TH1NK MP
Autoimmunity Research Foundation
A metagenomic understanding of chronic disease
MAY 2012 • VOLUME 2 • ISSUE 1

Back to Basics

From MPKB, edits by Chris Benediktsson and Janet Raty

The Autoimmunity Research Foundation team presents at international conferences. Progress on pure olmesartan continues and we are in contact with the FDA, but we are most concerned about you, our readers. That is why we are taking this time to review the basics of the MP in an easy-to-read Q&A format for this newsletter.

Q. What is the Marshall Pathogenesis?

A. First, it is helpful to know the word "pathogenesis" means the source or cause of an illness or abnormal condition. A fuller description is "The development of morbid conditions or of disease; more specifically the cellular events and reactions and other pathologic mechanisms occurring in the development of disease." (http://medical-dictionary.thefreedictionary.com/pathogenesis)

"The Marshall Pathogenesis... posits that chronic diseases (Th1 illnesses), are the result of infection by an intraphagocytic, metagenomic microbiota of chronic microbial forms that we often refer to as the Th1 pathogens." (http://mpkb.org/home/protocol)

We often focus only on the "Marshall Protocol" because it is an action or choice. However, the Marshall Pathogenesis explains the logic or reason for an action or choice.

On a relatively modest scale, the pathogenesis describes the science underpinnings of the Marshall Protocol to medical providers, friends and family as a framework for action and choice. We understand the science is replicable, with the correct skills and tools, to verify facts that guide the clinical application of the Marshall Protocol.

In a more global perspective, the Marshall Pathogenesis provides profound direction-changing implications on what is known about disease or disorders. It occupies a unique position in history to clarify previous scientific research, using in silico modeling and other advanced technologies with what is seen in a clinic setting. And in a very real way, the Marshall Pathogenesis refocuses medical solution goals to cure, instead of management, this way:

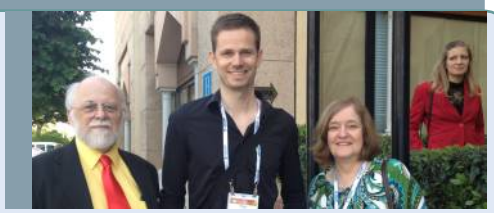
“The Marshall Pathogenesis, upon which the Marshall Protocol is grounded, is a description for how microbes interfere with the innate immune response. These pathogens survive and reproduce by disrupting the Vitamin D Nuclear Receptor, an evolutionarily consistent mechanism for survival, which leads to the development of chronic inflammatory diseases. Because these diseases are fundamentally bacterial in nature, the conditions are referred to as the 'Th1 diseases.' The Marshall Pathogenesis is supported by an emerging array of evidence, including clinical evidence, evolutionary evidence, some in silico data, and environmental sampling studies.” (http://mpkb.org/home/pathogenesis)

Timeline

From MPKB.org, vimeo.com and trevormarshall.com, with edits by Trevor Marshall, Chris Benediktsson and Janet Raty

2012

2012 May, Granada, Spain International Congress on Autoimmunity.

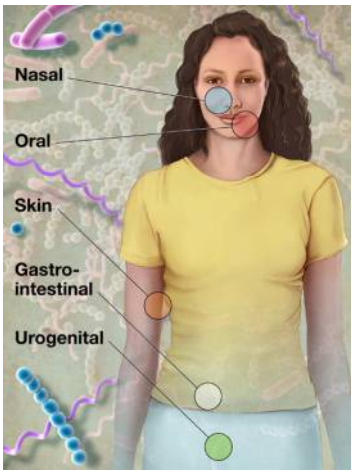


- Lindseth I. Keynote, “Treatment of Chronic Fatigue Syndrome as an Immunological Disorder”
- Marshall TG. Plenary "The Microbiome, which Feeds a Myriad of Autoimmune Diseases” and keynote "The Human Microbiome is the infection at the Heart of Autoimmune Disease"

Q. What is Microbiota?

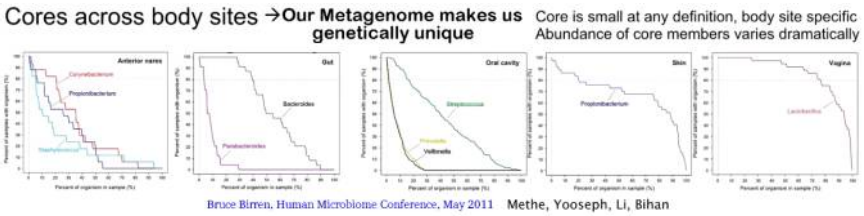
A.

Microbiota are the the microscopic living organisms in a specific region or area, like a human body. They are also called microbial flora. Microbes can be bacteria, fungus, virus or protozoa. According to a recent National Institutes of Health (NIH) estimate, 90% of cells in the human body are bacterial, fungal, or otherwise non-human.
(Turnbaugh PJ, Ley RE, Hamady M, Fraser-Liggett CM, Knight R, Gordon JI The human microbiome project. *Nature.* 2007;449:804-10.)

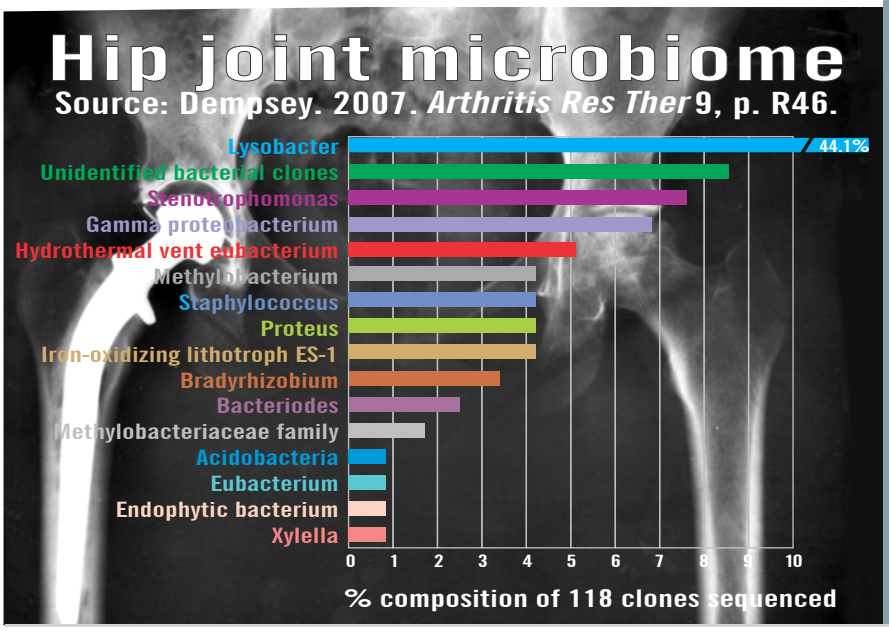


Although experts agree that microbes enjoy a commensal relationship with human hosts, only a fraction of human microbiota have been characterized, much less identified. The sheer number of non-human genes represented by human microbiota communities—there are millions in our "extended genome" compared to the nearly 23,000 in the human genome—implies we have just begun to fathom the full extent microbes act to facilitate their own survival.
(www.ncbi.nlm.nih.gov/pubmed/20388071)

The NIH's ongoing initiative, the Human Microbiome Project, has started to catalog the human microbiome, also referred to as the human metagenome, because traditional in vitro methods for culturing individual species in the lab have drastically under-represented the size and diversity of actual microbe populations.



One study employed a newer technology, high throughput genomic sequencing, on removed prosthetic hip joints. Those results revealed the previously-unknown presence of hydrothermal vent eubacteria, a species once thought only to persist in the depths of the ocean.
(http://mpkb.org/home/pathogenesis/microbiota)



2011

2011 January Proal AD, Albert PJ, Blaney GP, Lindseth IA, Benediktsson C, Marshall TG. Journal publication: *Cellular and Molecular Immunology,* "Immunostimulation in the era of the metagenome."

2011 April Dalian, China Marshall TG, BIT's 2nd World DNA and Genome Day, "The human microbiome lies at the heart of autoimmune disease."

2011 May, Dalian, China Marshall TG, NeuroTalk 2011, "The human microbiome is the mechanism fueling neurodegenerative disorders."

2011 November, Singapore Marshall TG. 5th Asian Congress on Autoimmunity "Why vitamin D is more effective in early stage disease than late stage disease." Biological complexity of Human Microbiome leads to semi-infinite Interactome, and combined metagenomes from microbes living in and on human body accumulate to cause chronic disease/Autoimmune Disease.

2011 November, Singapore Goetze-Pelka R. 5th Asian Congress on Autoimmunity, "Psychiatric and neurologic comorbidities as systemic dysfunctions."

2011 December Raty J, Benediktsson C, Marshall TG. ARF Newsletter Publication begins: *Th1nk MP*.

2010

2010 Proal AD, Albert PJ, Marshall TG. Book chapter: *Metagenomics of the human body,* "Autoimmune disease and the human metagenome."

2010 May, Ljubljana, Slovenia 7th International Congress on Autoimmunity,
Presentations:
- Marshall TG. "Olmesartan overcomes antibiotic resistance, inducing recovery from advanced autoimmune disease"
- Blaney G. "Olmesartan medoxomil and treatment of autoimmune disease"
- Proal A. "Metagenomic symbiosis between bacterial and viral pathogens in autoimmune disease"

Posters:
- Raty J, Benediktsson C, Marshall TG. "Bipolar disorders and autoimmune disease share a similar etiology"
- Albert P. "Web 2.0 offers new opportunities for patient care and research"

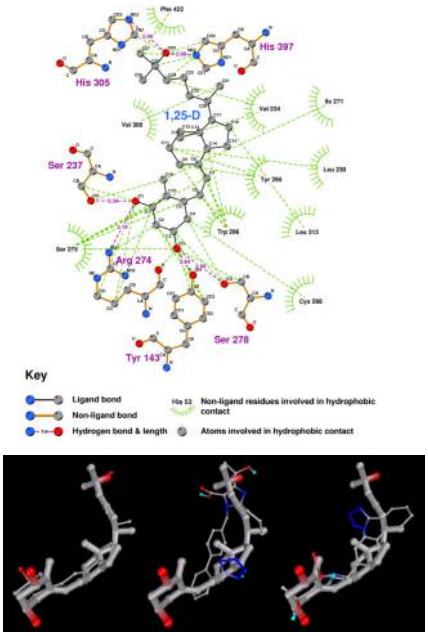
2010 Aug St. Louis, MO Proal AD. Human Microbiome Research Conference, "Successive infection: a model for how metagenomic communities shift to become more pathogenic over time."

2010 Aug St. Louis, MO Albert PJ. Human Microbiome Research Conference "Multiple reports of symptom exacerbation on immunostimulatory treatment for autoimmune disease."

2010 Sep Clydebank Scottland Marshall TG. The Scottish Summit on Vitamin D and Multiple Sclerosis, "MS Pathogenesis and Impact of Vitamin D Supplementation on Disease Progression." (http://vimeo.com/15278736)

2010 Oct Marshall TG, seminar at 4M-Klinikken in Oslo, Norway. (http://vimeo.com/17444948)

2010 Dec ARF launches online video conferences. Guest speakers, announcements of pure olmesartan research, production and submissions to FDA. (http://marshallprotocol.com/conferences/)



[Top] 2D LigPlot of 1,25-D bound into the VDR ligand binding pocket. **Note:** The core structure of the hydrogen-bonded residues is expanded to a 'ball-and-stick' format, so as to show the atoms involved in hydrogen bond formation. (http://www.tbiomed.com/content/3/1/1/figure/F5)

[Bottom] VDR-docked configurations for 1,25-D and Olmesartan, with superimposition showing both conformations. **Note:** Models depicted as "thick" and "thin" solely for visual clarity. Carbon atoms shown as grey, oxygen shown as red, nitrogen as blue, polar hydrogen as blue-white. Non-polar hydrogens not displayed. (http://www.tbiomed.com/content/3/1/1/figure/F3)

2006 Trevor G Marshall, Robert E Lee and Frances E Marshall
Theoretical Biology and Medical Modelling, **"**Common angiotensin receptor blockers may directly modulate the immune system via VDR, PPAR and CCR2b**"**
2006 3:1 doi:10.1186/1742-4682-3-1
(http://www.tbiomed.com/content/3/1/1)

Q. What is the VDR and how does it work?

A.

The NR1I1 or VDR (also called the calcitriol receptor) is a nuclear receptor, a class of proteins found within the interior of cells. These proteins are responsible for sensing the presence of hormones and certain other molecules. Nuclear receptors are unique from other classes of receptors because of their ability to directly interact with and control the expression of genomic DNA.

Some molecules (or ligands) which bind to the nuclear receptor activate (agonize) and some inactivate (antagonize). It is believed approximately 95% to 98% of ligands inactivate nuclear receptors. Nuclear receptors play a significant role in immune response and many drugs, supplements and substances in food and drink are immunosuppressive. Expression of a large number of genes is regulated by nuclear receptors and ligands that activate receptors have profound effects.

Different cell types have different nuclear receptors. Immune cell VDR use two endogenous or "native" ligands, the two main forms of vitamin D in the human body: 25-hydroxyvitamin D (25-D) and 1,25-dihydroxyvitamin D (1,25-D). Non-native or exogenous ligands also inactivate or activate, depending on their molecular structure.

Ligands ***compete*** to dock into nuclear receptors. A greater molecule concentration can displace competing molecules. Affinity occurs in a logarithmic or sliding scale. VDR binding with ligand 1,25-D tends to be much less common than 25-D by a factor of 1,000 or more. But an increase in 1,25-D and decrease in 25-D can tilt the odds in favor of 1,25-D, and vise versa. Affinity and whether a ligand inactivates or activates a nuclear receptor can be validated with in silico modeling. Although less precise, it is also possible to measure this in vitro.

When activated by 1,25-D, the VDR transcribes thousands of genes. The VDR functions in regulating calcium metabolism. It is becoming increasingly clear, however, that the clinically-accepted role of Vitamin D metabolites—regulating calcium homeostasis—is just a small subset of the functions actually performed by these hormones.

2009

2009 Five peer-reviewed papers from ARF Research**:**
- Proal AD, Albert PJ, Marshall TG, *Autoimmunity Reviews* "Autoimmune disease in the era of the metagenome"
- Albert PJ, Proal AD, Marshall TG, *Autoimmunity Reviews* "Vitamin D: the alternative hypothesis"
- Blaney G, Albert PJ, Proal AD, *Annals of the New York Acade5my of Sciences* "Vitamin D metabolites as clinical markers in autoimmune and chronic disease"
- Proal AD, Albert PJ, Marshall TG, *Annals of the New York Academy of Sciences* "Dysregulation of the Vitamin D Nuclear Receptor may contribute to the higher prevalence of some autoimmune diseases in women"
- WaterhouseJC, PerezTH, Albert PJ, *Annals of the New York Academy of Sciences* "Reversing bacteria-induced Vitamin D Receptor dysfunction is key to autoimmune disease"

2009 July, Chengdu, China ARF collaboration. West China Hospital trial of Marshall Protocol on ankylosing spondylitis.

2009 Aug, Dailan, China Marshall TG. 4th China Medicinal Biotech Forum"The VDR nuclear receptor is a novel proxy for MTSS1 and MTUS1 in breast, bladder and colorectal cancers."

2009 May, Beijing, China Proal AD, International Congress of Antibodies "Antibodies and infection in the era of metagenome."

2009 Apr, Prague, Czech Republic Marshall TG, Workshop on Chlamydial Infection "Clinical observations."

2009 Apr, Prague, Czech Republic Marshall TG Workshop on Chlamydial Infection "It is time to bury Koch—Infectious Disease transitions to an understanding of the Metagenome."

2008

2008 April UCLA Marshall TG. Aging "The VDR nuclear receptor is key to understanding 'diseases of the aging'."

2008 February Marshall TG. *Bioessays*, "Vitamin D discovery outpaces FDA decision making." (www.ncbi.nlm.nih.gov/pubmed/18200565)

2008 September Portugal Marshall TG. Session chair on Vitamin D at Porto International Congress on Autoimmunity. Also:
- Marshall TG. "VDR receptor competence induces recovery from chronic autoimmune disease"
- Perez TH. "Bacteria induced vitamin D receptor dysfunction in autoimmune disease: theoretical and practical implications for interpretation of serum vitamin D metabolite levels"
- Proal AD. "Vitamin D induced dysregulation of nuclear receptors may account for higher prevalence of some autoimmune diseases in women"
- Blaney G. "Vitamin D metabolites as clinical markers in autoimmune and chronic illness"

2008 December Foshan, China Marshall TG. Keynote at World Gene Congress: "Understanding human disease requires study of a metagenome, not just the human genome."

2008 December Sichuan University Marshall TG. Clinical seminar at West China Hospital: "The Marshall Protocol in a clinical environment—observations from the initial cohort."

2007

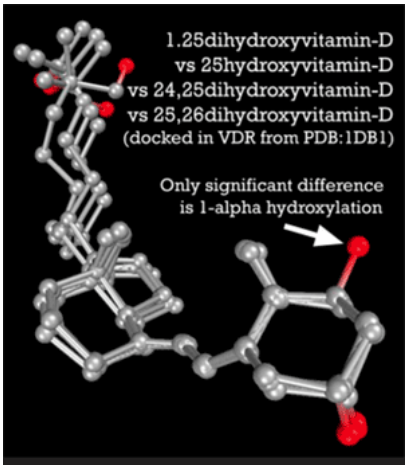
2007 July Marshall presents abstract at Metagenomics 2007, **"**Bacterial Capnine Blocks Transcription of Human Antimicrobial Peptides." Proof of concept for hypothesis that disease-causing bacteria can produce ligands which disable the Vitamin D Receptor.

One of the VDR's key functions is the transcription of antimicrobial peptides. In humans, when the VDR is activated, TLR2 is expressed on the surface of certain cells to recognize native or foreign substances, and then passes on appropriate signals to the cell and/or the nervous system. When activated, TLR2 also allows the immune system to recognize gram-positive bacteria, including Staphylococcus aureus, Chlamydia pneumoniae, and Mycoplasma pneumoniae and TLR2 protects against intracellular infections such as Mycobacteria tuberculosis.
(http://mpkb.org/home/pathogenesis/innate_immunity#nuclear_receptors_and_ligands .
Additional references found in MPKB.org article.)

In *Homo sapiens*, or humans but not other species, Cathelecidin, TLR2 and beta-Defensins are transcribed by the VDR.
(http://mpkb.org/home/publications/marshall_cancer_2009)

Q. What is vitamin D?
A.

During the last century, through early perceptions and tools, "vital amines" were identified and given alphabetical designations. "Vitamin D" was misidentified as a necessary dietary nutrient instead of being recognized as a secosteroid, tightly regulated by the body. We now know all forms of D belong to a family of lipids called secosteroids—very similar in structure to steroids, except two B-ring carbon atoms of the typical four steroid rings are not joined as they are in steroids.

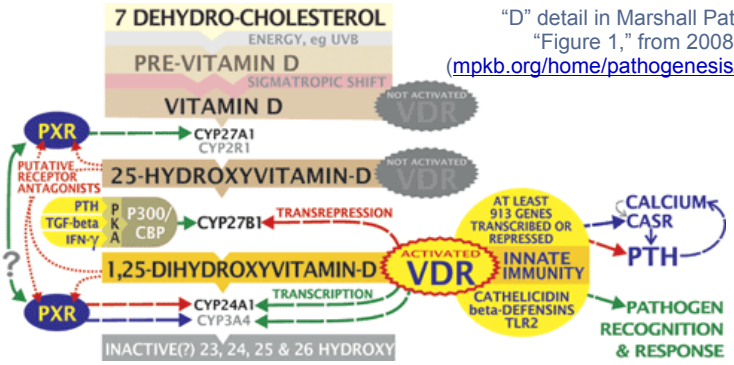


1,25-D is different than 25-D in that it possesses a single 1-alpha hydroxylation. (See arrow.)

The additional hydroxylation stabilizes helix 12 in the VDR, binding the promoter which allows VDR activation and subsequent transcription of thousands of genes.

The level of each D metabolite is affected by a complex network of feedback interactions involving multiple enzymes and receptors, further revealing mis-categorized vitamin D is regulated more like a steroid than a nutrient.

In scientific literature, researchers sometimes distinguish between "steroid" and "secosteroid," but not always. This is an indication of how secosteroids behave. All vitamins D act very much like steroids, binding nuclear receptors and modulating immune response. Growing evidence reveals secosteroids D causes adverse side effects like chronic disease over time, just like anabolic steroids and corticosteroids. (http://mpkb.org/home/pathogenesis/vitamind)



"D" detail in Marshall Pathogenesis "Figure 1," from 2008 *Bioessay*. (mpkb.org/home/pathogenesis/vitamind)

2006

2006 January Marshall TG, Lee R, Marshall F. *Theoretical Biology & Medical Modeling* paper explains how angiotensin receptor blockers directly modulate immune system via the VDR, PPAR and CCR2b nuclear receptors. (http://ncbi.nlm.nih.gov/pubmed/16403216)

2006 March Marshall TG. Visiting Professor Lecture Series, FDA's CDER, "Molecular genomics offers new insight into the exact mechanism of action of common drugs: ARBs, statins and corticosteroids." (http://mpkb.org/home/publications/marshall_fda_cder_2006)

2006 March ARF awarded orphan drug status for antibiotic minocycline.

2006 June LA, California, USA ARF conference, "Recovering from Chronic Disease: Sarcoidosis, Autoimmunity, AIDS and Cancers." Notable speakers include Alan Cantwell MD.

2006 July Marshall TG. US Patent and Trademark Office (published June 2007) How an antibiotic-based therapy can treat and prevent AIDS and cancer.

2006 July Marshall TG. Poster at "Days of Molecular Medicine," the first of three annual presentations alternating between Karolinska (Sweden) and Harvard (USA).

2006 October Marshall TG. American Academy of Environmental Medicine, *"A new approach to treating intraphagocytic CWD bacterial pathogens in sarcoidosis, CFS, Lyme and other inflammatory diseases."*

2006 December Marshall TG. Second US patent filed for broader range of chronic diseases and method of killing intracellular bacteria.

2006 Blaney G, et al. *"High levels of active 1,25-dihydroxyvitamin D despite low levels of the 25-hydroxyvitamin D precursor: implications of dysregulated vitamin D for diagnosis and treatment of chronic disease. Vitamin D: New Research."*

2005

2005 March, Chicago ARF's 'Recovering from Chronic Disease' conference. Speakers include Lida Mattman PhD and UK Dr. Andrew Wright.

2005 June Marshall publishes English version of Russian paper "Antibacterial Therapy Induces Remission in Sarcoidosis."

2005 July Marshall TG. US Patent and Trademark Office (Published February 2006), novel method of killing stealth intracellular bacteria which cause many Th1 and "autoimmune" diseases.

2004

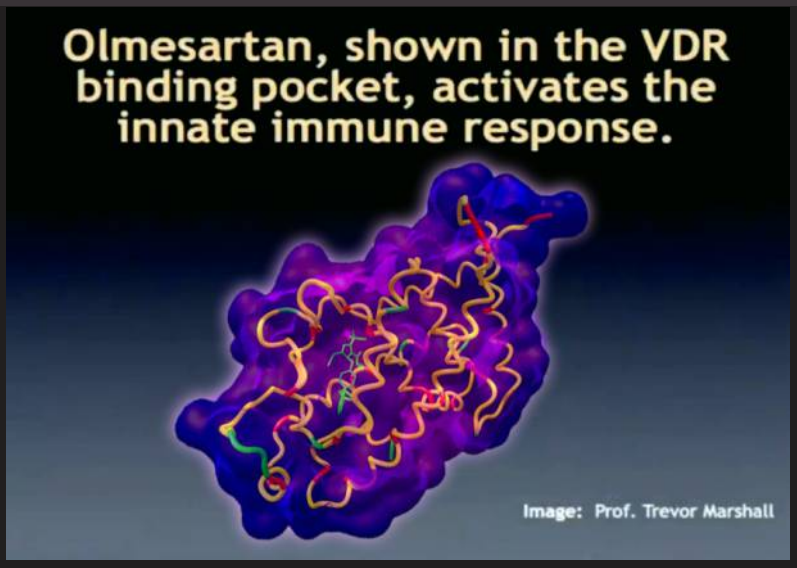
2004 April Autoimmunity Research Foundation established.

2004 June Marshall F. Marshall TG. online peer-reviewed journal *Autoimmunity Reviews*, later in print edition, "Sarcoidosis succumbs to antibiotics: implications for autoimmune disease." (http://mpkb.org/home/publications/marshall_autoimmunity_reviews_2003)

2004 July Marshall speaks at 4th International Congress on Autoimmunity in Budapest.

2004 July Marshall registers MarshallProtocol.com website. Aussie Barb joins and begins invaluable study site role.

2004 December Kazan, Russia *The Journal of the Interregional Clinical-Diagnostic Center*, (ISSN: 1726-6149), Russian translation, Marshall's paper "Antibacterial Therapy Induces Remission in Sarcoidosis" in a special issue.



"Olmesartan is safe and well-tolerated at doses from 120-240mg/day." Dr Greg Blaney, of Vancouver, BC, reported at the 7th International Congress on Autoimmunity, held in Ljubljana, Slovenia, May 2010:
"Olmesartan Medoxomil: a novel VDR agonist and sub-inhibitory antibiotics in the treatment of advanced autoimmune disease."
(http://youtu.be/X0y0PcVJ5Ss or http://mpkb.org/home/publications/blaney_autoimmunity_2010)

A transcript of this presentation is available at:
http://autoimmunityresearch.org/abstracts/Ljubljana_Blaney_transcript.pdf

Q. What is olmesartan? How does it work?

A.

Patients on the Marshall Protocol (MP) take olmesartan (Benicar), a drug whose actions are well known, every six hours. A growing body of research supports the use of olmesartan as a part of a curative therapy for chronic disease. In general, olmesartan tends to be prescribed for its antihypertensive properties due to the fact that is an angiotensin receptor blocker.

For the purposes of the MP, olmesartan has two primary actions: it reduces inflammation by blocking the Nuclear Factor-kappaB cytokine pathway and it is an agonist of the Vitamin D Receptor (VDR). As a VDR agonist, olmesartan activates the innate immune response. Research supports the safety of doses used by MP patients. Olmesartan has minimal interactions with other drugs and is one of the safest drugs on the market.

A growing body of research supports the use of olmesartan as a part of a curative therapy for chronic disease. In general, olmesartan tends to be prescribed for its antihypertensive properties due to the fact that is an angiotensin receptor blocker.

Q. Why, why, why does the MP take so darned long?

A.

The MP takes on an individualized timetable to safely remove pathogens, working with each individual's immune system via stimmulation. It is tempting to compare the MP to immunosuppression—a quick goal to mask or manage symptoms—but working with a body's immune system takes time.

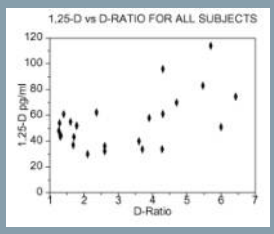
Length of treatment varies per individual by:
Degree of illness – If disease is advanced, symptoms debilitating, or

2003

2003 January Marshall meets Dr. Alan Cantwell; papers by Lida Mattman discussed.
(http://www.ncbi.nlm.nih.gov/pubmed/8711683 , http://www.ncbi.nlm.nih.gov/pubmed/4111724 , and http://www.ncbi.nlm.nih.gov/pubmed/4100503)

Seperately, images from Emil Wirostko's group finally confirm cytoplasmic microbiota of intraphagocytic, L-form bacteria drive biochemical changes observed in autoimmune disease sarcoidosis.
(http://www.ncbi.nlm.nih.gov/pubmed/15043554 , http://www.ncbi.nlm.nih.gov/pubmed/8622416 , and http://www.ncbi.nlm.nih.gov/pubmed/7892050)

2003 January Marshall's multi-drug Protocol takes root. Marshall TG, Marshall F. *Clinmed*, "New Treatments Emerge as Sarcoidosis Yields Up its Secrets"
(http://clinmed.netprints.org/cgi/content/full/2003010001)



Scatter plot of all serum 1,25-D and serum 25-D values measured in study cohort of sarcoidosis patients (21 patients, 24 data sets). Data from "New Treatments Emerge as Sarcoidosis Yields Up its Secrets" (http://clinmed.netprints.org/cgi/content/full/2003010001)

2002

2002 March Marshall registers Sarcinfo.com forum website to discuss the therapy for those with sarcoidosis.

2002 May Marshall introduces ARB olmesartan (Benicar).

2002 August Marshall TG, Marshall F. *NetPrint*, "Valsartan Dosing Regime Modulates Psychotic Events in Two Sarcoidosis Patients."
(http://clinmed.netprints.org/cgi/content/full/2002080006)

The case report shares early observations that valsartan (Diovan) induced hallucinations and psychedelic dreams. Pathogenic description of sarcoidosis described in *Netprint,* "Remission in Sarcoidosis."
(http://clinmed.netprints.org/cgi/content/full/2002080004)

2002 September "Remission in Sarcoidosis" (summary) published at Mercola.com. Early adopter Belinda Fenter begins pulsed minocycline.

2002 December Early adopter Meg Mangin begins pulsed tetracycline.


CMAJ October 15, 2002 vol. 167 no. 8

Letters

Puzzling vitamin D results

Trevor G. Marshall

Research Director, Yarc Inc., Thousand Oaks, Calif.

I am puzzled by the seasonal mean values for 1,25- dihydroxy vitamin D [1,25-$(OH)_2$D] published in Table 2 of the article by Diana Rucker and colleagues.[1] They are about twice as high as those from a similar study done in Denmark,[2] which showed a mean of 29 pg/ml (75.4 pmol/L).

Two of the seasonal mean values (168.1, 148.9) are above the normal range quoted for the assay (45–145). This assay range seems to be correct, but the study data seem to be high.

I am particularly concerned that this study did not place much greater emphasis on the values of the active hormone 1,25-$(OH)_2$D than on the intermediate metabolite 25-hydroxy vitamin D [25(OH)D]. This is especially important in elderly populations, as extra- renal hydroxylase activity in inflammatory macrophages has been shown to generate a normal 1,25-$(OH)_2$D value from depressed levels of circulating 25(OH)D.

Trevor G. Marshall Research Director Yarc Inc. Thousand Oaks, Calif.

References

1. Rucker D, Allan JA, Fick GH, Hanley DA. Vitamin D insufficiency in a population of healthy western Canadians. *CMAJ* 2002;166(12):1517–24.[Abstract/Free Full Text]
2. Brot C, Jorgensen NR, Sorensen OH. The influence of smoking on vitamin D status and calcium metabolism. *Eur J Clin Nutr* 1999;53(12): 920–6. [Medline]

http://www.cmaj.ca/content/167/8/849.1.full

vital organs severely compromised, immunopathology takes more time. MP patients must improve with all due caution and decide what level of symptoms are tolerable while a doctor monitors biological processes to make sure they stay within acceptable limits.

Degree of health desired.

Prior use of immunosuppressants and immunomodulatory medications.

Fibrosis – When the immune system fails to kill a pathogen, it encases diseased tissue in collagen. This is known as fibrosis. Individuals with fibrosis can expect to be working fibrosis pockets for an extended length of the treatment, as tissue slowly remodels. (http://mpkb.org/home/patients/mp_duration)

Chris Benediktsson (Tiburon, California, USA) – Executive Vice President of ARF. Over 30 years experience as a senior manager with both the private and public sector, ranging from operations management of an international media company, to senior staff with the State of Alaska Court System. He has a lifelong interest in practical science, prepared environmental assessments for the FAA, drafted municipal code for regulation of recreational water resources for the Municipality of Anchorage, and lobbied and presented technical testimony to state legislature, served as an accounting, business development and management consultant for private and public sector organizations including Municipality of Anchorage, Lottery Alaska, ABC Alaska, Builder's Bargains Stores, and several Alaska Native corporations

Janet Raty (Portland, Oregon, USA) – ARF research. BYU (UT) BFA in Illustration and MsEd in Supervision and Administration from Bank Street College of Education (NY). Her career spans traditional print and online content publishing, but features educational leadership and assisting C-level executives and strategists visually describe, publish and implement.

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1974-2001

1974 Papua New Guinea sun effect compared to 1986 Sweden, Marshall realizes D associated with disease.

1978-1982 Marshall TG. PhD thesis portable, programmable pumps pulse GnRH and LHRH to treat cryptorchidism, and male and female infertility. (www.ncbi.nlm.nih.gov/pubmed/6135766 and www.ncbi.nlm.nih.gov/pubmed/6428734)

1981 Toronto, Canada Marshall views early IBM in silico model: a few nano-seconds of life of a humulin molecule.

1984 Marshall TG. PhD thesis "Modeling and simulation in diabetes care." Mathematical modeling of human disease.

1999 California, USA. Sartans 8-hr cycle calculated.

2001 Study of sarcoidosis neurological response to ARBs. Marshall submits paper explaining bacterial pathogenesis etiology of sarcoidosis. Three revisions rejected as having no potential interest for readers.

2001 March First early adopter, Elaine E. begins ARB.

2001 December Nilsson et al. ***Journal of Infectious Disease*** shows deceased sarcoidosis patients contain ***Rickettsia helvetica*** genetic material in cell cytoplasm of sarcoid granulomas. (www.ncbi.nlm.nih.gov/pubmed/11930323)

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autoimmunityresearch.org

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A metagenomic understanding of chronic disease
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