy fpr cancer therapy, are provided. Methods for treatment or therapy of prostate cancer in a human is provided, the method comprising: administering an effective amount of a botanical composition that is effective for reducing androgen receptor protein expression; and administering concurrently an effective amount of a compound having anti-androgen activity, wherein the concurrent administration of the compound and the botanical composition achieves a therapeutic effect that is more effective than either agent alone.

Publication number	WO2012061790 A1
Publication type	Application
Application number	PCT/US2011/059471
Publication date	May 10, 2012
Filing date	Nov 4, 2011
Priority date	Nov 4, 2010
Also published as	CA2816855A1, CN103327994A, 4 More »4 More »
Inventors	James Dao, Jeffrey Dao, 8 More »8 More »
Applicant	Genyous Biomed International
Export Citation	BiBTeX, EndNote, RefMan

Patent Citations (7), Non-Patent Citations (52), Referenced by (3), Classifications (10), Legal Events (4)

External Links: Patentscope, Espacenet

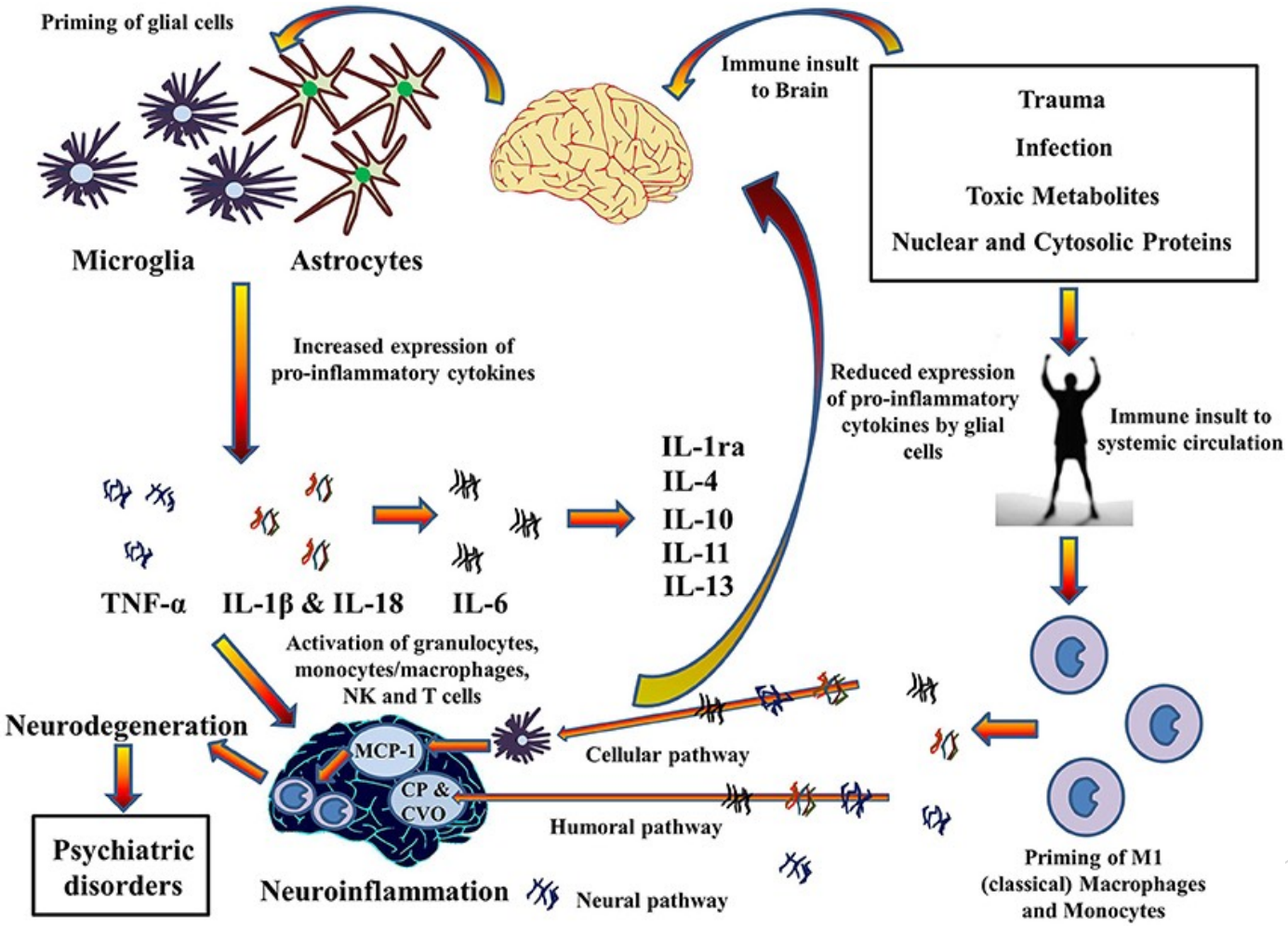
A CLINICAL STAGE BIOPHARMACEUTICAL COMPANY HARNESSING THE POWER OF PLANTS.



Xenotropic Murine Leukemia Virus-related Virus-associated Chronic Fatigue Syndrome Reveals a Distinct Inflammatory Signature

in vivo *25*: 307-314 (2011)

VINCENT C. LOMBARDI[1], KATHRYN S. HAGEN[1], KENNETH W. HUNTER[4], JOHN W. DIAMOND[2†], JULIE SMITH-GAGEN[3], WEI YANG[3] and JUDY A. MIKOVITS[1]



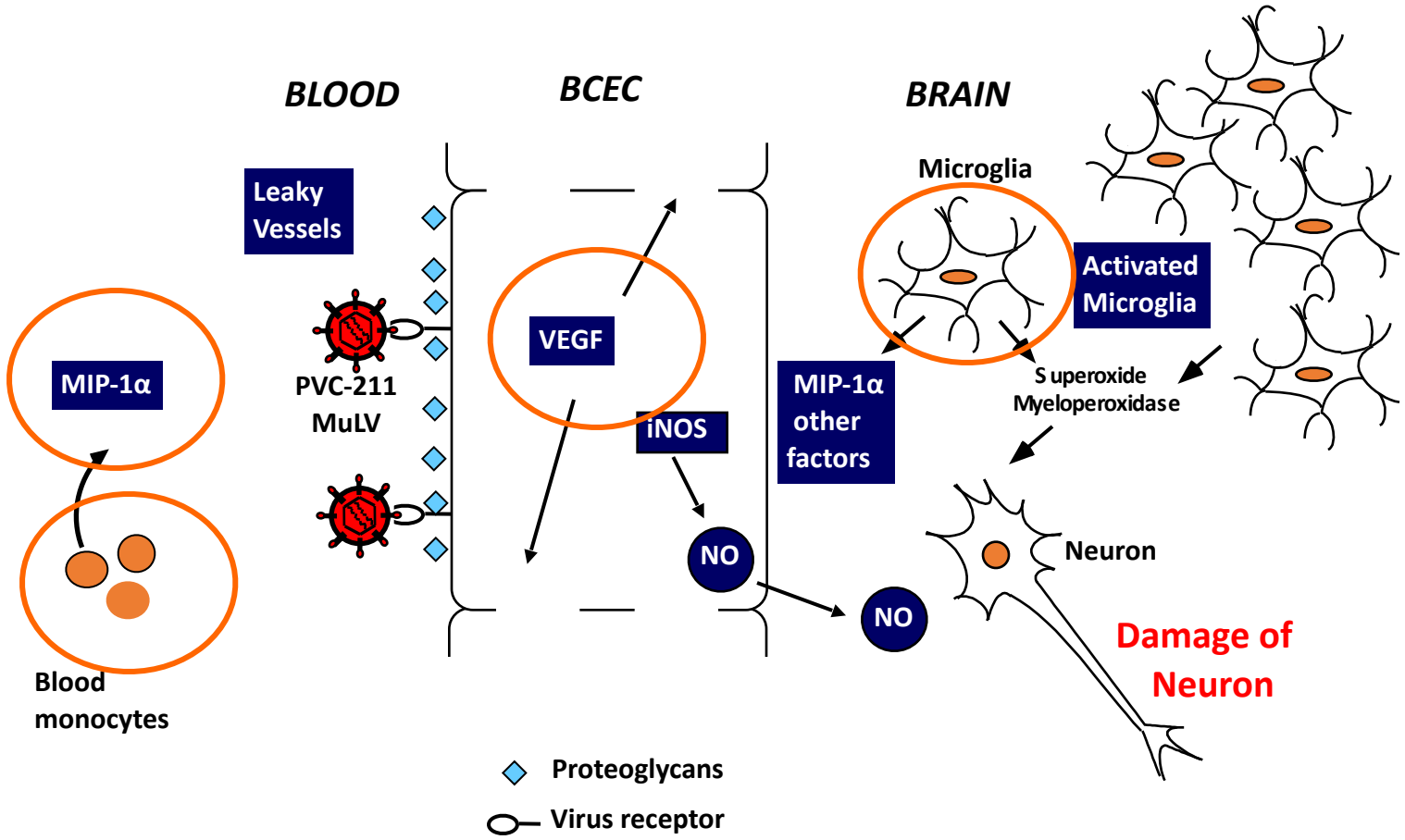
Chronic innate immune activation leads to inflammation and immune dysregulation

- Presence of CD20+ CD23+ B cells, not normally seen in healthy subjects, and activated APCs in some ME/CFS, CLD patients are similar to the myeloid and B cell defects described in other retroviral associated Diseases.
- The significant changes in the myeloid compartment including phenotypes are suggestive of activation of Antigen Presenting Cells (APCs) .
- Increased , γδT Cells clonality in ME/CFS, CLD, CLL, MCL
- Increased NKT compartment together with increased NKT to NK ratio.
- Major changes in inflammasome

Conclusion

Results suggests a similar Disease cycle of chronic innate immune activation leading to an immune dysregulation and chronic immunosuppression and may guide future research towards the development of biomarkers and treatment targets

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

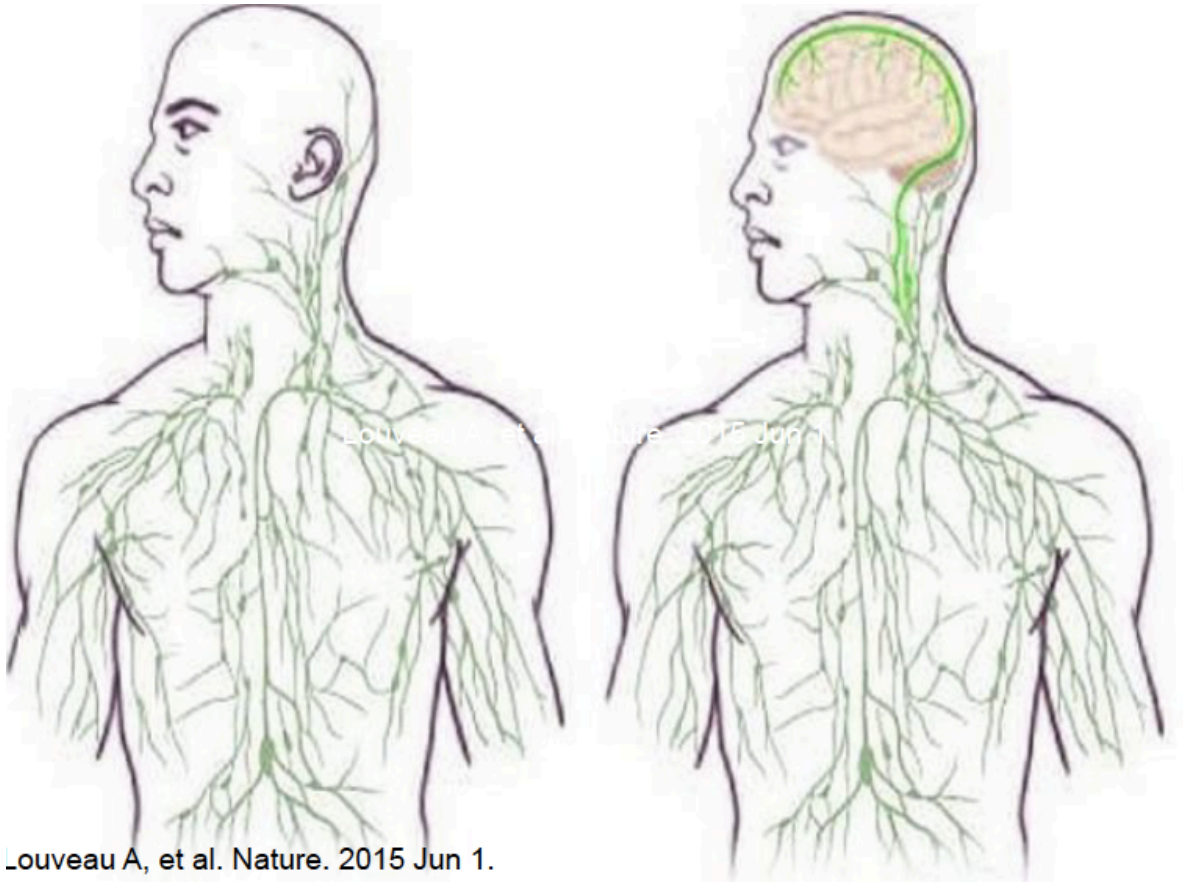
PUBLIC RELEASE: 1-JUN-2015

Missing link found between brain, immune system -- with major disease implications

Implications profound for neurological diseases from autism to Alzheimer's to multiple sclerosis

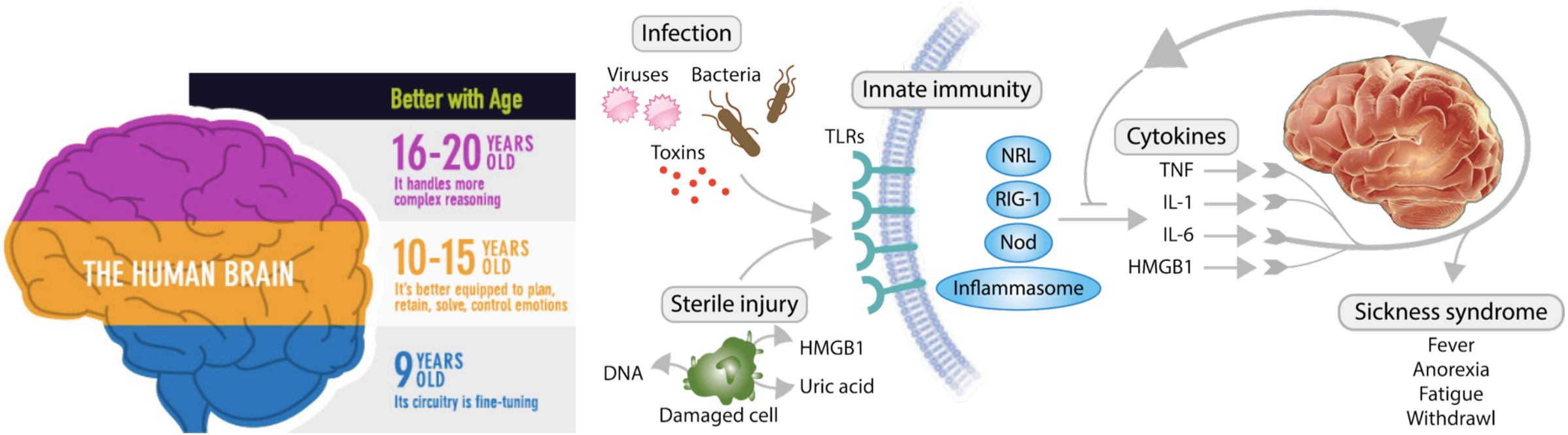
Structural and functional features
of central nervous system
lymphatic vessels

• We discovered functional lymphatic vessels lining the dural sinuses. These structures are able to carry both fluid and immune cells from the cerebrospinal fluid, and are connected to the deep cervical lymph nodes. The discovery of the central nervous system lymphatic system may call for a reassessment of basic assumptions in neuroimmunology and sheds new light on the aetiology of neuroinflammatory and neurodegenerative diseases associated with immune system dysfunction.



Louveau A, et al. Nature. 2015 Jun 1.

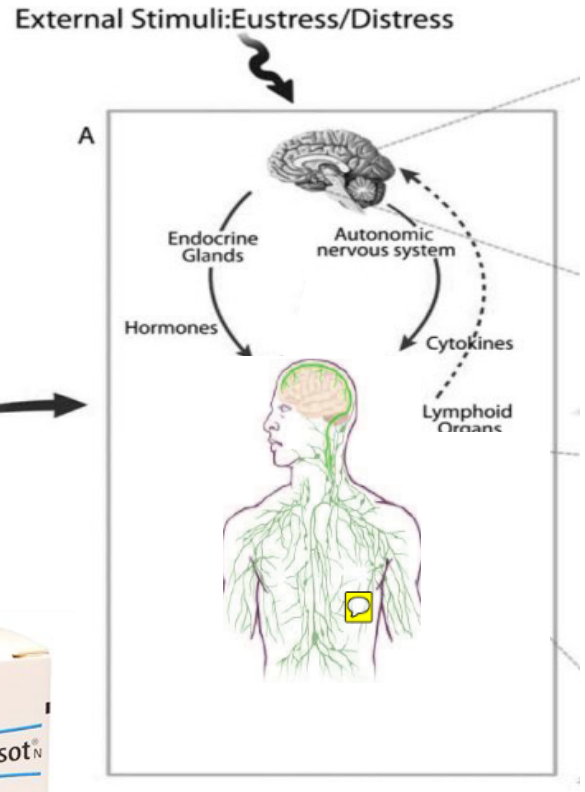
Danger of Inoculation During key Developmental Phases



Most Vulnerable: pregnant women, infants, teens, elderly, male vs female

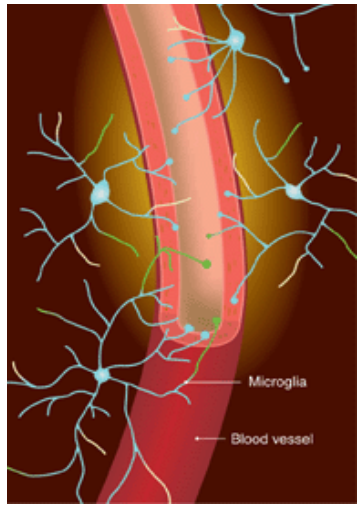
Chronic Disease involves every aspect of Human Biology.

From birth the developing Brain and Immune system are Inextricably linked



Stimuli

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



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

others

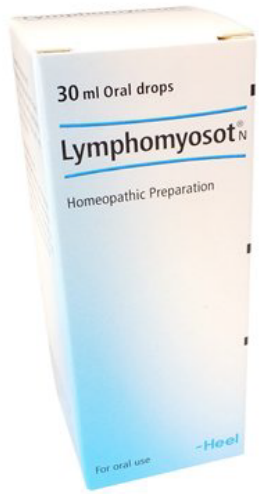
Homeostasis

Phagocytosis of debris, neurotrophins

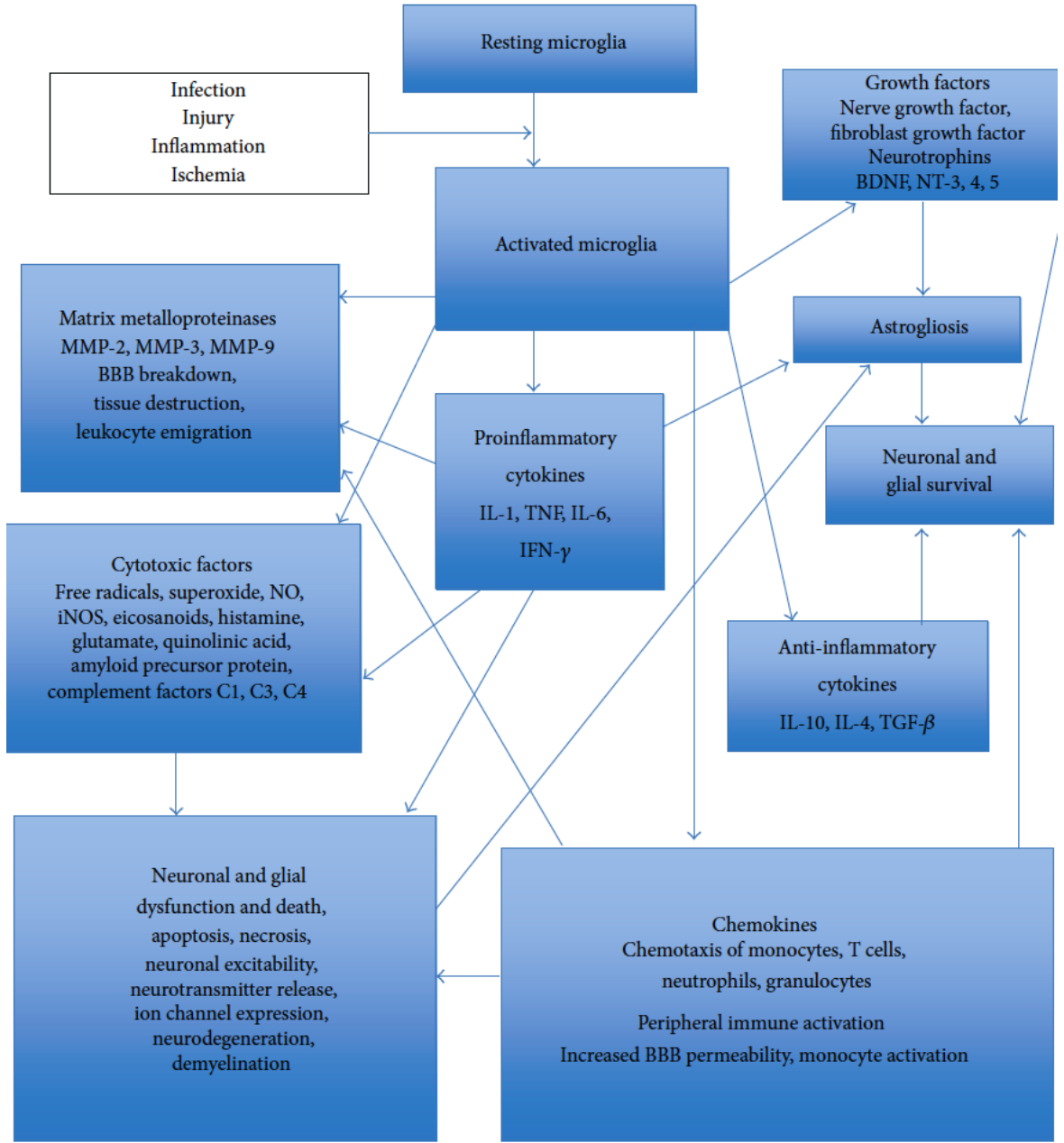
Damage

Neurotoxins, phagocytosis of normal neurons, apoptosis

NeuroImmune Disease and Cancer



Central role of micgroglia in Neuroinflammation



Rameshe Et. Al. 2013

Mediators of inflammation

VACCINE AIDS = COVID19: Autoimmune, Autoinflammatory Disease & Cancer *Unintended* Consequences of 3 DECADES LIABILITY FREE VACCINES

Prostate Cancer*	Crohn's Disease*	Gulf War Syndrome*
Breast Cancer *	Hashimoto's Thyroiditis*	Autism / ASD*
Multiple Myeloma*	Polymyositis*	Multiple Sclerosis*
Non-Hodgkins Lymphoma*	Sjogren's Syndrome *	Parkinson's*
Chronic Lymphocytic Leukemia*	Bechet's Disease*	ALS*
Mantle Cell Lymphoma*	Primary Biliary Cirrhosis*	Fibromyalgia*
Hairy Cell Leukemia*	Inflammatory Bowel Disease*	Chronic Lyme Disease*
Bladder Cancer *	Psoriasis, Dermatitis	OCD*
Colorectal Cancer*	Diabetes*	ADHD*
Kidney Cancer *	Cardiovascular Disease*	PTSD*
Ovarian Cancer*	ME / CFS*	Psychosis*
	Lupus/SLE*	Rheumatoid Arthritis*

**Neuroendocrine Tumors*

KEY to IMMUNITY is do not defile the TEMPLE of GOD

NEVER GET ANOTHER VACCINE

Earliest Symptoms reflect immune damage

- SKIN: Rash
- Gastrointestinal tract: GERD, leaky Gut
- Hematopoietic (Blood) Cells: Myelodysplasia
- Brain: Blood Brain Barrier lymphatics: edema

VACCINE INJURY:
Assessment, Treatment and Prevention

- Immunity is not static; it changes with age, with many unique features in early life.
- 36 years of research in Immunotherapy Inform Immune related adverse events in vaccination
- T cell responses to vaccination differ from those induced by infectious challenge
- The Brain cannot tolerate the introduction of antigens without eliciting an inflammatory immune response
- Development of novel age-specific vaccine formulations and delivery systems is likely warranted.
- Antiquated Concept: ONE SIZE FITS ALL: ONE SIZE Clearly DOES NOT FIT ALL

1986theact.com NVICP Justice Denied: HBV vaccine at birth when DNA Methylation resets

IGF-1: A Biomarker of Human Aging and the Development of Chronic Disease regulated by DNA Methylation

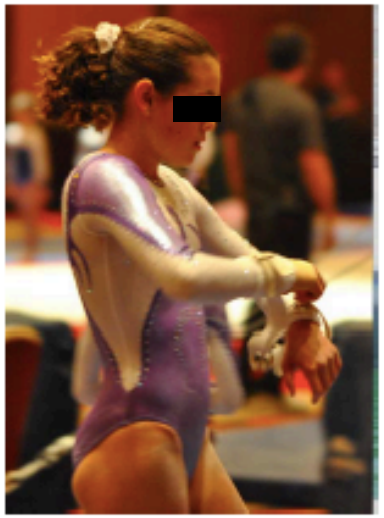
					References
Lifelong physiological properties of IGF-1	Growth	Bone metabolism	Lipid and glucose metablolisms	Neuroprotection	15, 16, 22, 23, 26-28 55-88, 101-108, 121-133, 156-175, 169, 206, 225-233, 270-275, 290, 295-306, 357-359, 398
	Neurgenesis and synaptogenesis	Anabolizing	Antioxidant and antiinflammatory	Antiapoptotic	
	Genital development	Proliferative	Hepato and cardioprotrection	Mitochondrial protection	
Processes involved in longevity	Genetic stability	Stress resistance	Metabolic control	Telomere shortening	238

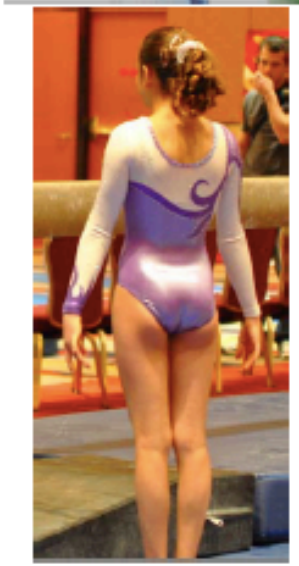
IGF-1

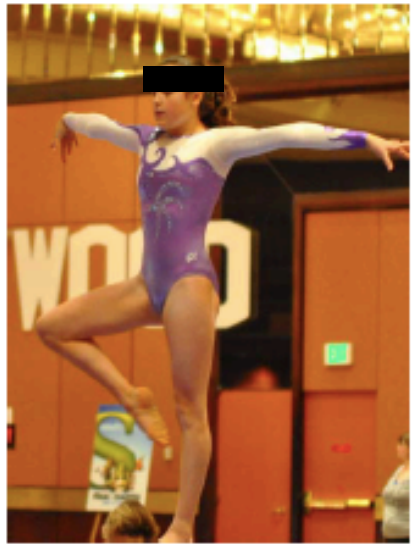
GARDASIL INJURY

Death, Leukemia, Psychosis, Cardiac Arrest, Autoimmune Disease, Alopecia, Sterility in 25% of those vaccinated

Jessica – Before Vaccine







Jessica – After Vaccine

IS IT GARDASIL INJURY OR NON-HIV AEIDS?

Lauren After Gardasil

Is it Gardasil Injury or COVID Hair loss? Is there a difference?

Poisons (ADJUVANTS): Aluminum, LPS (ENDOTOXIN), Xenoestrogens, Arsenic in Vaccines food & water target Innate Immune responses

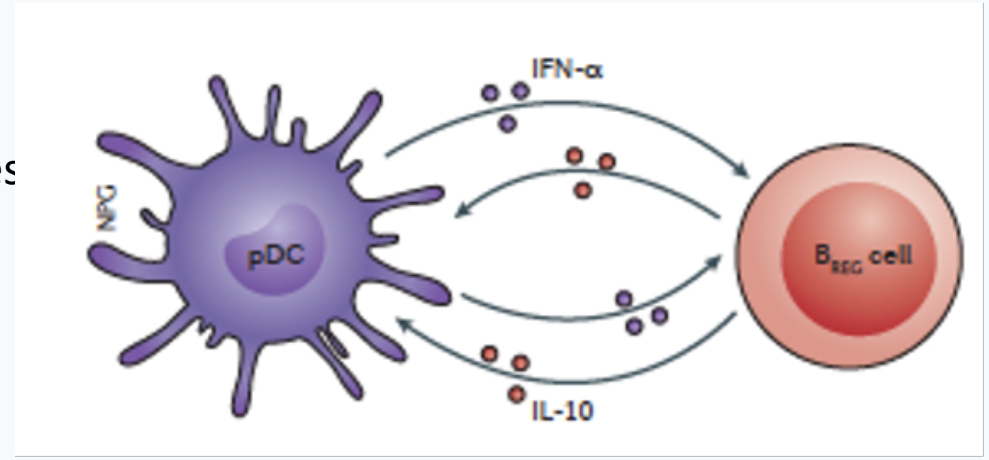
Lupus is an autoimmune inflammatory disease in which the body produces antibodies causing the immune system to affect the skin, joints, blood and kidneys.

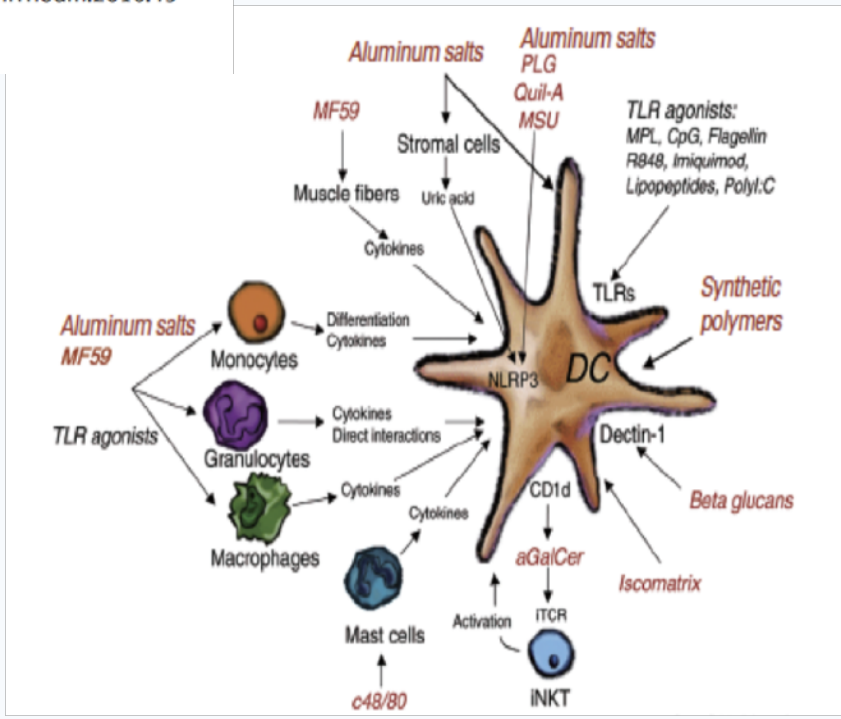
Symptoms include:

- Skin rashes/ Inflammation
- Arthritis/ Joint Pain
- Extreme Fatigue
- Anemia/ Blood Disorders
- Kidney Damage
- Immune Disorder
- Antinuclear Antibodies

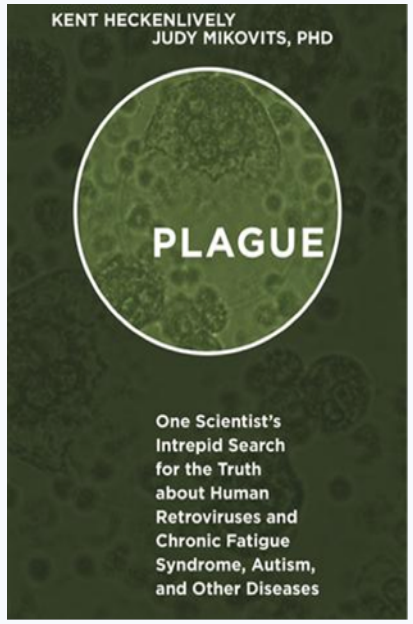
Nature Reviews Rheumatology | Published online 24 Mar 2016; doi:10.1038/nrrheum.2016.43

Compromised pDC–B_{REG} cell crosstalk

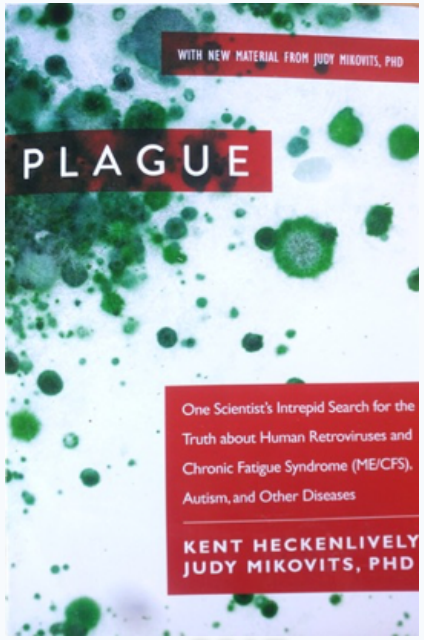




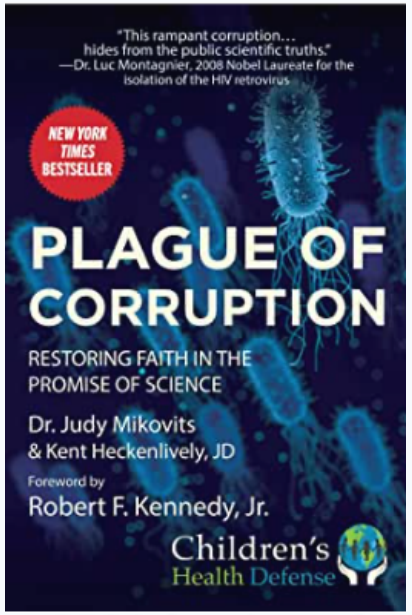
THE FATE OF THOSE WHO FIGHT THE DARKNESS



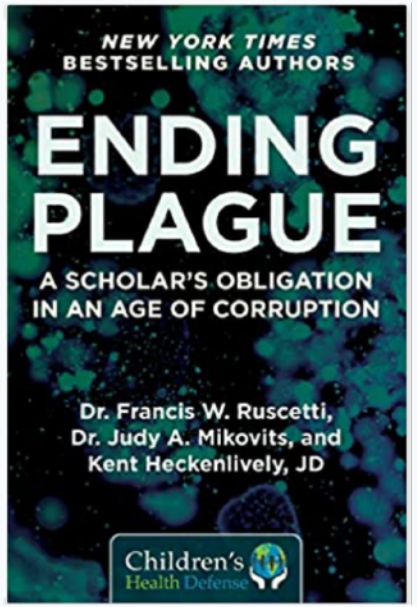
2014 *(James 1:19-22)*



2017



2020 *(Psalm 91)*



2021 *(Ephesians 5:11)*

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'The great enemy of truth is very often not the lie – deliberate, contrived and dishonest – but the myth – persistent, persuasive and unrealistic. Too often we hold fast to the cliches of our forebears. We subject all facts to a prefabricated set of interpretations. We enjoy the comfort of opinion without the discomfort of thought'. John F. Kennedy, Commencement Address, Yale University, June 11, 1962

HIV-1: A Virus that Never Comes Alone

Cell Host & Microbe
Short Review

War and Peace between Microbes: HIV-1 Interactions with Coinfecting Viruses

Andrea Lisco,[1] Christophe Vanpouille,[1] and Leonid Margolis[1,*]
[1]Program in Physical Biology, Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD 20892, USA
*Correspondence: margolil@mail.nih.gov
DOI 10.1016/j.chom.2009.10.010

HIV-1 disrupts the homeostatic equilibrium between the host and coinfecting microbes, facilitating reactivation of persistent viruses and invasion by new viruses. These viruses usually accelerate HIV disease but occasionally create conditions detrimental for HIV-1. Understanding these phenomena may lead to anti-HIV-1 strategies that specifically target interactions between HIV-1 and coinfecting viruses.

Host Virus/Microbe Equilibrium is Disrupted by HIV-1

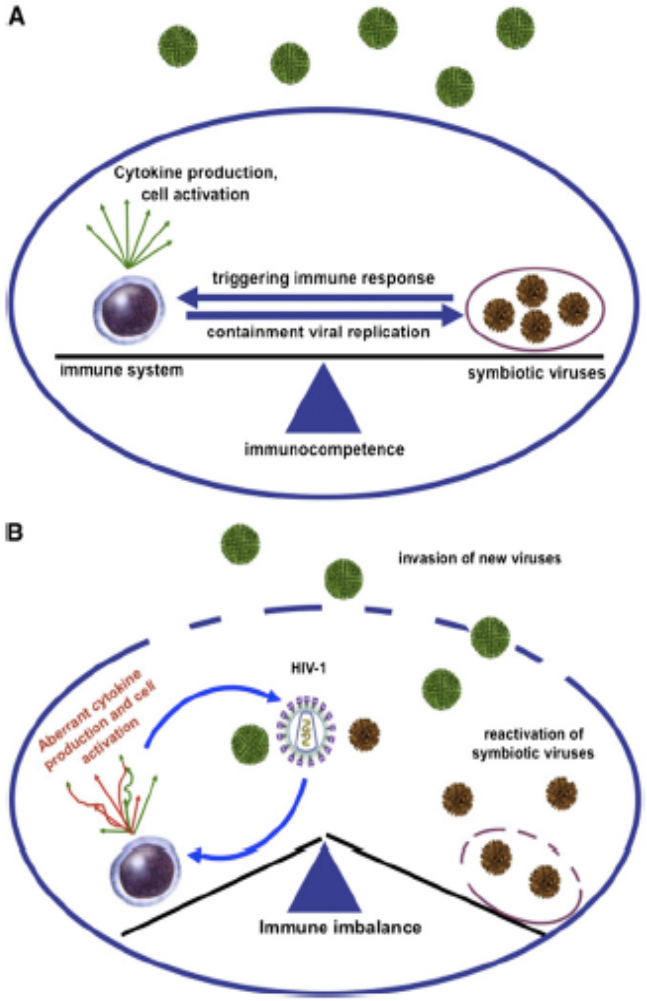
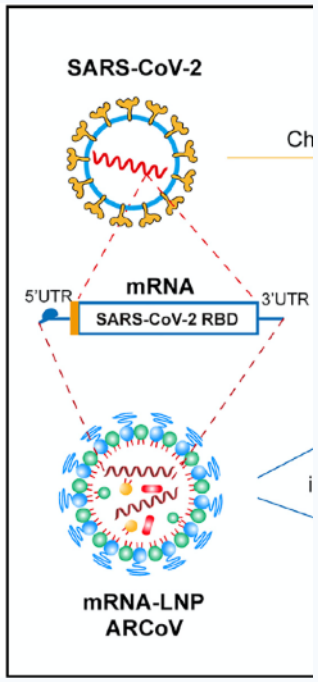


Table 1. Mechanisms of Interactions between HIV-1 and Coinfecting Viruses

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

ITS ALL ABOUT INFlammation

Breakdown of cell membranes and release of the PLA2…starts inflammation
Damage so severe lungs are filling up…brain is fooled because it happens rapidly!



SCAN ME

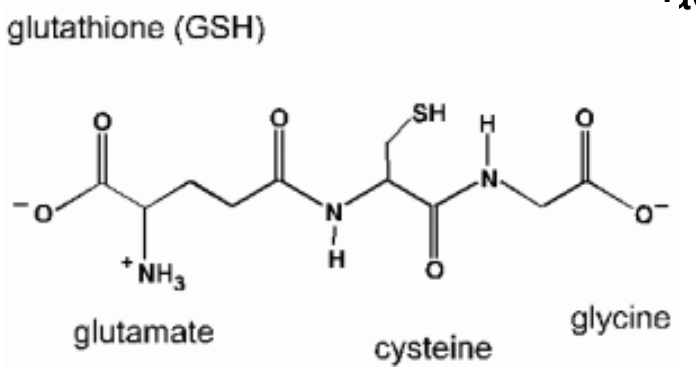
Detoxing that synthetic Lipid Nano Particle
(SARS-CoV2 virus & COVID Vaccine)

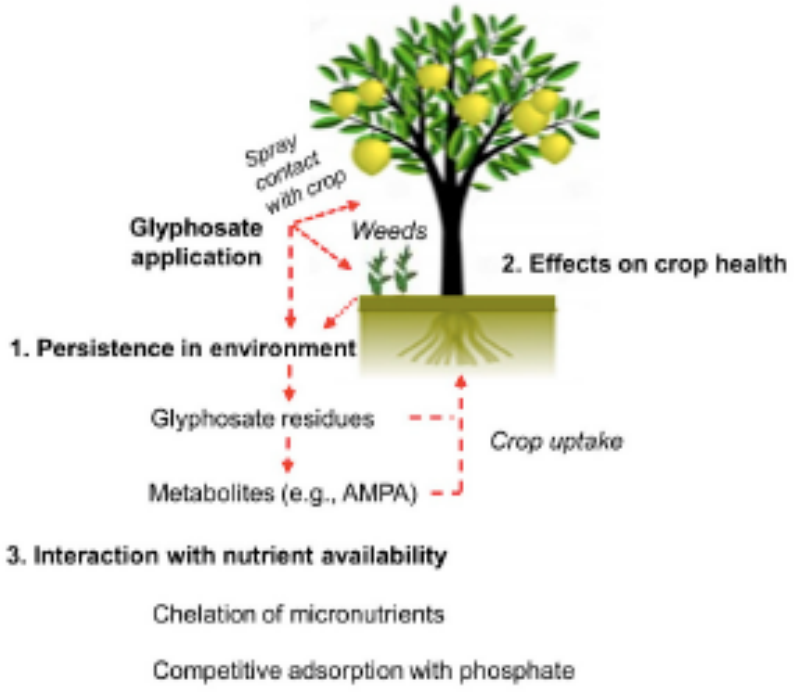
- Ozone therapies
- Specialized Pro resolving mediators
- Chlorine Dioxide, MMS, CDS

Glyphosate: Damages Key GOD GIVEN antioxidant Glutathione

Produced by the liver, glutathione is made up of three amino acids: Lcysteine, glycine, and L-glutamate

SCAN ME





American Chemical Society
Public Health Emergency Collection
Public Health Emergency COVID-19 Initiative

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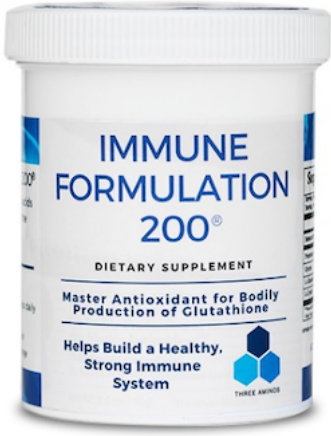
PMCID: PMC7263077
PMID: 32463221

Endogenous Deficiency of Glutathione as the Most Likely Cause of Serious Manifestations and Death in COVID-19 Patients

Alexey Polonikov*

Author information · Article notes · Copyright and License information Disclaimer

Endogenous glutathione deficiency appears to be a crucial factor enhancing SARS-CoV-2-induced oxidative damage of the lung and, as a result, leads to serious manifestations, such as acute respiratory distress syndrome, multiorgan failure, and death in COVID-19 patients. When the antiviral activity of GSH is taken into account, individuals with glutathione deficiency seem to have a higher susceptibility for uncontrolled replication of SARS-CoV-2 virus and thereby suffer from an increasing viral load. The severity of clinical manifestations in COVID-19 patients is apparently determined by the degree of impaired redox homeostasis attributable to the deficiency of reduced glutathione and increased ROS production. This assumption can be supported by our findings. In particular, COVID-19 patients with moderate and severe illness had lower levels of glutathione, higher ROS levels, and greater redox status (ROS/GSH ratio) than COVID-19 patients with a mild illness. Long-term and severe manifestations of COVID-19 infection in one of our patients with marked glutathione deficiency suggest that the degree of glutathione decrease correlates negatively with viral replication rate and that an increasing viral load exacerbates oxidative damage of the lung. This finding suggests that the virus cannot actively replicate at higher levels of cellular glutathione, and therefore, milder clinical symptoms are observed with lower viral loads.



SUPPLEMENT FACTS

Servings Per Container			62
Serving Size			1 Scoop (1.6g)
Amount per serving			
Calories			0
		Standard DV	% Daily Value*
Selenium (from selenomethionine)	4.5 mcg	75 mcg	6%
Proprietary Amino Acid Blend	1450 mg		
Glycine			
L-Glutamine			
L-Cystine			

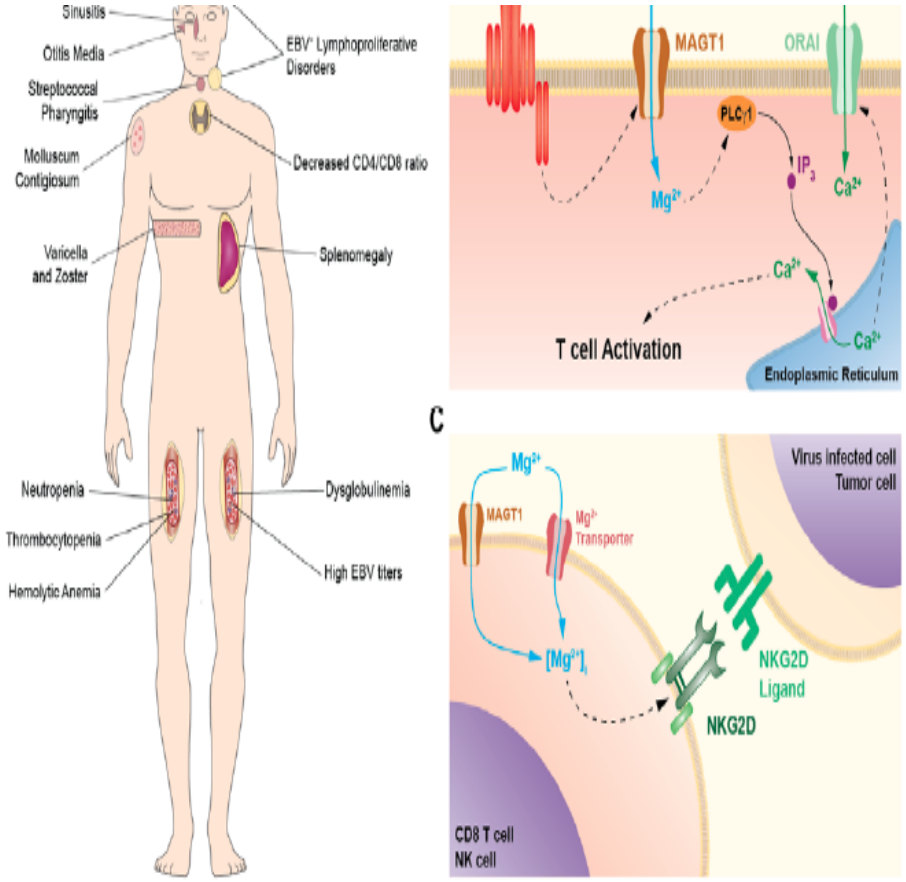
Glyphosate in our soil -> our plants are SICK -> Does toxic food cause COVID?

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)

XMEN- New Primary Immune Deficiency

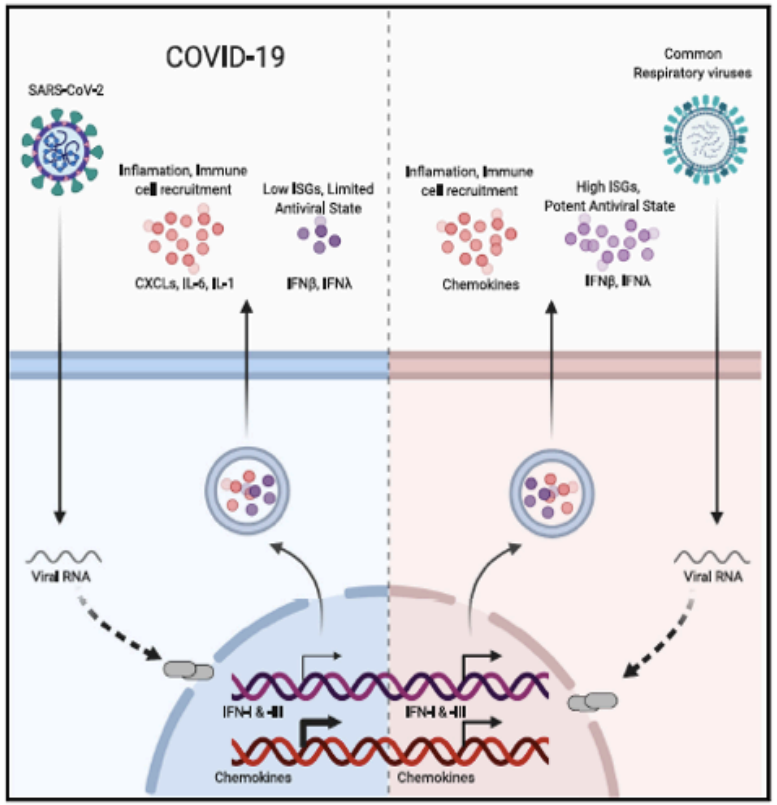


C

Mg2+ Transporter deficiency results in defective T and NK cell Function

Imbalanced IFN Response to RNA Viruses Drives Development of Autoimmune, Autoinflammatory Disease & Cancer

Graphical Abstract



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In Brief

In comparison to other respiratory viruses, SARS-CoV-2 infection drives a lower antiviral transcriptional response that is marked by low IFN-I and IFN-III levels and elevated chemokine expression, which could explain the pro-inflammatory disease state associated with COVID-19.

Cell Host & Microbe
Short Article

Gut Dysbiosis Promotes M2 Macrophage Polarization and Allergic Airway Inflammation via Fungi-Induced PGE_2

Yun-Gi Kim,[1,2,5] Kankanam Gamage Sanath Udayanga,[1,2] Naoya Totsuka,[1,2] Jason B. Weinberg,[4] Gabriel Núñez,[5] and Akira Shibuya[1,2,3,*]
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http://dx.doi.org/10.1016/j.chom.2013.12.010

Only certain antibiotic promote fungal overgrowth in the gut, suggesting
Specific commensal bacteria have the ability to prevent colonization of Candida

Treatment: Celebrex

- Infection/inflammation of GI Tract
- Lymphadenopathy
- Splenomegaly
- Multi-organ Granuloma accumulation
- Idiopathic Thrombocytopenia (ITP)
- Autoimmune hemolytic Anemia

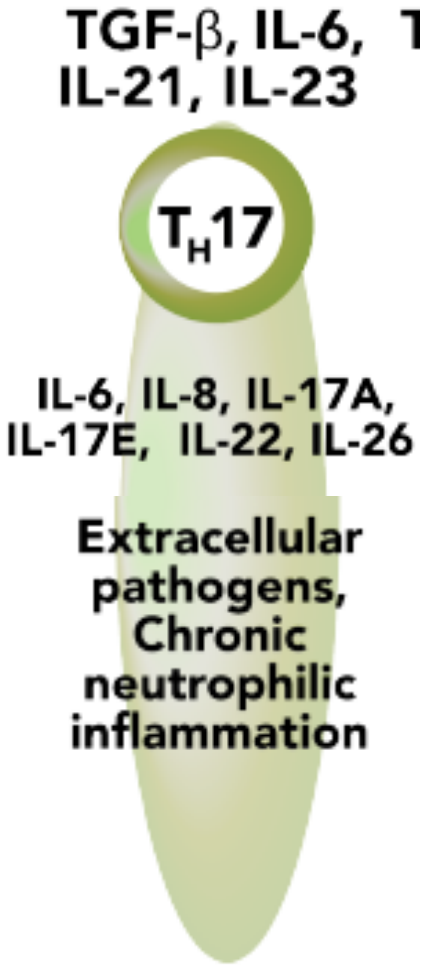
Mutations in at least ten genes Including TNFRSF13B

- Key to survival and proliferation of B cells

- mast cell disease
- Asthma
- Protection against nematode infections

Genetic Susceptibility?: Erlers Danlos Syndrome (EDS)

Autoimmune Disease Development & Molecular Mimicry



- Asthma
- MS
- RA
- Psoriasis
- Complement mediated Hypersensitivity

- Atopic Dermatitis
  - Psoriasis
  - Melanoma

Clin Rheumatol
DOI 10.1007/s10067-015-2969-z

REVIEW ARTICLE

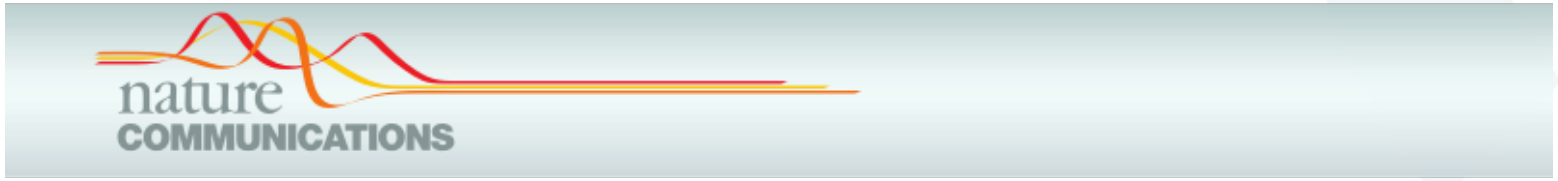
Hypothesis: Human papillomavirus vaccination syndrome—small fiber neuropathy and dysautonomia could be its underlying pathogenesis

Manuel Martínez-Lavín[1]

Received: 8 April 2015 / Revised: 5 May 2015 / Accepted: 5 May 2015

Disruption of the blood-brain barrier triggers a cascade of events that results in autoimmunity and brain damage characteristic of multiple sclerosis

- a single drop of blood in the brain is sufficient to activate an autoimmune response akin to multiple sclerosis (MS)
- introduction of blood in the healthy brain is sufficient to cause peripheral immune cells to enter the brain
- which then go on to cause brain damage.



ARTICLE

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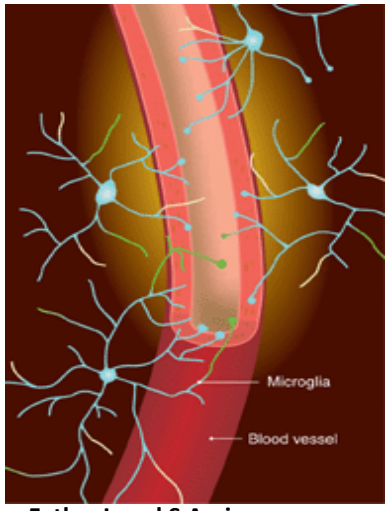
Blood coagulation protein fibrinogen promotes autoimmunity and demyelination via chemokine release and antigen presentation

Jae Kyu Ryu[1], Mark A. Petersen[1,2], Sara G. Murray[1], Kim M. Baeten[1], Anke Meyer-Franke[1], Justin P. Chan[1], Eirini Vagena[1], Catherine Bedard[1], Michael R. Machado[1], Pamela E. Rios Coronado[1], Thomas Prod'homme[3,4], Israel F. Charo[5], Hans Lassmann[6], Jay L. Degen[7], Scott S. Zamvil[3,4] & Katerina Akassoglou[1,3,4]

So what happens when a healthy brain is injected with ?

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

others

Damage
Neurotoxins, phagocytosis of normal neurons, apoptosis

Neurodegenerative disorders

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