

SARS-CoV2 Prevention and treatment of COVID19
The key is a healthy Immune and Endocannabinoid system

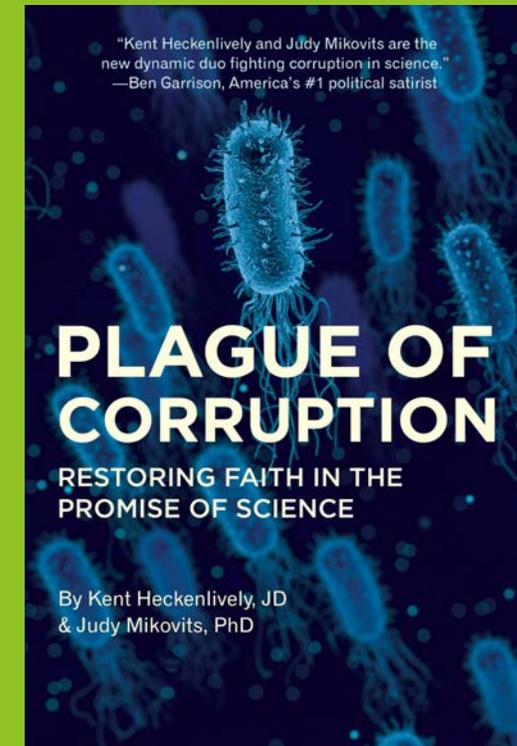
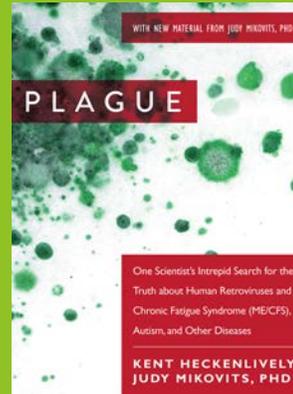
Dr. Judy Mikovits PhD
Biochemistry Molecular Biology

CANNA WORLD EXPO

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Censorship & Cover-up of Scientific Discovery led to Plague of Chronic Disease

- ▶ In Plague of Corruption we raise the broader question
- ▶ of the enormous risk of using animal tissue in research & mixing of it with human tissue
- ▶ for the development of medical therapies while covering up value and efficacies
- ▶ of natural product therapies like cannabis, homeopathy, energy therapies and other medicinal plants



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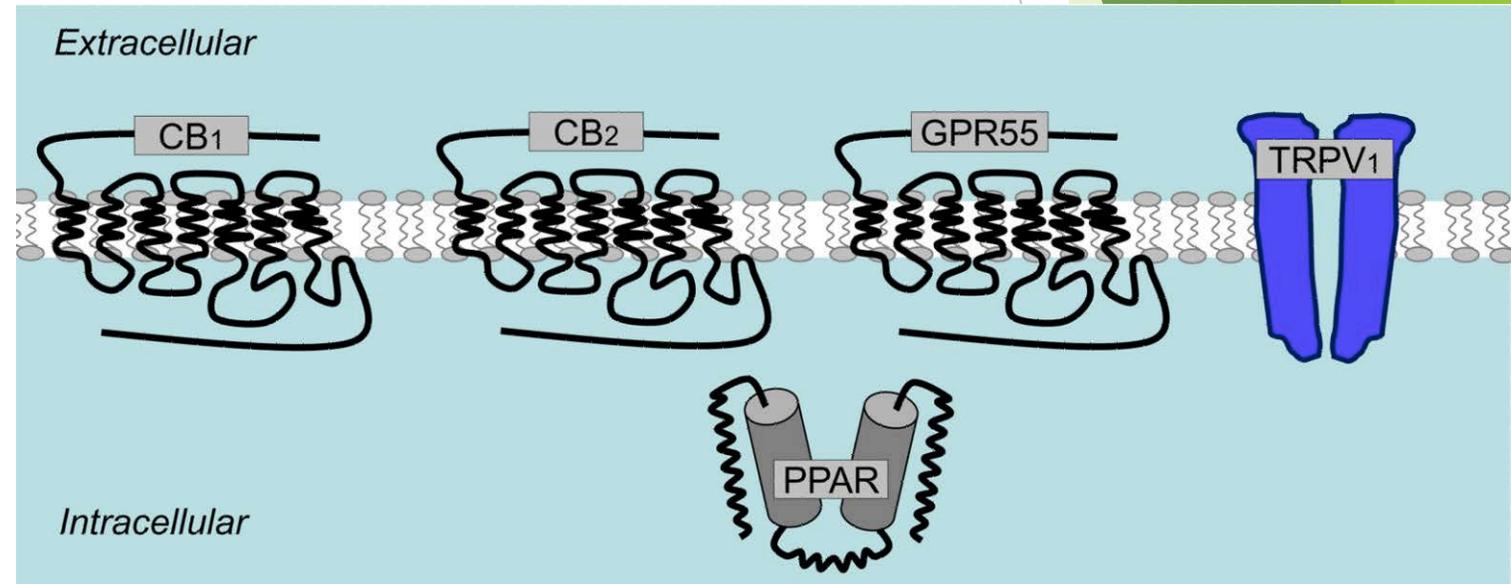
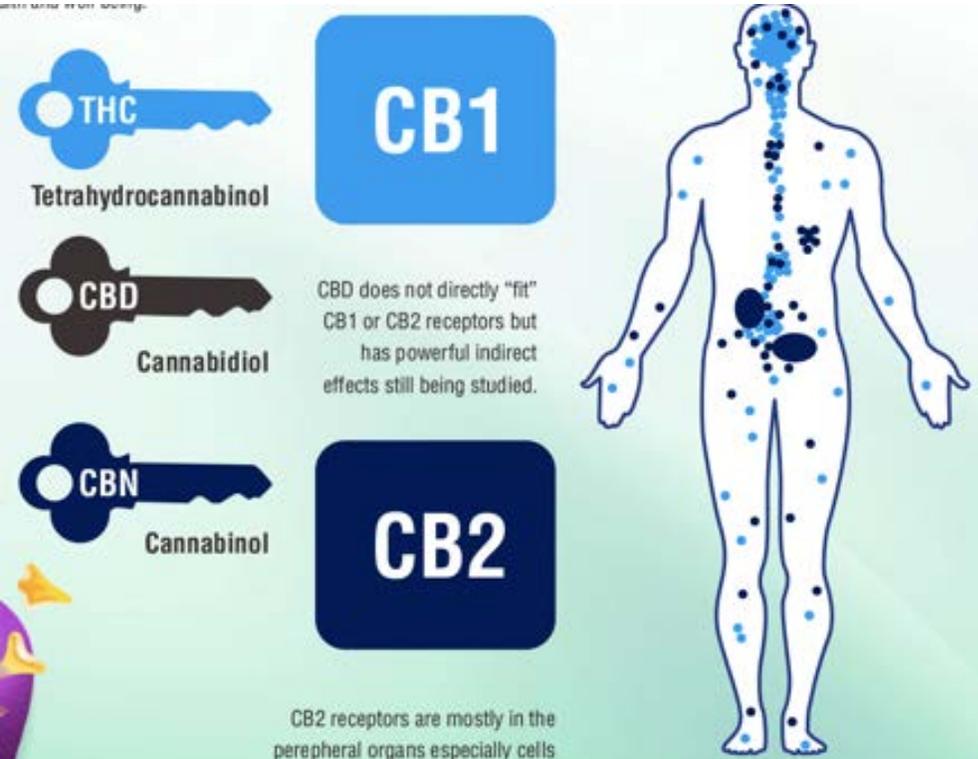
M.A.R.C. Inc.
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The Human Endocannabinoid System (eCS)

Key Regulator of stem cell development, Immune Homeostasis & Neuroimmune Health

A signaling system that helps to modulate all other physiological, behavioral, and energetic processes in the body.

Glia. 2010 July ; 58(9): 1017–1030



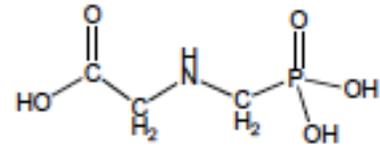
Anxiety
Depression
Sleep Disorders
Pain
Itch
Wound healing

- *neuroprotection & plasticity*
- *immunity & inflammation*
- *apoptosis & carcinogenesis*
- *pain and emotional memory*
- *Supports detoxification:*
 - *repairs Fibrosis*
 - *fatty Liver disease*

Glyphosate: Damages Key Intracellular antioxidant Glutathione

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

Chemical structure

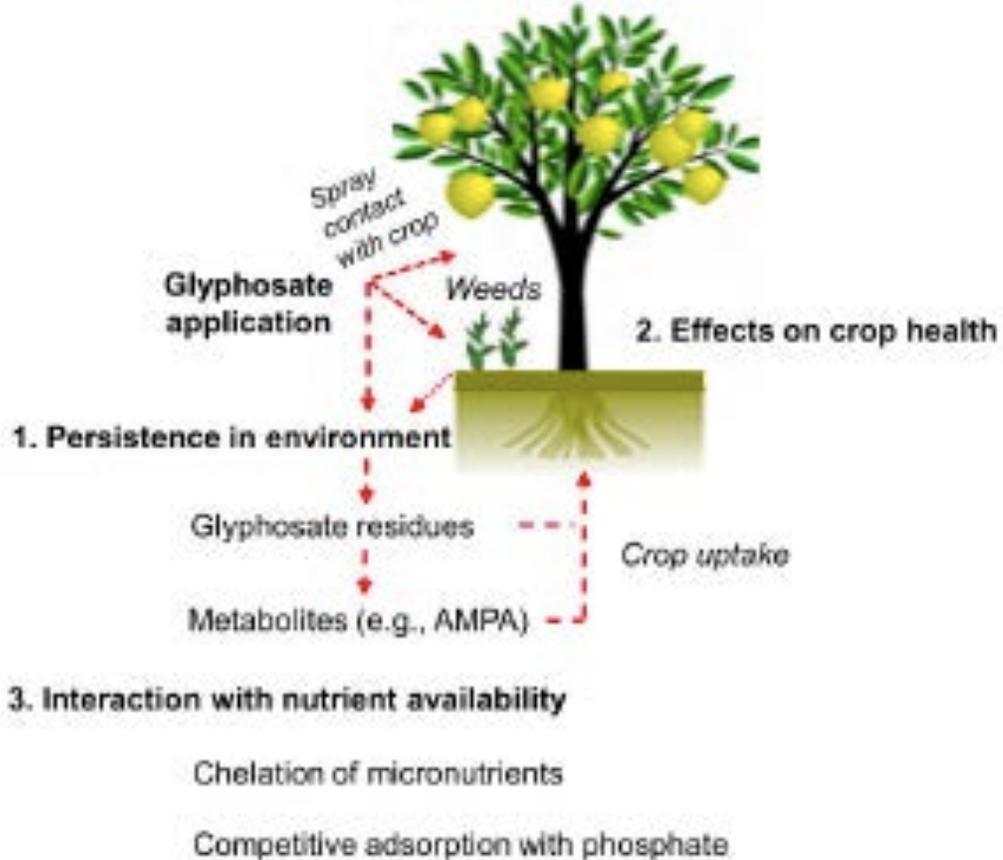


CAS number

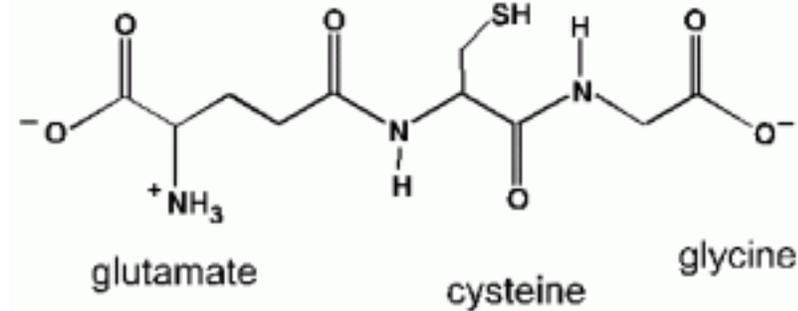
1071-83-6

Chemical name

N-(phosphonomethyl) glycine



glutathione (GSH)



How Glutathione Works In the Body:

- Helps break down nutrients
- Regulates immune response
- Protects against oxidative stress

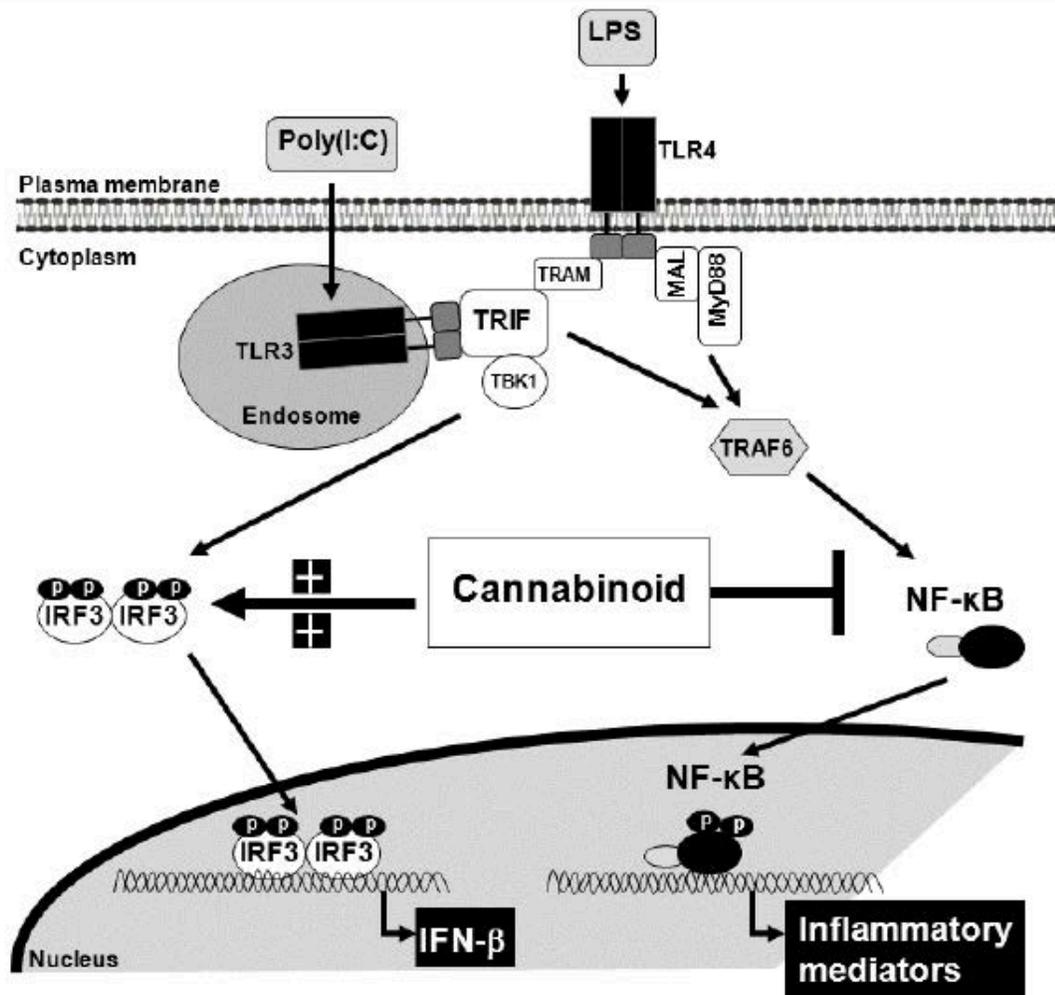
Coronavirus outbreak raises question: Why are bat viruses so deadly?



The Australian black flying fox is a reservoir of Hendra virus, which can be transmitted to horses and sometimes humans.

It's no coincidence that some of the worst viral disease outbreaks in recent years — SARS, MERS, Ebola, Marburg and likely the newly arrived COVID-19 virus — originated in bats.

- Generally, vigorous physical activity high metabolic rates lead to higher tissue damage due to an accumulation of reactive molecules, primarily free radicals.
- key trick of many bats' immune systems is the hair-trigger release of a signaling molecule called interferon-alpha, which tells other cells to “man the battle stations” before a virus invades



Cannabinoids are Anti-Viral and Reduce Inflammation

Viruses cause Disease by Dysregulating Key Immune molecules modulated by the eCS The Dimmer switch of Inflammation: Putting out the Fire

Comparison of cellular gene expression in Ebola-Zaire and Ebola-Reston virus-infected primary human monocytes

C. Xiang¹, H. Young², H. Alterson¹, D. Reynolds², M. Bittner³, Y. Chen², G. Gooden², Y. Jiang¹, P. Meltzer², J. Trent¹, J. Mikovits² & K. Anderson¹

¹Virology Division, US Army Medical Research Institute of Infectious Diseases,

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Ebola viruses are filamentous, enveloped, nonsegmented RNA viruses. Although most Ebola viruses, notably Ebola-Zaire virus, are highly infectious for primates and can cause severe haemorrhagic diseases, Ebola-Reston virus does not cause serious disease in humans. Microarray technology was employed to compare cellular gene responses to Ebola-Zaire and Ebola-Reston virus infection of primary human monocytes, the early targets of Ebola-Zaire virus infection. We found that approximately 200 of 1,400 human genes on the array exhibited changes in expression in response to Ebola-Zaire virus infection after 24 hours. Most affected genes were upregulated in their level of expression, including cytokine and chemokine genes (IL-1, IL-1, IL-6, IL-8, IL-15, MIP-1, MIP-1 and TNF), genes involved in regulation of cell cycle or apoptosis and other genes involved in signal transduction. The gene expression profile from Ebola-Reston-infected monocytes was totally different from that observed with Ebola-Zaire virus. The results from northern-blot or ribonuclease protection assays confirmed the array data. The possible influence of differences in cellular gene expression observed between Ebola-Zaire and Ebola-Reston viruses on the ability of these viruses to cause diseases will be discussed.

Research [\(/tags/106\)](#) In-Press Preview [\(/tags/113\)](#) Cell biology [\(/tags/16\)](#)

Immunology [\(/tags/25\)](#) Free access | [10.1172/JCI122462](https://doi.org/10.1172/JCI122462)

<https://doi.org/10.1172/JCI122462>

TGF β -induced epigenetic deregulation of SOCS3 facilitates STAT3-signaling to promote fibrosis

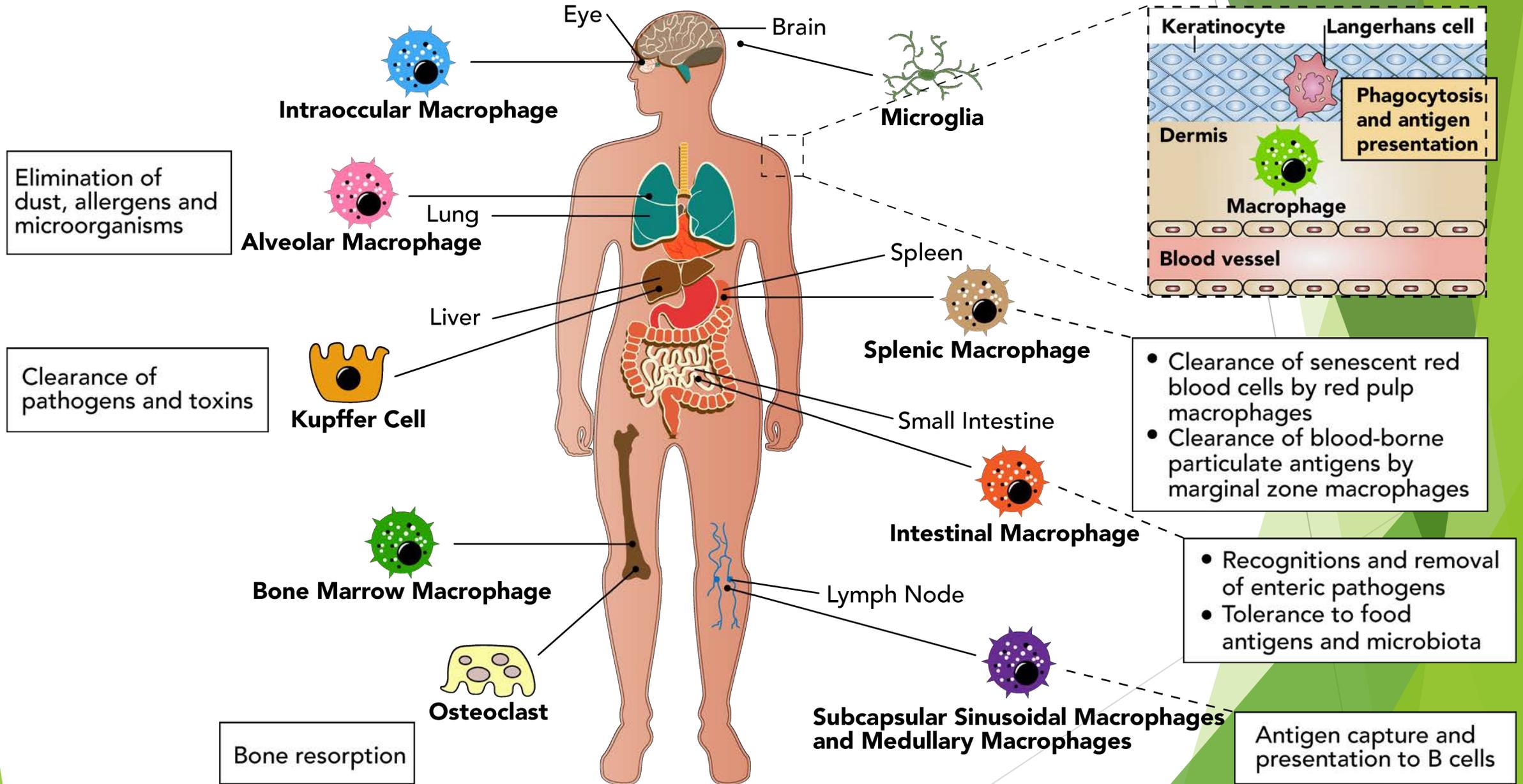
Clara Dees, Sebastian Pötter, Yun Zhang, Christina Bergmann, Xiang Zhou, Markus Lubber, Thomas Wohlfahrt, Emmanuel Karouzakis, Andreas Ramming, Kolja Gelse, Akihiko Yoshimura, Rudolf Jaenisch, Oliver Distler, Georg Schett, and Jörg H.W. Distler

First published January 28, 2020 - [More info](#)

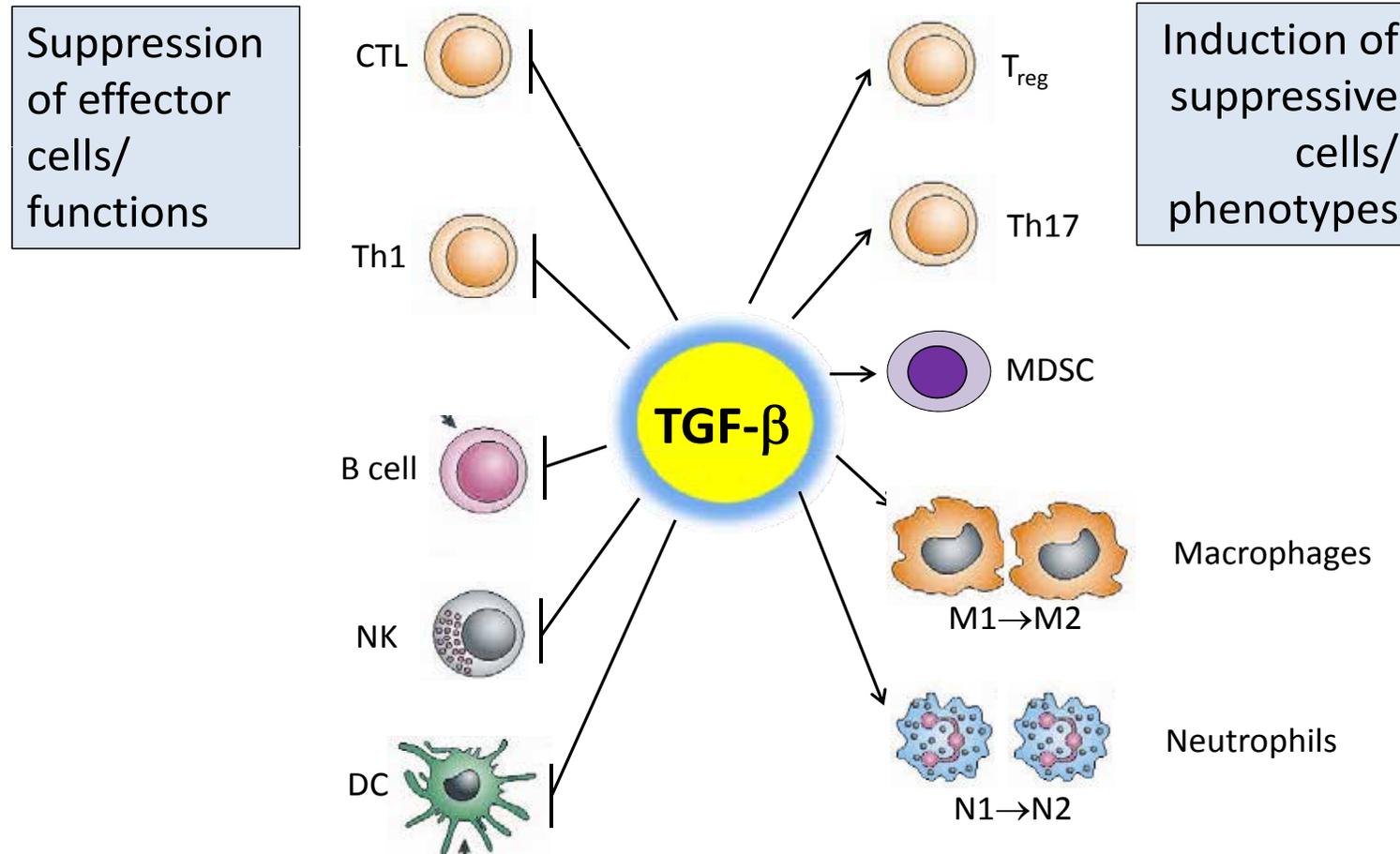
^ Abstract

Fibroblasts are key-effector cells in tissue remodeling. They remain persistently activated in fibrotic diseases, resulting in progressive deposition of extracellular matrix. Although fibroblast activation maybe initiated by external factors, prolonged activation can induce an “autonomous”, self-maintaining pro-fibrotic phenotype in fibroblasts. Accumulating evidence suggests that epigenetic alterations play a central role to establish this persistently activated pathologic phenotype of fibroblasts. We demonstrated that in fibrotic skin of patients with systemic sclerosis (SSc), a prototypical idiopathic fibrotic disease, transforming growth factor- β (TGF β) induced the expression of DNA-

Tissue Macrophages perform Key Homeostatic Functions Modulated by Cannabinoids



Cellular targets of TGF- β -driven immune suppression



TGF- β Effects Many Cells In The Immune System

Normal level of TGF- β is a **good soldier** promoting the survival of effector cells

Immune system in equilibrium

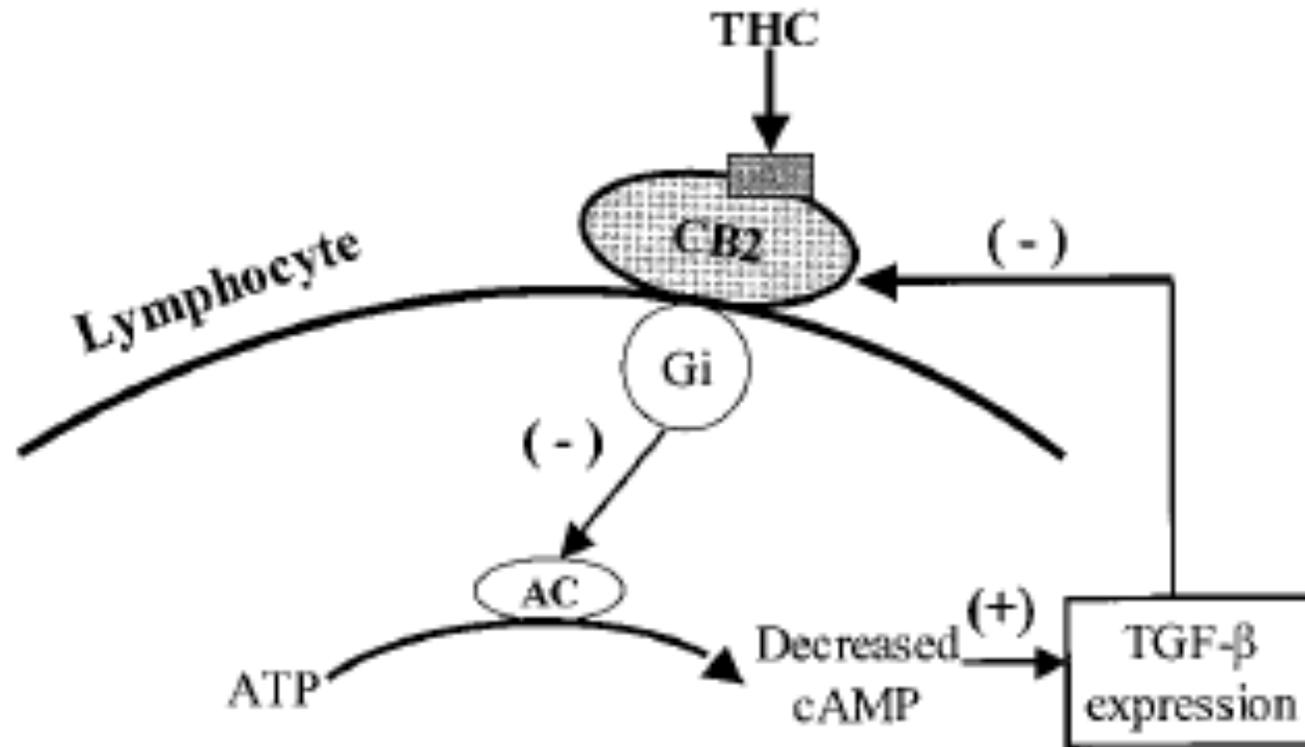
Elevated TGF- β is a bad general in promoting cancer

Immune system out of equilibrium

Autocrine and Paracrine Regulation of Lymphocyte CB2 Receptor Expression by TGF- β

Brian Gardner,^{*,†,1} Li X. Zu,^{*,†,1} Sherven Sharma,^{*,†,‡} Qian Liu,^{§,1} Alexandros Makriyannis,^{§,1} Donald P. Tashkin,[†] and Steven M. Dubinett^{*,†,‡,2}

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REVIEW

Taming THC: potential cannabis synergy and phytocannabinoid-terpenoid entourage effects

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Keywords

cannabinoids; terpenoids; essential oils; THC; CBD; limonene; pinene; linalool; caryophyllene; phytotherapy

Received

19 November 2010

Revised

29 December 2010

Accepted

12 January 2011

Article

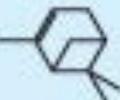
Terpenoids and Phytocannabinoids Co-Produced in *Cannabis Sativa* Strains Show Specific Interaction for Cell Cytotoxic Activity

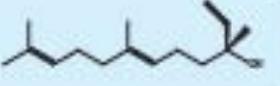
Dvora Namdar^{1,*}, Hillary Voet¹, Vinayaka Ajjampura¹, Stalin Nadarajan¹, Einav Mayzlish-Gati², Moran Mazuz¹, Nurit Shalev¹ and Hinanit Koltai¹

¹ Institute of Plant Sciences, Agricultural Research Organization, Volcani Center, Bet Dagan 7505101, Israel

² Israeli Gene Bank, Volcani Center, Bet Dagan 7505101, Israel

* Correspondence: dvoran@volcani.agri.gov.il

Terpenoid	Structure	Commonly encountered in	Pharmacological activity (Reference)	Synergistic cannabinoid
Limonene		 Lemon	Potent AD/immunostimulant via inhalation (Kosori et al., 1995) Anxiolytic (Carvalho-Feltes and Costa, 2002; Paltini Aidi et al., 2006) via 5-HT _{1A} (Kornya et al., 2004) Apoptosis of breast cancer cells (Vigashin et al., 1998) Active against acne bacteria (Kim et al., 2008) Dermatophytes (Sanguinetti et al., 2007; Singh et al., 2010) Gastro-oesophageal reflux (Harris, 2002)	CBD CBD CBD, CBG CBD CBG THC
α -Pinene		 Pine	Anti-inflammatory via PGE-1 (Zil et al., 1988) Bronchodilatory in humans (Falk et al., 1993) Acetylcholinesterase inhibitor, aiding memory (Ferry et al., 2003)	CBD THC THC, CBD
β -Myrcene		 Herb	Blocks inflammation via PGE-2 (Lorenzetti et al., 1991) Analgesic, antagonized by naloxone (Rao et al., 1990) Sedating, muscle relaxant, hypnotic (de Vile et al., 2002) Blocks hepatic carcinogenesis by aflatoxin (de Oliveira et al., 1997)	CBD CBD, THC THC CBD, CBG
Linalool		 Lavender	Anti-anxiety (Russo, 2001) Sedative on inhalation in mice (Buchbauer et al., 1992) Local anesthetic (Re et al., 2000) Analgesic via adenosine A _{2A} (Peters et al., 2004) Anticonvulsant/anti-glutamatergic (Silabekdy et al., 1995) Potent anti-leishmanial (do Socorro et al., 2003)	CBD, CBG THC THC CBD CBD, THC, CBG ?

β -Caryophyllene		 Pepper	AI via PGE-1 comparable phenylbutazone (Stalle et al., 1988) Gastric cytoprotective (Tambe et al., 1996) Anti-leishmanial (Carpelli et al., 1997) Selective CB ₂ agonist (100 nM) (Gertsch et al., 2008) Treatment of psoriasis? (Kosak et al., 2007) Treatment of addiction? (Xi et al., 2010)	CBD THC ? THC THC CBD
Caryophyllene oxide		 Lemon balm	Decreases platelet aggregation (Lin et al., 2002)	THC CBG, CBG
Terpinolene		 Orange	Sedative (Jirrel et al., 1972) Skin penetrant (Cornwell and Barry, 1994) Potent antimicrobial (Lopes et al., 1999; Rodriguez Goulet et al., 2004) Anti-leishmanial activity (Amada et al., 2005)	THC, CBG - ? ?
Phytol		 Green tea	Breakdown product of chlorophyll Prevents Vitamin A toxicogenesis (Arnhold et al., 2002) TCGA via SSADH inhibition (Yang et al., 2002)	- - CBG



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The cannabinoid CB₂ receptor-selective phytocannabinoid beta-caryophyllene exerts analgesic effects in mouse models of inflammatory and neuropathic pain



biomolecules



Review

Therapeutic Potential of α - and β -Pinene: A Miracle Gift of Nature

Int. J. Mol. Sci. **2014**, *15*, 13637-13648; doi:10.3390/ijms150813637

OPEN ACCESS

International Journal of
Molecular Sciences

ISSN 1422-0067

www.mdpi.com/journal/ijms

Article

Celastrol Attenuates Inflammatory and Neuropathic Pain Mediated by Cannabinoid Receptor Type 2

Molecules **2012**, *17*, 3524-3538; doi:10.3390/molecules17033524

OPEN ACCESS

molecules

ISSN 1420-3049

www.mdpi.com/journal/molecules

Review

Terpenoids as Potential Anti-Alzheimer's Disease Therapeutics

Cannabis Can be Consumed in Multiple ways: Capsules, Powder or Raw juice

ECS & the Gut-Brain-Skin Axis

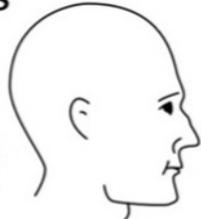
Beta-amyloid, Neurofibrillary tangles



Propionibacterium acnes



Enteric microbiota
Propionibacterium
acnes



Probiotics
Prebiotics
THC
CBD
THCA
CBDA



Cannabis capitata glandular



Cannabis Harlequin Leaves



Combinations of natural products which interact with the Endocannabinoid System

Disease specific solutions



Pathogens and Disease, 74, 2016, ftw059

doi: 10.1093/pathpd/ftw059

Advance Access Publication Date: 11 July 2016

Minireview

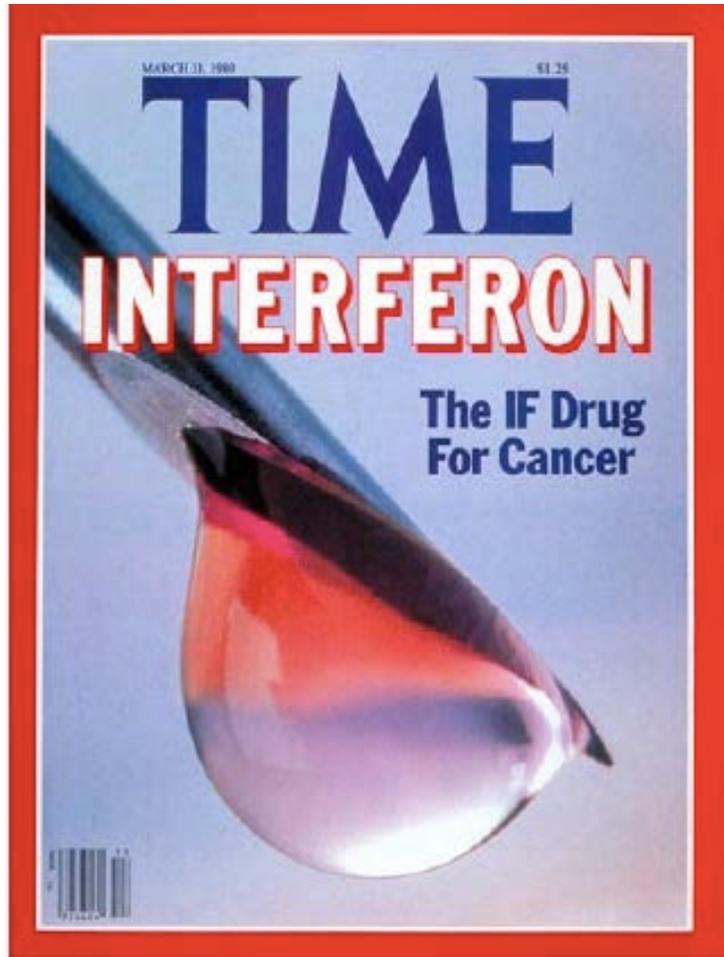
MINIREVIEW

Control of autoimmune inflammation by celestrol, a natural triterpenoid

ABSTRACT

Celestrol is a bioactive compound derived from traditional Chinese medicinal herbs of the Celastraceae family. Celestrol is known to possess anti-inflammatory and anti-oxidant activities. Our studies have highlighted the immunomodulatory attributes of celestrol in adjuvant-induced arthritis (AA), an experimental model of human rheumatoid arthritis (RA). RA is an autoimmune disease characterized by chronic inflammation of the synovial lining of the joints, leading eventually to tissue damage and deformities. Identification of the molecular targets of celestrol such as the NF- κ B pathway, MAPK pathway, JAK/STAT pathway and RANKL/OPG pathway has unraveled its strategic checkpoints in controlling arthritic inflammation and tissue damage in AA. The pathological events that are targeted and rectified by celestrol include increased production of pro-inflammatory cytokines; an imbalance between pathogenic T helper 17 and regulatory T cells; enhanced production of chemokines coupled with increased migration of immune cells into the joints; and increased release of mediators of osteoclastic bone damage. Accordingly, celestrol is a promising candidate for further testing in the clinic for RA therapy. Furthermore, the results of other preclinical studies suggest that celestrol might also be beneficial for the treatment of a few other autoimmune diseases besides arthritis.

Celestrol is a naturally occurring inhibitor of a central inflammatory cytokine IL6



Why the FDA bothered to stop the sale of a product doing so much good in pets.

Then I realized that FDA did not need a reason

► A young FDA investigator came to my Amarillo office and announced her intentions to inspect my facilities. I phoned my FDA consultant, a former FDA employee who had been head of compliance for veterinary medicine. Even though I was an intrastate operation with approval from the Texas Department of health, my consultant advised me to allow her to inspect my laboratory. She spent 4 hours visiting my small facility (only about 500 sq. ft.) and then called her boss in Dallas. I heard her tell her boss over the phone that she could not find anything wrong. After discussing the situation with her boss and obtaining some instructions, she asked to see my records on salt purchase.

► After looking at my salt purchase invoices she announced, "I think I can get you." She then asserted that the salt used in my product had crossed state lines. Even though the IFN α was manufactured in Texas, the bottle was manufactured in Amarillo, the label was printed in Amarillo and sales were only in Texas, the FDA threatened to take me to court unless I destroyed all product and ceased all Pet Interferon Alpha sales because the salt, purchased in Texas, had originally been mined out of Texas. The woman said that there are no salt mines in Texas (not true). I "voluntarily" ceased sales of Pet Interferon Alpha rather than undergo the expense of a battle with the FDA

SCIENTIFIC REPORTS

OPEN

Transient Cannabinoid Receptor 2 Blockade during Immunization Heightens Intensity and Breadth of Antigen-specific Antibody Responses in Young and Aged mice

Received: 31 August 2016
Accepted: 12 January 2017
Published: 17 February 2017

Emmanuel Dotsey¹, Irina Ushach¹, Egest Pone², Rie Nakajima¹, Algis Jasinskas¹,
Donovan A. Argueta³, Andrea Dillon³, Nicholas DiPatrizio³, Huw Davies¹, Albert Zlotnik⁴,
Peter D. Crompton⁵ & Philip L. Felgner¹



Workshop, July 22, 2009 – Public Health Implications of XMRV Infection

Center for Cancer Research (CCR)

Center of Excellence in HIV/AIDS & Cancer Virology (CEHCV)

Introduction – In 2006, the human retrovirus XMRV (xenotropic murine leukemia virus-related virus) was identified and reported to be associated with certain cases of prostate cancer. Although the public health implications of this finding were not immediately clear, a series of presentations at the most recent Cold Spring Harbor Laboratory meeting on Retroviruses provided additional support for this linkage and suggested that the number of individuals infected with XMRV is significant enough to be a cause for public concern. In view of these developments, it was deemed appropriate for NCI to convene a small group of intramural and extramural scientists and clinicians with expertise in this area to provide the NCI leadership with recommendations on future directions. The following summarizes the scientific presentations and resulting round-table discussion among workshop participants.

Organizers

Stuart Le Grice, Ph.D. CEHCV	HIV Drug Resistance Program & Head,
John Coffin, Ph.D. CCR	Tufts University & Office of the Director,

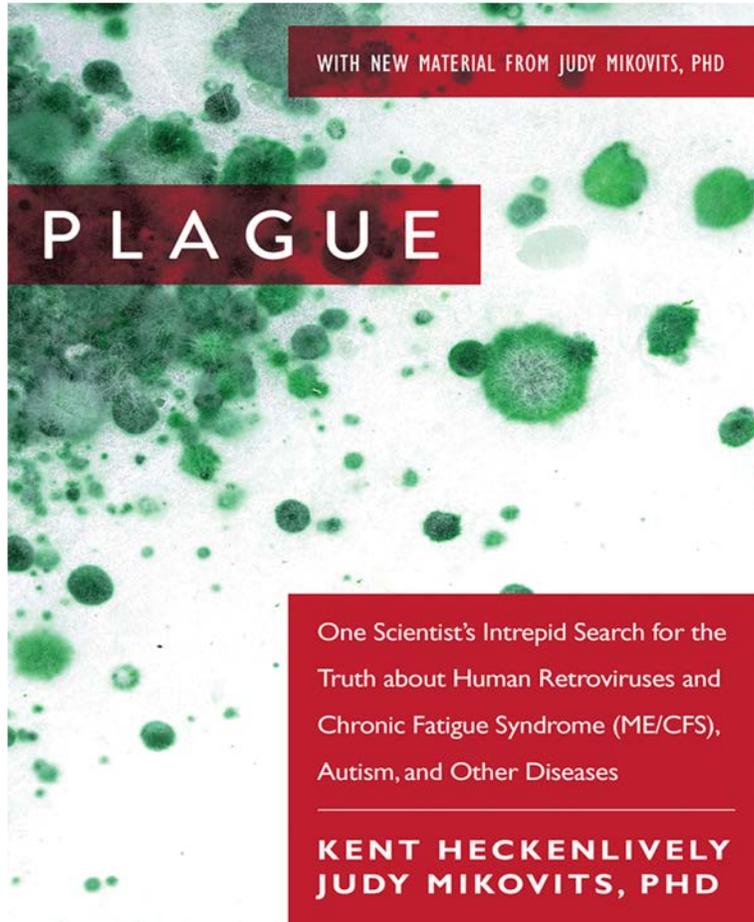
Participants

Carlos Cordon-Cardo, M.D., Ph.D.	Columbia University
Stephen Goff, Ph.D.	Columbia University
Eric Klein, M.D.	Cleveland Clinic
Robert Silverman, Ph.D.	Cleveland Clinic
A. Dusty Miller, Ph.D.	Fred Hutchinson Cancer Research Center
Ila Singh, M.D., Ph.D.	University of Utah
Judy Mikovits, Ph.D.	Whittemore Peterson Institute, University of Nevada

Stephen Hughes, Ph.D.	HIV Drug Resistance Program, NCI
Vineet KewalRamani, Ph.D.	HIV Drug Resistance Program, NCI
Douglas Lowy, M.D.	Laboratory of Cellular Oncology, NCI
John Schiller, Ph.D.	Laboratory of Cellular Oncology, NCI
Chris Buck, Ph.D.	Laboratory of Cellular Oncology, NCI
William Dahut, M.D.	Medical Oncology Branch, NCI
James Gulley, M.D., Ph.D.	Laboratory of Tumor Immunology and Biology, NCI
Jeffrey Schlom, Ph.D.	Laboratory of Tumor Immunology and Biology, NCI
W. Marston Linehan, M.D.	Urologic Oncology Branch, NCI
Charles Rabkin, M.D.	Division of Cancer Epidemiology & Genetics, NCI

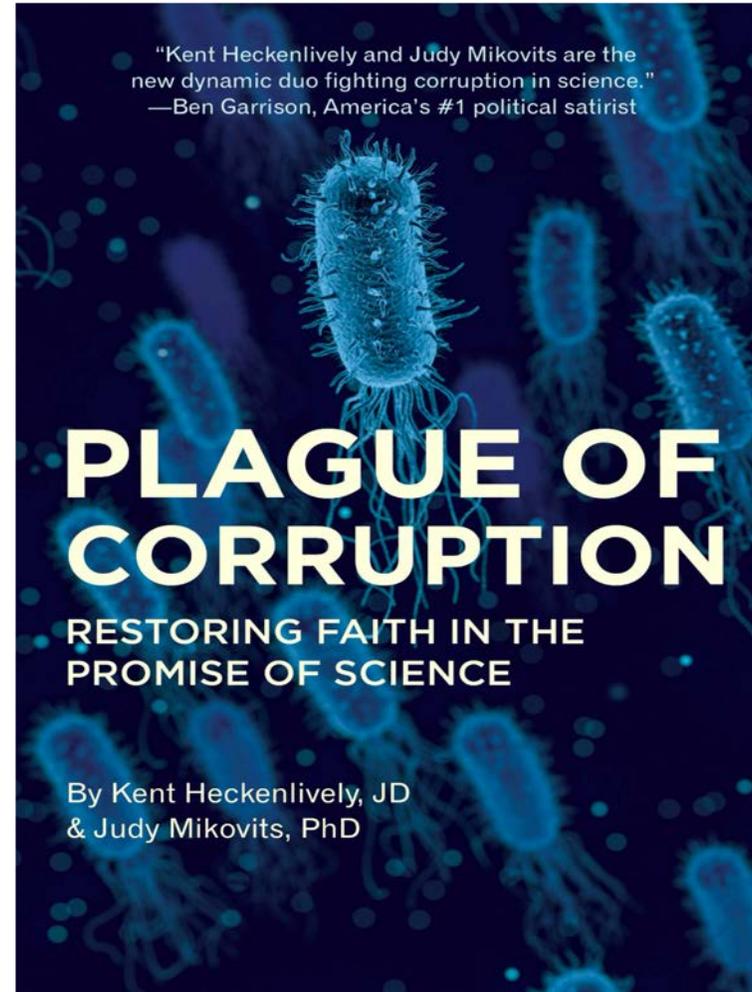
WHO & WHY?

Censorship & Cover-up of Scientific Discovery led to Plague of Chronic Disease



That is the inconvenient truth.

“Retroviruses and environmental toxins led to this explosion of chronic diseases,”



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