Silicon Valley Health Institute

Host of the Smart Life Forum

Next Meeting: Thursday, March 19, 2015

Main Presentation: Aristo Vojdani, PhD, MSc, CLS

"How Environmental Factors Induce Autoimmune Disorders"

Smart Life Forum Presentation Location

Cubberley Community Center

Room H1

4000 Middlefield Road

Palo Alto, California

Directions on our website:

www.SVHI.com

For those who cannot attend, you can view livestreaming at

http://bit.ly/Zpld3o

See our archived videos at

http://tinyurl.com/smartlifeforum

Meet Aristo Vojdani!
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Announcements & Upcoming Events

Upcoming Speakers:

APRIL 2015
Harry Massey - "Energy Medicine"

MAY 2015
Christopher Shade, PhD - "How To Detox"

Upcoming Foundation for Mind Being Research Meeting (FMBR)

Saturday, March 28, 2015 @ 7:30pm

Jerry Kroth, PhD - "Aliens and Man"

Please visit www.FMBR.org for more info.

If you have questions please email *susanrdowns@hotmail.com.*

Thank you.

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Presentation Speaker: Aristo Vojdani, PhD, MSc, CLS!

Aristo Vojdani is a professor of neuroimmunology, Carrick Institute for Graduate Studies. Faculty member, Preventive Medicine, Loma Linda University. Faculty member, National University of Health Sciences at the Lincoln College of Professional, Graduate and Continuing Education. Past associate professor, Charles Drew/UCLA School of Medicine and Science. His research on environmental triggers in complex diseases resulted in the development of numerous antibody arrays for the detection of many autoimmune disorders. Holds 15 US patents for laboratory assessments. Published 160 scientific articles. CEO and Technical Director, Immunosciences Lab. Chief Scientific Advisor, Cyrex Labs. On the editorial board of six scientific journals. Received the Herbert J. Rinkel Award, the Linus Pauling, PhD Award, and the F. R. Carrick Research Institute's Lifetime Achievement Award.

(End of Meet Aristo Vojdani!)

Main Presentation
by Aristo Vojdani, PhD, MSc, CLS

"How Environmental Factors Induce Autoimmune Disorders"

The human body is an incredibly complex system, and since time immemorial man has struggled to work out what exactly his gut and his brain have to do with his health, and whether perhaps the two are connected somehow. Today we know that the parallels between the gut and brain immune systems are too self-evident to deny. These similarities extend to the actual structures, mechanisms and even biochemistries of the two systems: the gut immune barrier and the blood-brain barrier (BBB). Bidirectional signaling between the brain and gut has been confirmed by numerous studies. In fact, this communication between the gut and brain is ongoing from birth, and plays a significant role in shaping how the brain is wired. The gut's influence on the brain cannot be overestimated, so much so that it can be called a second brain. Studies have linked gut microbiota dysbiosis to brain-linked disorders such as depression, anxiety, multiple sclerosis, autism, and autoimmune disorders.

Human autoimmune diseases affect roughly 5-10% of the world's population and impose a significant burden on the quality of life and health care resources through morbidity and mortality. The development of an autoimmune disease is a very complex process. Autoimmunity arises when the host's immune system is directed against self-tissue antigens. Accumulating evidence has suggested a close interplay between genetic factors and environmental triggers such as infections, toxicants and some dietary components in the pathogenesis of autoimmune diseases. In relation to the role of heritability in autoimmunity, genome-wide association studies have reported that genetics accounts for only a minority of autoimmune disorders. Consequently, since 1997 research and publications devoted to environmental triggers in autoimmunity has grown by an average of 7% every year.

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The mechanisms by which environmental factors induce autoimmunity have been described variously as involving gut microbiota dysbiosis, enhanced intestinal permeability, cross-reactivity, over-stimulated or dysregulated activation of innate and adaptive immune response, aberrant cell death, or the binding of toxicants to tissue proteins with the subsequent formation of neoantigens. Based on these mechanisms, infection, xenobiotics and dietary components can induce alterations in self-tissue proteins to which the immune system is normally self-tolerant, consequently eliciting cellular or IgG, IgM or IgA antibody response, resulting first in autoimmune reactivity and subsequently in autoimmune disease. The antibodies produced in this response can be detected 5-10 years before the actual onset of various autoimmune disorders, so that a window of opportunity exists for early intervention and prevention of autoimmune disease.

Based on this interaction between the gut and brain immune systems, the following will be the key concepts of this presentation:

Key Concepts of this Presentation

- The gut and brain are co-dependent parts of a complex immune system, interacting with and affecting each other's function.
- Gut microbiota play a significant role in the gut-brain axis.
- Modern lifestyles and environmental triggers, by changing the gut microbiota, are contributing to the autoimmune epidemic in our society today.
- Environmental triggers, either directly or by induction of oxidation or citrullination (conversion of arginine to citrulline) contribute to gut barrier and BBB dysfunction and autoimmunities.
- The mechanisms by which xenobiotics, infection and dietary proteins induce autoimmune reactions.
- Predictive antibodies can be detected 5-10 years before the actual onset of various autoimmune disorders. This provides a window of opportunity for early intervention and prevention.

Using these predictive antibodies, clinicians can DETECT the environmental triggers, REMOVE these triggers from the patient's environment, then REPAIR the patient's disrupted intestinal and/or blood-brain barriers.

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Editor's Notes

Dr. Vojdani is in my opinion is the world's leading expert in this area. Examples of environmental toxicants that compromise the body's ability to distinguish between self and others include Bisphenol A (BPA), (Kharrazian 2014); mercury, (Gill et al. 2014); asbestos (Pfau et al. 2014); trichloroethene (also called trichloroethylene or TCE) (Gilbert KM et al 2014); organic solvents and air pollution (Calderon-Garciduenas et al 2015); mold/ mycotoxins (Campbell AW et al. 2003) and possibly periodontal infections (Burazor I et al.2014).

Dietary triggers include any food to which a sensitivity develops. Common culprits are gluten, milk, lectins (Vojdani A 2015), a Western diet (Myles 2014) and possibly salt. Salt influences the development of naive CD 4 helper T cells into the pathogenic Th 17 cells and hence affect the innate immune system and macrophage function. (Kleinewietfeld Metal. 2013).

In addition to environmental influences, composition of the intestinal microbiota shapes a healthy immune response or predisposition to disease (Round, Mazmanian 2009). If the microbiome is disrupted, it can become permeable allowing food particles to pass through it. The body's immune system sees such food particles as foreign invaders and mounts an immune response against these escaped food particles. This immune response includes antibodies which attack the food substance. Through molecular mimicry (mistaken identity), these food antibodies can attack genetically similar parts of the body. As an example, gluten is very highly processed in the US and has many components against which a person can develop antibodies. If a person develops antibodies against gluten, they are called "gluten sensitive" which is far more common than celiac disease.

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If a person is sensitive to one of the many components in gluten, the antibodies may attack genetically similar organs such as the thyroid, purkinje cells in the cerebellum (the balance cells) and the beta cells in the pancreas (e.g., GAD 65 antibodies and/or islet cells). This could lead to Hashimoto's Thyroid disease, cerebellar disorders (gluten ataxia), and an autoimmune type of diabetes leading to a need for taking insulin (diabetes 1.5). A recent study found changes in the microbiome to be directly associated with the development of diabetes 1 (Kosti et al. 2015).

The gut is intimately connected to the brain. A permeable ("leaky") gut is thought to be associated with a permeable blood-brain-barrier, which would allow unwanted toxins to enter into the brain. The standard antigliadin antibody test performed by physicians will not accurately measure antibodies to all of the components in gluten.

Diet, stress (Hawrelak, Myers, 2004), oxidative stress (Qiao et al 2013), and environment affect the intestinal microbiome. Dietary causes of dysbiosis include fructose (Payne et al. 2012), food additives (Csaki 2011), artificial sweeteners (Payne et al 2012), and infant formula as infant (Guaraldi, Salvatori 2012), maternal diet (Myles et al. 2013), a high fat diet (de wit et al. 2012) and any food to which the person is sensitive can all lead to dsybiosis. The standard American diet shifts the microbiota within 24 hours (Tumbaugh et al. 2009).

Environmental factors that can lead to dysbiosis include heavy metal exposure (Fazell et al. 2011); exposure to magnetic fields (Medvedeva et al, 2012); airborne particulate matter (Salim et al 2013); Glyphosate (Monsanto's popular Roundup herbicide) (Samsel , Seneff 2013); antibiotics, maternal flora (Fanaro et al 2003); mode of birth (Adlerbert, Wold 2009). Causes of intestinal permeability include dysbiosis, inflammation (Hietbrink et al 2009), food additives (Csaki 2011), coffee (Cibickova et al. 2004), alcohol (Wang et al, 2014), parasites and bacteria (Lievin-Le Moal. 2013), endotoxins (O'Dwyer ST, 1988), dietary fat (Danielsen 2013), processed food (Rapin, Wiernsperger 2010), malnutrition (Rodriguez et al, 1996), prescription hormones (e.g.' Birth control pills) (Looijervan Langene et al 2011), mold (Sheveleva et al. 2004), microtoxin (Grenier, Applegate, 2013), C difficile (Moore et al 1990), dental infections (Yeoh et al 2013), airborne particulate matter (Salim et al. 2013), NSAIDS

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such as aspirin and ibuprofen (Sigthorsson et a., 1998), antacids, magnesium deficiency (Weglicki et al. 2013), high levels of exercise (Lamprecht et al. 2012), grains and refined carbohydrates.

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(End of Main Presentation)

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