

**25<sup>th</sup> Annual International Symposium  
on  
Man and His Environment in Health and Disease**

**Special Focus  
The Autonomic Nervous System including Nutrition, Function and  
Dysfunction, Electromagnetics and Prevention**

**Sponsored by  
American Environmental Health Foundation  
and  
University of North Texas Health Science Center**

**Physician Accreditation/Credit:**

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education and the American Environmental Health Foundation. The University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education is accredited by the ACCME to provide continuing medical education for physicians.

The University of North Texas Health Science Center at Fort Worth is accredited by the American Osteopathic Association to award continuing medical education to physicians.

**Credit**

The University of North Texas Health Science Center at Fort Worth designates this educational activity for a maximum of 22.75 AMA/PRA Category 1 Credits™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

The University of North Texas Health Science Center anticipates this program for 22.75 hours in Category 2A CME credit hours, pending approval from the American Osteopathic Association.

**Nursing Accreditation/Credit:**

The University of North Texas Health Science Center at Fort Worth is an approved provider of continuing nursing education by the Texas Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.

This activity meets Type I criteria for mandatory continuing education requirements toward relicensure as established by the Board of Nurse Examiners for the State of Texas.

This activity is approved for a maximum of 22.75 Contact Hours. To receive a certificate of successful completion, participants must attend the activity and complete and return the attendance record/credit request form and the evaluation form at the end of the activity.

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## **FINANCIAL CONSIDERATION**

AEHF is a nonprofit organization that was founded in 1975 to provide education and research into Environmental Medicine. This year's Symposium is our 25<sup>th</sup> Annual International Symposium and is our major vehicle for educating the medical professional.

Funding for the symposium is provided by registration fees from physicians and exhibitors. Proceeds from the AEHF store cover the shortfall between registration fees and expenses for the conference. AEHF does not receive grants or any outside financial support for our education. Donations are accepted and used toward research into environmental medicine.

# INTRODUCTION

## SYMPOSIUM PURPOSE

Since 1981, the International Symposium has been recognized as one of the most advanced medical forums in the world addressing the research and treatment of environmental effects on health and disease. The 2007 conference will focus on “The Autonomic Nervous System including Nutrition, Function and Dysfunction, Electromagnetics and Prevention.” This Conference presents the most current information available while providing guidelines to identify, diagnose, treat and to prevent environmentally triggered responses in the body.

## GOALS OF THE MEETING

- ! To provide new insights into the mechanisms and the environmental causes behind many problems seen by the physician.
- ! To present new diagnostic and treatment modalities to help improve the quality of care for your complex patients.
- ! To provide concepts, tools that will enhance the physicians practice.

## OBJECTIVES OF THE MEETING

- ! Improve the outcome of treating patients with a thorough understanding of the Autonomic Nervous System.
- ! Use new concepts and treatments related to the Autonomic Nervous System to help better diagnose and manage patients.
- ! Apply the concepts of this conference to your practice by using nutrition and environmental manipulation for the treatment.
- ! Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness in relation to the Autonomic Nervous System

## INTENDED AUDIENCE

M.D.=s, D.O.=s, D.D.S.’s medical students, nurses, nutritionists and other health professionals interested in the concepts and practice of Environmental Medicine, Occupational Medicine and Toxicology.

## EDUCATIONAL FORMATS

- ✂ Plenary
- ✂ Panels Discussions
- ✂ Case Studies
- ✂ Question & Answer Sessions.

## CONFERENCE FORMAT

The AEHF Committee has selected some of the leading experts in the fields of chronic disease, nutrition and chemical sensitivity.

Each speaker=s presentation will last approximately 20 minutes and will be followed by a 10 minute question and answer session. All speakers are encouraged to use any and all appropriate audio/visual aids. (A brief outline of the speech is included in this booklet.)



## **GIVEN IN COOPERATION**

**William J. Rea, M.D., F.A.C.S.**

Symposium Chairman,  
American Environmental Health Foundation,  
Environmental Health Center - Dallas,  
Dallas, Texas

**Bertie B. Griffiths, Ph.D.,**

Environmental Health Center - Dallas  
Dallas, Texas

**William J. Meggs, M.D., Ph.D.**

Brody School of Medicine, East Carolina University  
Department of Emergency Medicine  
Greenville, NC

**Doug Seba, Ph.D.**

Independent Marine Scientist  
Alexandria, VA

# 25<sup>th</sup> Annual International Symposium on Man and His Environment in Health and Disease

## Schedule of Proceedings, Table of Contents

### Thursday, June 7, 2007

#### Schedule

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Meggs, M.D., Ph.D., William J., "Neurotoxicity of Autonomic Nervous System I: Etiology"

Monro, M.D., Jean, "Neurophysiological Assessments of Autonomic Function"

Sandstrom, Ph.D., Monica, "Studies on ANS in Electrical Hypersensitive People"

Hillman, Ph.D., Donald, "EMF in Homes, Workplaces, and Schools, Part 1"

Suleman, M.D., Amer, "Differential Diagnosis of Sinus Tachycardia"

Kilburn, M.D., Kaye H., "Heart Rate Variation and Orthostasis in Adults Exposed to Chemicals"

Griffin, Ph.D., Dale, "Desert Dust and Respiratory Disease"

Nagy, M.D., Lisa, "The Recognition of Dysautonomia and It's Relationship to Oral Galvanism"

Smith, Ph.D., Cyril W., "Electromagnetics and the ANS"

Sinatra, M.D., Stephen T. "Metabolic and Mitochondrial Defense, The Missing Link in Heart Disease"

#### CASE STUDY:

Lieberman, M.D., Allan D., "Epidemic of Aflatoxin Poisoning in Contaminated Pet Food"

### Friday, June 8, 2007

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Rapp, M.D., Doris, "Political Aspects of the Good, Bad, and Ugly in Medicine"

Sinatra, M.D., Stephen T., "Plaque Reversal, the Solution"

Stark, M.D., Martha, "The Wisdom of the Matrix: Regulation, Balance, and Harmony"

Lee, Ph.D., Tang G., "Attenuating Electromagnetic Radiation in Buildings"

Ott, Ph.D., Riki, "Toxicological Paradigm Shifts after the Exxon Valdez Oil Spill"

Pangborn, Ph.D., Jon, "Perpetuation of Inflammation by Epigenetic Influences"

Cheney, M.D., Ph.D., Paul R., "Cardiac Diastolic Dysfunction in Chronic Fatigue Syndrome"

Abou-Donia, Ph.D., Mohamed B., "Organophosphorus Compound-Induced Neurotoxicity"

Weisler, M.D., Richard, "Elevated Rates of Suicides and Cancers in a Downwind Neighborhood: Why?"

Dalela, M.D., Divakar, "Benign Prostatic Hyperplasia - Can Dietary Modulations Prevent it?"

Overberg, Ph.D., C.C.N., R.D., Ron, "Organic Acid Analysis and Chemical Sensitivity"

McCarter, M.D., Stephanie, "Building Considerations for a Low EMF Home"

#### CASE STUDY:

Ross, M.D., Gerald H., "Toxic Buildings From One Simple Common Procedure"

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Meggs, M.D., Ph.D., William J., "Neurotoxicity of Autonomic Nervous System II: Diagnosis & Treatment"

Rea, M.D., William J. "Treatment of Autonomic Dysfunction"

Cheney, M.D., Ph.D., Paul R., "High Patent Foramen Ovale (PFO) Frequency in Chronic Fatigue Syndrome"

Sandstrom, Ph.D., Monica, "Provocation with Mobil Phone Signals"

Griffin, Ph.D., Dale, "Desert Dust Microbiology and Human Health"

Simon, M.D., Theodore R., "Functional Imaging in 2007"

Suleman, M.D., Amer, "Postural Orthostatic Tachycardia Syndrome"

Dalela, M.D., Divakar, "Diet & Cancer Prostate: Interrelationships & Implications"

Ott, Ph.D., Riki, "Sociological Paradigm Shifts after the Exxon Valdez Oil Spill"

Runow, M.D., Klaus-Dietrich, "Poisoned Children - Toxic Metal Intoxication in Kosovo"

Smith, Ph.D., Cyril W., "The ANS - a Unified Approach"

Lee, Ph.D., Tang G., "Beyond 60 Hz EMF to Radio-Frequencies"

Hillman, Ph.D., Donald, "EMF in Homes, Workplaces, and Schools, Part 2"

CASE STUDY:

Dean, D.O., Amy, "Vasculitis Responsive to Environmental & Osteopathic Medical Intervention"

Reiter, Ph.D., Russel J., "The Use of Melatonin to Protect Against Toxic Agents"

Runow, M.D., Klaus-Dietrich, "Detoxification of Heavy Metal Intoxication in Children"

Kilburn, M.D., Kaye H., "Mold/Mycotoxins Impair Neurobehavioral Function"

Pangborn, Ph.D., Jon "Biochemistry - Gone - Wrong in Autism: Some Remedies"

Abou-Donia, Ph.D., Mohamed B., "Autoantibodies against Nervous System Proteins as Biomarkers for Brain Injury"

Nagy, M.D., Lisa "Why Horse Women are Crazy - Mycotoxicosis"

Rapp, M.D., Doris, "The Fetus to Adult, Good, Bad, and Ugly in Medicine"

Suarez, M.D., Jesus, "Young Man Dizziness Treated with Immunotherapy"

# 25<sup>th</sup> ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT Schedule

Thursday, June 7, 2007

**7:00 a.m. REGISTRATION**

**8:20 WELCOME: William J. Rea, M.D.**

**8:25 MODERATOR: Doug Seba, Ph.D.**

8:30 Doug Seba, Ph.D., "Update 2007: The ANS in a Toxic World"  
8:50 Q & A

9:00 William J. Rea, M.D., "Syndrome of Autonomic Dysfunction"  
9:20 Q&A

9:30 William J. Meggs, M.D., Ph.D., "Neurotoxicity of Autonomic Nervous System I: Etiology"  
9:50 Q&A

**10:00 BREAK**

10:30 Jean Monro, M.D., "Neurophysiological Assessments of Autonomic Function"  
10:50 Q & A

11:00 Monica Sandstrom, Ph.D., "Studies on ANS in Electrical Hypersensitive People"  
11:20 Q & A

11:30 Donald Hillman, Ph.D.: "EMF in Homes, Workplaces, and Schools, Part 1"  
11:50 Q & A

**12:00 LUNCHEON IN SPURS RESTRAUNT**

1:00 **MODERATOR: William J. Meggs, M.D., Ph.D.**

1:00 Amer Suleman, M.D., "Differential Diagnosis of Sinus Tachycardia"  
1:20 Q & A

1:30 Kaye H. Kilburn, M.D., "Heart Rate Variation and Orthostasis in Adults Exposed to Chemicals"  
1:50 Q & A

2:00 Dale Griffin, Ph.D., "Desert Dust and Respiratory Disease"  
2:20 Q & A

2:30 Lisa Nagy, M.D., "The Recognition of Dysautonomia and It's Relationship to Oral Galvanism"  
2:50 Q & A

**3:00 BREAK**

3:30 Cyril W. Smith, Ph.D., "Electromagnetics and the ANS"  
3:50 Q & A

4:00 Stephen T. Sinatra, M.D., "Metabolic and Mitochondrial Defense, The Missing Link in Heart Disease"  
4:20 Q & A

4:30 CASE STUDY: Allan D. Lieberman, M.D., "Epidemic of Aflatoxin Poisoning in Contaminated Pet Food"  
Panel: Doctors Seba, Rea, Meggs, Suleman, Sandstrom, Kilburn, Griffin, Nagy, Smith, Sinatra

**6:00 AJOURN**

**THURSDAY, JUNE 7, 2007**

**ABSTRACTS**

**AND**

**HANDOUTS**

## Objectives & Notes

**Doug Seba, Ph.D.**

Date of talk: Thursday, June 7, 2007, 8:30am

P.O. Box 1417, #323  
Alexandria, VA 22313

Phone: 703/949-1055

### Training:

Current Job Description:	Independent Marine Scientist
Medical School/ University Attended	University of Miami, Coral Gables, Florida
Other Information: (including titles of books or articles you have recently written):	Over 45 years experience with chemicals and the environment.

### SPEECH TITLE: “Update 2007: The ANS in a Toxic World”

At the end of this Presentation, the participant should be able to:

1. Understand that for 25 years the essence of this conference has been to make the connection between environmental stressors and adverse health effects.
2. Realize that environmental phenomenon, such as xenobiotics combined with fate and transport mechanisms, can have major impact on the ANS.
3. Comprehend that adverse health effects on the ANS of patients can occur at great time and distance from their environmental origin.

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## Abstract

It is entirely appropriate that the 25th anniversary of this annual Conference focuses on the dysfunction of the autonomic nervous system for that process is at the core of the definition of the typical environmental illness patient. This Conference has for a quarter-century put the spotlight on the fact that, for most patients, their environmental illness, whether from chemical, physical, or biological sources, or a combination of them, ANS symptoms are a keystone in both the diagnosis and the treatment of the patient.

An enduring theme throughout the history of this Conference is the correct acknowledgement of this relationship to environmental pollutants because it is a root cause of environmental illness and, unfortunately, pollution endures.

This is a general review to set the tone for the Conference. Highly selected examples will be drawn from a mix of media, website, and scientific publications relevant to the current timeline. Some examples of ANS processes will be personally applied by the reviewer to ongoing research in wildlife anomalies in the Bitterroot mountains of Montana and the incursion of African dust storms into the Western Hemisphere.

Equally important are changes in the political/legal landscape of the definitions of risk assessment and regulatory processes in regard to the exposures that particularly affect environmentally sensitive patients.

The fate and transport of endocrine disruptors from distant sources and mechanisms of reconcentration are continuing research interests of the presenter. Hormones are really the messengers of life and their disruption portends drastic effects on our ecosphere. Particular attention will be given to the fact that some of the environmental stressors can come vast distances from their source, be disbursed temporally and geographically, and thus maintain patients in a constant state of ANS dysfunction.

### Selected Digital References

DOI available @ <http://dx.doi.org/>  
[www.plosgenetics.org](http://www.plosgenetics.org) DOI:10.1371/journal.pgen.0030005  
[www.plosgenetics.org](http://www.plosgenetics.org) DOI:10.1371/journal.pgen.0030006.g002  
Reproductive Toxic. DOI:10.1016/j.reprotox.2006.10.002  
<http://www.msnbc.msn.com/id/16908487/>  
Tox. App. Pharmacol. DOI:10.1016/j.taap.2003.11.008  
<http://news.mc.duke.edu/news/article.php?id=7496>  
<http://www.organicconsumers.org/school/leukaemia012006.cfm>  
Environ. Health. Pst. DOI:10.1289/ehp.5830  
<http://www.rcep.org.uk/webmap.htm>  
[www.plosbiology.org](http://www.plosbiology.org) DOI:10.1371/journal.pbio.0050035  
<http://medicine.iupui.edu/flockhart/table.htm>  
<http://www.ombwatch.org/article/blogs/279/19>  
<http://www.bushsecrecy.org/>  
<http://www.uschamber.com/issues/index/regulatory/costbenefitanalysis.htm>  
<http://www.gao.gov/newitems/d7464t.pdf>  
<http://www.nap.edu/catalog/11811.html>  
<http://www.epa.gov/opptintr/library/pubs/archive/oscp.htm>  
<http://pubs.acs.org/cen.news/84/i44/8444notw2.html>  
<http://pubs.acs.org/cen/editor/84/8444edit.html>  
Envir. Sci. Technol. DOI:10.1021/es0622709  
Envir. Sci. Technol. DOI:10.1021/es06083n

## Objectives & Notes

**William J. Rea, M.D.**

Date of talk: Thursday, June 7, 2007, 9:00am

Environmental Health Center - Dallas  
8345 Walnut Hill Lane, Ste. 220  
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### Training:

Current Job Description:	Founder and President of the Environmental Health Center - Dallas
Medical School/ University Attended	Ohio State University College of Medicine
Internship:	Parkland Memorial Hospital
Residency:	University of Texas Southwestern Medical School
Board Certifications:	American Board of Surgery, American Board of Thoracic Surgery, American Board of Environmental Medicine
Other Information: (including titles of books or articles you have recently written):	Optimum Environments for Optimum Health and Creativity (book); Averse Health Effects of Indoor Molds.

### SPEECH TITLE: “**Syndrome of Autonomic Dysfunction**”

At the end of this Presentation, the participant should be able to:

1. Recognize that multiple autonomic syndrome exists.
2. Recognize the clinical autonomic syndrome.
3. Apply the knowledge acquired in the clinical practice.

*The American Environmental Health Foundation and the University of North Texas Health Science Center is not responsible for the contents of this presentation. AEHF has not altered or modified the contents of the information provided by this speaker.*



## Objectives & Notes

**William J. Meggs, M.D., Ph.D.**

Date of talk: Thursday, June 7, 2007, 9:30am

Brody School of Medicine, East Carolina University  
600 Moye Blvd., Room 3ED311.  
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Greenville, NC 27834-4354

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### Training:

Current Job Description:	Physician and Research Scientist
Current Faculty Appointments:	Professor, Brody School of Medicine
Medical School/ University Attended	University of Miami, Miami, Florida
Internship:	University of Rochester
Residency:	University of Rochester
Board Certifications:	Medical Toxicology, Allergy and Immunology, Internal Medicine, Emergency Medicine
Other Information: (including titles of books or articles you have recently written):	Book: The Inflammation Cure, McGraw-Hill (2003)/ Fallacies in Refutation of Causality, published in Clinical Toxicology, 2005. Sustained oxygenation without ventilation, published in Amer. Journal Emergency Medicine in 2005. Pressure Immobilization Bandages delay of toxicity of coral snake bites, published in Annuals of Emergency Medicine, 2005.

### SPEECH TITLE: "Neurotoxicity of Autonomic Nervous System I: Etiology"

At the end of this Presentation, the participant should be able to:

1. The anatomy and physiology of autonomic nervous system as it relates to toxic exposures will be reviewed.
2. The mechanisms by which toxins affect the autonomic nervous system
3. A discussion will be given of the differences between acute and chronic autonomic toxicity
4. Specific agents that affect the autonomic nervous system will be discussed.

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## Neurotoxicity of the Autonomic Nervous System I: Etiology

### Goals and Objectives

- The anatomy and physiology of autonomic nervous system as it relates to toxic exposures will be reviewed.
- The mechanisms by which toxins affect the autonomic nervous system
- A discussion will be given of the differences between acute and chronic autonomic toxicity
- Specific agents that affect the autonomic nervous system will be discussed.

### Conclusions

A number of substances can adversely affect the autonomic nervous system, causing both acute and chronic toxicity. Acute poisonings affecting the autonomic nervous system most frequently result from disturbances of neurotransmission in the parasympathetic and sympathetic branches of the autonomic nervous system. Examples include cholinergic agents such as organophosphate insecticides, anticholinergic agents such as plants containing belladonna alkaloids, and sympathemimetic agents such as cocaine and amphetamines. Chronic autonomic dysfunction most commonly results from direct damage to nerve cells involved in autonomic regulation. Organophosphate insecticides, the nerve gas sarin, and heavy metals can produce permanent damage that results in autonomic dysfunction, though this rarely occurs in isolation from other neurological deficits.

### References

[http://microvet.arizona.edu/Courses/VSC401/autonomicNervous\\_files/image004.jpg](http://microvet.arizona.edu/Courses/VSC401/autonomicNervous_files/image004.jpg)

Geotz C. Textbook of Clinical Neurology. 3<sup>rd</sup> Edition. Saunders. 2007.

Goldfrank L et al, editors. Goldfrank's Toxicologic Emergencies, 8<sup>th</sup> Edition. McGraw-Hill, 2006.

Spencer CS, Schaumberg HH. Experimental and clinical neurotoxicology. 2<sup>nd</sup> edition. Oxford University Press. 2000.

## Objectives & Notes

### Jean Monro, M.D.

Breakspear Hospital  
Hertfordshire House  
Wood Lane, Paradise Estate  
Hemel Hempstead, Herts HP2 4FD  
England

Date of talk: Thursday, June 7, 2007, 10:30am

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Email: jmonro@breakspearmedical.com

### Training:

Current Job Description:	Medical Director of The Breakspear Hospital, England
Medical School/ University Attended	London Hospital Medical School, England
Residency:	London Hospital
Board Certifications:	MB, BS, MRCS, LRCP, FAAEM, DipIBEM, MACOEM
Other Information: (including titles of books or articles you have recently written):	Treatment of cancer with mushroom products. Arch Environ Health 2003;58:533-7 Coriolus. Available from www.jintmed.com

### SPEECH TITLE: “Neurophysiological Assessments of Autonomic Function”

At the end of this Presentation, the participant should be able to:

1. Autonomic nervous system controls every organ in the body; therefore clinical tests must reflect this.
2. There is a novel neurophysiological methodology that allows organ-specific examination of the autonomic system.
3. Quantitative assessment of the autonomic nervous system is available for monitoring progress of treatment of patients suffering from the effects of environmental toxicity.

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# Breakspear Hospital



Title: **NEUROPHYSICAL ASSESSMENTS OF AUTONOMIC FUNCTION**

Venue: **25<sup>TH</sup> ANNUAL INTERNATIONAL SYMPOSIUM ON MAN AND HIS ENVIRONMENT,  
DALLAS, 7<sup>TH</sup>-10<sup>TH</sup> JUNE 2007**

Author: **JEAN A MONRO  
MB, BS, MRCS, LRCP, FAAEM, DIBEM, MACOEM  
Medical Director, Breakspear Hospital, Wood Lane, Hemel Hempstead,  
Herts, HP2 4FD, UK**

In Association  
with: **DR PETER JULU  
MBChB, MSc, PhD  
Autonomic Neurophysiologist/Senior Research Fellow, Breakspear Hospital, Wood Lane,  
Hemel Hempstead, Herts, HP2 4FD, UK**

## ABSTRACT

### *Examination of Cardiovascular reflexes and cardiac parasympathetic function*

The resting cardiac parasympathetic activity, or cardiac vagal tone (CVT) is measured heartbeat-by-heartbeat in a quiet laboratory with subdued light where the room temperature is maintained at  $24 \pm 1$  °C using the NeuroScope™ system (MediFit Instruments Ltd, London, UK) as previously described by Julu (1992). We used the effect of baroreceptor stimulation on the sino-atrial node affecting the R-R intervals to measure the CVT, because it is mediated through the vagal tone and it is detectable in the ECG P-P intervals (Eckberg, 1976). The NeuroScope™ quantifies this baroreflex function using atropine-derived clinical units of a linear vagal scale (LVS). The LVS was first derived by Julu (1992) and is now validated for clinical use in humans (Delamont et al., 1998) and in animals (Little et al., 1999). Arterial blood pressure (BP) is recorded continuously and non-invasively using the Finapres (Ohmeda, Eagleswood, USA). The systolic (SBP), mean arterial (MAP) and diastolic (DBP) BP in the digital artery of one finger are calculated by the VaguSoft® software (MediFit Instruments Ltd, London, UK). The NeuroScope™ system quantifies the output of the arterial baroreceptors and this was validated in real-time by stretching the walls of empty carotid sinuses during carotid endarterectomy surgery (Sigaudou-Roussel et al., 2001). All data is recorded in real-time using the VaguSoft® software.

The carotid sinus is massaged to assess the cardiodepressor and vasodepressor functions of arterial baroreceptors. A 10 second cycle of deep breathing is used to examine the respiratory modulation of CVT at brainstem level to assess the central cardiorespiratory coupling. Graded doses of phenylephrine injections are used to determine the threshold and slopes of baroreceptor responses using the Oxford methods (Smyth et al., 1969). The cardiac response to orthostasis is quantified using our modified 30:15 ratio test (Julu and Hondo, 1992). The Finapres is used to identify dysfunctions in BP regulation during orthostasis as follows: Variation of MAP by more than 25 mmHg would suggest

orthostatic instability, while a sustained fall in DBP by 10 mmHg or more within three minutes of assuming an erect posture compared with the level in supine position is an indication of orthostatic hypotension (1996). The level of BP within 1-2 minutes of a change in posture are important for the assessment of the neural control of BP (Wieling and Shepherd, 1992; Wieling and van Lieshout, 1992).

Cardiac baroreflex responsiveness during isometric exercise (BRR) indicates the central gain of the baroreflex system in the brain. It is quantified from the R-R intervals and BP changes in response to the pressor effect of 3-minute isometric contraction of the muscles of the forearm. The patient applies 50% of his or her maximum sustainable grip force to our special machine and maintains a constant force for 3 minutes. We define BRR as the absolute value of the quantity  $|\Delta R-R/\Delta SBP|$ . Where:  $\Delta R-R$  is the change in R-R interval in the third minute of isometric exercise associated with a corresponding change in systolic BP ( $\Delta SBP$ ) compared with baseline levels (Julu et al., 1996b). The heart rate (HR) response to Valsalva's manoeuvre is assessed while the subject actively maintains an intrathoracic pressure of 40 mmHg for 15 seconds. The ratio of the longest R-R interval immediately following the cessation of positive intrathoracic pressure to the shortest R-R interval during the positive intrathoracic pressure is known as Valsalva's ratio. The average value of the Valsalva's ratio in three manoeuvres is the subject's response.

### ***Examination of Sympathetic functions***

Emotional Sudomotor Function (ESF) in the skin is assessed by measuring the Galvanic skin responses to emotional (or mental) sweating in the palms of the hands and soles of the feet. The Galvanic skin response is evoked by a single inspiratory gasp. Thermoregulatory Vasomotor Function (TVF) in the skin is assessed using a cold challenge, which is applied to one hand while recording the skin blood flow in the contralateral limbs. The cold challenge was achieved by first immersing the hand in warm water (40 °C) for two minutes to maximise vasodilatation before transferring it into cold water (10 °C) for another two minutes to evoke a vasoconstriction. The cooled hand was then re-immersed in warm water for a further two minutes. The skin blood flow in the dorsum of all four limbs is measured simultaneously using a four-channel laser Doppler flow meter (Moor Instruments, Axminster, UK). Normal responses are indicated by decreases of blood flows in both contralateral limbs during the cold challenge and a return to baseline levels in response to re-warming. Failure of TVF is indicated either by no effect of the cold challenge on the skin blood flow, or a paradoxical increase of blood flow in the contralateral limbs. Sympathetic cardioaccelerator function in the heart and sympathetic vasoconstrictor response in the skeletal muscles are assessed during isometric exercise by measuring the change in heart rate (HR) and DBP respectively, in the third minute of isometric exercise compared with the level just before the onset of the exercise. The change in the vascular resistance that determines DBP during isometric exercise is linearly related to the muscle sympathetic activity measured by microneurography (Halliwill et al., 1996). On the other hand, the increase in HR during isometric exercise measured continuously in our laboratory is closely related to the change in DBP, thereby proving the sympathetic origin of this cardioacceleration (Ewing et al., 1974; Julu et al., 1996a). By the third minute of isometric exercise, the withdrawal of both vagal and baroreflex regulation of the cardiovascular system would be complete (Julu et al., 1996a), leaving only the sympathetic effects on the system. This manoeuvre allows selective assessment of the sympathetic function in the heart and skeletal muscles. We use the gradient of BP rise in the ejection period of the cardiac cycle as an index of beat-to-beat left ventricular inotropic function. The sympathetic adrenergic function in the splanchnic vascular bed is assessed during Valsalva's manoeuvre as follows. If an active intrathoracic pressure of 40 mmHg is maintained for 15 s, the SBP will change in five phases represented by Roman numerals as I, IIe, IIIi, III and IV (Low, 1992). Phase I is the initial rise in BP due to pressure on the great vessels in the thorax, Phase IIe is a sharp drop in SBP and pulse pressure due to the sustained reduction in venous return caused by the positive intrathoracic pressure. Phase IIIi is the recovery of both the SBP and the pulse pressure against the positive intrathoracic pressure and is due to sympathetic mobilisation of a reserved volume of blood from the splanchnic vascular bed stimulated by the crisis of blood volume in the heart. This is a physiological process called "auto-transfusion" (Keele et al., 1982). The inferior vena cava carries more than two-third of the venous return (Keele et al., 1982), but venous return from the lower limbs depends largely on the muscle pump effect. Our subjects carry out the Valsalva's manoeuvre in a sitting position without moving the lower limbs while leaning forwards to exclude any contribution from vasoconstriction or the muscle pump effect in the lower limbs to the recovery of pulse pressure and the SBP, allowing us to assess the splanchnic sympathetic adrenergic function selectively using SBP Phase IIIi in Valsalva's manoeuvre. Phenylephrine injection is used to assess denervation hypersensitivity that would indicate if there were significant damage to the sympathetic postganglionic nerve fibres.

## **Conclusion**

We have a novel neurophysiological methodology for selective, non-invasive and quantitative assessment of the functions of various autonomic target-organs. Our methods allow real-time monitoring of brainstem functions in a manner that has never been done before and for the first time we have the potential of correlating brainstem autonomic functions with electroencephalograms. The methodology is ideal for investigating the effects of environmental toxins on the autonomic nervous system.

## Reference List

1. (1996) Consensus statement on the definition of orthostatic hypotension, pure autonomic failure, and multiple system atrophy. The Consensus Committee of the American Autonomic Society and the American Academy of Neurology. *Neurology* 46: 1470.
2. Delamont RS, Julu PO, Jamal GA (1998) Sleep deprivation and its effect on an index of cardiac parasympathetic activity in early nonREM sleep in normal and epileptic subjects. *Sleep* 21: 493-498.
3. Eckberg DL (1976) Temporal response patterns of the human sinus node to brief carotid baroreceptor stimuli. *J Physiol (London)* 258: 769-782.
4. Ewing DJ, Irving JB, Kerr F, Wildsmith JA, Clarke BF (1974) Cardiovascular responses to sustained handgrip in normal subjects and in patients with diabetes mellitus: a test of autonomic function. *Clin Sci Mol Med* 46: 295-306.
5. Halliwill JR, Taylor JA, Eckberg DL (1996) Impaired sympathetic vascular regulation in humans after acute dynamic exercise. *Journal Of Physiology-London* 495: 279-288.
6. Julu POO (1992) A linear scale for measuring vagal tone in man. *J Auton Pharmacol* 12: 109-115.
7. Julu POO, Hansen S, Barnes A, Jamal GA (1996a) Continuous measurement of the cardiac component of arterial baroreflex (ccbr) in real-time during isometric-exercise in human volunteers. *Journal Of Physiology-London* 497P: P 7-P 8.
8. Julu POO, Hansen S, Jamal GA (1996b) Is baroreflex responsiveness (brr) a function of height during isometric muscle-contraction in human volunteers. *Journal Of Physiology-London* 491P: P 85.
9. Julu POO, Hondo RS (1992) Effects of atropine on autonomic indices based on electrocardiographic R-R intervals in healthy volunteers. *J Neurol Neurosurg Psychiatry* 55: 31-35.
10. Keele CA, Neil E, Joels N (1982) Cardiac performance during exercise. In: Samson Wright's Applied Physiology (Keele CA, Neil E, Joels N, eds), pp 107-111. Oxford New York Toronto: Oxford University Press.
11. Little CJ, Julu PO, Hansen S, Reid SW (1999) Real-time measurement of cardiac vagal tone in conscious dogs. *American Journal of Physiology* 276: H758-H765.
12. Low PA (1992) Evaluation of autonomic function. *Curr Opin Neurol Neurosurg* 5: 461-463.
13. Sigaud-Roussel D, Julu POO, Gaunt ME (2001) Brainstem cardiovascular regulation during stretch-stimulation of the luminal surface of the carotid sinus in anaesthetised human subjects. *J Physiol Lond* 536.P: 147P-148P.
14. Smyth HS, Sleight P, Pickering GW (1969) Reflex regulation of arterial pressure during sleep in man. A quantitative method of assessing baroreflex sensitivity. *Circulation Research* 24: 109-121.
15. Wieling W, Shepherd JT (1992) Initial and delayed circulatory responses to orthostatic stress in normal humans and in subjects with orthostatic intolerance. *Int Angiol* 11: 69-82.
16. Wieling W, van Lieshout JJ (1992) Maintenance of postural normotension in humans. In: *Clinical Autonomic Disorders* (Low PA, ed), pp 69-77. Boston Toronto London: Little, Brown and Company.

## Objectives & Notes

### Monica Sandström, Ph.D.

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M. Sandström, E. Lyskov, R. Hörnsten, K. Hansson Mild, U. Wiklund, P. Rask, V. Klucharev, B. Stenberg and P. Bjerle. Holter ECG monitoring in patients with perceived electrical hypersensitivity. *Internat J Psychophysiol.* 49; 227-235, 2003.

J. Wilen, A. Hohansson, N.a Kalezic, E. Lyskov and M. Sandström. Psychophysiological tests and provocation of subjects with mobile phone related symptoms. Published Online: 22 Nov 2005. DOI: 10.1002/bem.20195. *Bioelectromagnetics*27:pp 204-214, 2006.

**SPEECH TITLE: “Studies on ANS in Electrical Hypersensitive People”** (Monica Sandström, Ph.D., Amanda Johansson, Ph.D. student, National Institute for Working Life, Umeå, Sweden)

At the end of this Presentation, the participant should be able to:

1. Understand why we studied ANS in EHP
2. Understand the method used
3. Understand the result of the study.

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# Studies on ANS in Electrical Hypersensitive People

Monica Sandström, PhD, Amanda Johansson, PhD student

National Institute for Working Life and the Department of Occupational and Environmental Medicine, Umeå University, Umeå, Sweden

During the last 10 years we have performed a number of provocation studies on people with perceived electrical hypersensitivity (EHS) in a traditional approach. These studies rendered no conclusive answer to the question of a potential, causal relation between exposure to electromagnetic fields and the origin of symptoms. Therefore we extended our studies to encompass also more general reactivity and included physiological and neurophysiological examinations. How do people with EHS react to other physical factors in their environment and what are their physiological and neurological baseline data? In a number of experiments with this approach we found differences between EHS patients and matched controls without EHS symptoms.

In the first series of experimental setups we found that people with EHS symptoms were more sensitive to flickering light measured by increased evoked potential. The sympathetic skin responses to audio clicks showed increased amplitude as well as decreased latency and relative asymmetry compared to controls. A general physiological profile showed an imbalance in the autonomic regulation with a trend towards hypersympathothone and increased arousal. During cognitive tests the heart rate increased and furthermore we observed increased spontaneous electrodermal activity in the EHS group compared to controls. [1, 2]

In order to better characterize the balance of the autonomic regulation we used long-term monitoring of electrocardiogram (ECG) in fourteen EHS patient and a matched control group. Heart rate (HR) and heart rate variability (HRV) as well as magnetic fields were monitored for 24 hours.[3]

No difference was seen between groups in the hour-by-hour analysis of HR. However in the late evening a slightly lower HR was observed among EHS patients. The HRV analysis revealed that the high-frequency (HF) component of HRV, reflecting the activity in the parasympathetic branch of the autonomic nervous system, did not display the expected increase during night time in the EHS group. When separating sleeping and awake time also smaller differences between the two conditions in the EHS patients became apparent, both for the low frequency (LF) and HF components of the HRV spectrum. EHS patients displayed a disturbed pattern of circadian rhythms of HRV and a relatively 'flat' representation of hourly-recorded spectral power of the HF component of HRV. It is of interest in this context to mention that similar trends towards night time parasympathetic withdrawal have been found in subjects suffering from Gulf War syndrome [4]and fibromyalgia [5].

Analysis of the recorded 24 hour magnetic field exposure, revealed no differences between groups and no correlations were found between any of the recorded physiological parameters and magnetic field data.

However, the results of our studies indicated modest but distinctive signs of an autonomous imbalance with a trend toward sympathetic hyperactivity and a deviating circadian rhythm in people with EHS. An elevated reactivity to sensory stimuli was observed, in form of increased evoked potentials in response to flickering light and facilitated skin sympathetic responses in response to auditory stimulation. This sensory amplification and lability of the autonomous nervous system could be considered as signs of a physiological disposition characterized by stress vulnerability, an increased sensitivity to and a lower tolerance to physical environmental factors. These studies need to be followed up.

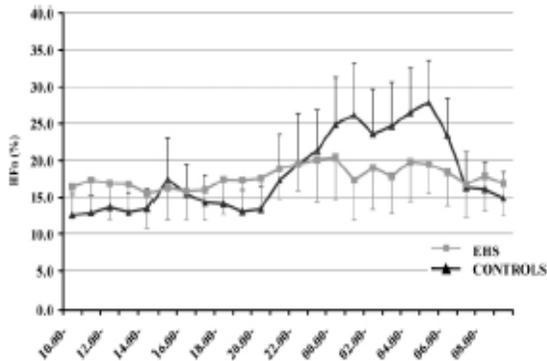


Fig. 2. Twenty-four-hour registration of normalized HF (mean values for each hour) from EHS and controls. In bars the 95% confidence intervals are shown ( $F=3.40$ ,  $P=0.001$ , for group differences).

The figures are from M. Sandström et al., *Holter ECG monitoring in patients with perceived electrical hypersensitivity*. *Internat J Psychophysiol.* 49; 227-235, 2003.

#### References

1. Lyskov, E., M. Sandström, et al. *Neurophysiological study of patients with perceived 'electrical hypersensitivity'*. *Int J Psychophysiol*, 2001. **42**(3): p. 233-41.
2. Lyskov, E., M. Sandström, et al. *Provocation study of persons with perceived electrical hypersensitivity and controls using magnetic field exposure and recording of electrophysiological characteristics*. *Bioelectromagnetics*, 2001. **22**(7): p. 457-62.
3. Sandström, M., E. Lyskov, et al., *Holter ECG monitoring in patients with perceived electrical hypersensitivity*. *Int J Psychophysiol*, 2003. **49**(3): p. 227-35.
4. Haley, R.,W. Vongpatanasin, et al., *Blunted circadian variation in autonomic regulation of sinus node function in veterans with Gulf War syndrome*. *American Journal of Medicine*, 2004. **117**(7): p. 469-78.
5. Martinez-Lavin, M., A. Hermosillo, et al., *Cardiac studies of autonomic nervous balance in patients with fibromyalgia: a heart rate variability analysis*. *Arthritis Rheum.*, 1998. **41**(11): p. 1966-71.



## Objectives & Notes

**Donald Hillman, Ph.D.**

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### SPEECH TITLE: “EMF in Homes, Workplaces, and Schools, Part 1”

At the end of this Presentation, the participant should be able to:

1. Recognize that chronic exposure to EMF can produce Electropathic Stress Syndrome in animals.
2. Identify and locate sources of EMF exposure on farms and animal environments.
3. Consider circadian blood ANS profiles and micronuclei analyses in diagnosis and treatment of stress syndrome.

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## Objectives & Notes

**Amer Suleman, M.D.**

Date of talk: Thursday, June 7, 2007, 1:00pm

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### SPEECH TITLE: “**Differential Diagnosis of Sinus Tachycardia**”

At the end of this Presentation, the participant should be able to:

1. Understand the mechanisms of sinus tachycardia
2. Identify common diseases that can cause sinus tachycardia
3. Able to understand and treat basic sinus rhythm problems

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## Objectives & Notes

**Kaye H. Kilburn, M.D.**

Date of talk: Thursday, June 7, 2007, 1:30pm

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Residency:	University of Utah: Medicine and pathology, Duke: cardiopulmonary, London: cardiology
Board Certifications:	Am Board Internal Medicine, Am Board Preventive Medicine, occupational Health
Other Information: (including titles of books or articles you have recently written):	“Chemical Brain Injury,” NY: John Wiley 1998; “Mold and Mycotoxins,” editor Washington DC: Heldref 2004; “Endangered Brains” Princeton Scientific Press 2004

### SPEECH TITLE: “Heart Rate Variation and Orthostasis in Adults Exposed to Chemicals”

At the end of this Presentation, the participant should be able to:

1. Orthostatic adjustment of blood pressure and heart rate vary greatly in chemical intolerant patients
2. Exposures to hydrogen sulfide and chlorine disorder blood pressure compensation more than mold exposure
3. Heart rate variation with breathing, sinus arrhythmia is more frequent in hydrogen sulfide exposed people and is correlated with the numbers of their neurological abnormalities

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## **Orthostasis, Heart Rate Variability and Autonomic Function**

**Kaye H. Kilburn, M.D.**  
**and**  
**Bradford E. Hanscom**

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We hypothesized that autonomic dysfunction would reduce heart rate variability measured as the standard deviation of cardiac RR interval. Low level autonomic dysfunction should also be detected by blood pressure and heart rate changes on standing from lying (orthostasis).

1. If there was autonomic dysfunction heart rate variability should decrease. This expectation based on minimal heart rate variability in patients with diabetes mellitus and in groups of workers exposed to organic solvents.
2. Deficient orthostatic compensation would be reflected by decreasing systolic and diastolic blood pressure and increased heart rate with light headedness or fainting.

### Background

The RR interval variation was measured during deep slow breathing rate 6 per minute in 39 community volunteers who were unexposed to chemicals. It was expressed as the standard deviation of RR, RR sd. The mean of the RRsd was 93.5 + 58.4 standard deviation, showing large variation, 16 to 257. Plus or minus two standard deviations or 116.8 exceeded the mean and one standard deviation gave a cutoff value of 35.1 it could not define abnormality. One and a half standard deviations was 87.6 (93.5-87.6=5.9) which was below any value observed so it raised doubts about the tests potential to discriminate abnormality. We examined all patients for orthostatic adjustment of blood pressure an independent observation of autonomic function.

### Methods

Sixty nine patients being tested for possible brain damage from chemicals divided into 4 groups. Group 1, 24 exposed to mold, group 2, 14 exposed to chemicals, group 3, 19 exposed to hydrogen sulfide, group 4, and 11 exposed to H<sub>2</sub>S and septic tank contents. They were measured for RRsd and for orthostatic changes after standing after lying using a pulse sensing sphygmomanometer. During this visit measurements were made of balance, reaction time, blink reflex, color discrimination, visual fields, hearing, grip strength, vibration threshold, odor perception and recognition and cranial nerve functions. Also recall, memory, problem solved perceptual motor speed and long term memory were assayed using standard physiological tests and correlated with autonomic function. I looked for relationships with these measurements using regression analysis in the groups and compared group for differences with analysis of variance (ANOVA)

### Results

The hydrogen sulfide exposed group n=19 showed heart rate variation as RRsd was modeled by a constant 5.07 minus 3.62 mean RR (heart rate) plus 2.78 POMS - 3.70 depression -2.92 systolic blood pressure difference from lying to standing.

**TABLE 1**

**Regression Equations for Hydrogen Sulfide Exposed People**

1. RRsd= 360.85 -3.60 RR + 1.80 POMS -8.23 Depression -2.86 systolic BP difference (variance explained r<sup>2</sup> 64%)
2. RRsd= 412.38 -3.93 HR -2.50 Depression -2.72 Systolic BP difference r<sup>2</sup> 48%
3. When the septic tank people were included n=30;  
RRsd = 372.21 -3.97 RR +2.00 POMS -3.53 depression -2.48 systolic BP difference  
r<sup>2</sup> 42%

Because depression is a component of POMs score we deleted it and POMs score independently from equation 1 and found that the variance explained with depression in and POMs out was 48%, contrasted to explaining 34% with POMS in and depression out. With both out the r<sup>2</sup> dropped to 32%. Thus affective status measured by POMs and depression appeared to have a major effect on heart rate variability in this group.

Usually adding observations increases statistical significance and confirms findings but did not do so in this instance. RRsd for mold exposed (n=24) had an coefficient for age of 2.04 but it only approached significance (p>.056). RRsd for chemical exposed (n=14) had no significant coefficient.

These findings raised doubts about the initial correlation. Slow heart (rates) provides more opportunities to find respiratory variability. A decrease in systolic blood pressure with standing is frequent in people with decreased physiological conditioning and usually correlates with increased heart rate (heart rate difference). No coefficient was found. None of the chemical exposed group had RRsd below 50, even though for RRsd 2 coefficients had positive direction and for all others were negative suggesting they were off setting.

**TABLE 2**

**Heart Rate Variability (Standard Deviation) of RR for 4 Exposed Groups Compared to Wick and Paul Unexposed Groups by Analysis of Variance**

**Mean RRsd and Standard Deviations**

	Wick N=39	Paul N=22
	93.5 ± 58.4	84.9 ± 57.8
14 Chem	146.9 ± 66.4 p>.007	p>.006
22 H <sub>2</sub> S	123.6 ± 71.9 p>.08	p>.056
11 Septic	126.1 ± 87.7 p>.15	p>.044
33 H <sub>2</sub> S + Septic	124.4 ± 76.1 p>.055	p>.056
22 Mold	112.5 ± 89.1 p>.3	p>.219

The people in chemically exposed groups had more heart rate variability- shown by higher mean RRsd than unexposed control people. This is in the opposite direction to a decrease that would be expected if they had autonomic dysfunction.

The final strategy (never give up) approach was to identify and characterize individuals in the groups with low heart rate variability and those with high variability. High variability was characteristic of the teen age members of the

groups especially those from one mold exposed family. But there were no other clues. Low variability, the other end of the distribution was equally bereft of clues

Actually, the control group had 10 of 50 subjects with RRsd below 45 and 10 above 150 and values were symmetrically distributed. Although this study was of small groups, convenience samples and categorized by major or dominant exposures these realities do not seem to invalidate conclusions.

### Conclusion

Reduced heart rate variability with deep breathing was no more frequent in 4 additional chemically exposed groups than in unexposed subjects. Community wide chlorine exposure was reported previously to reduce heart rate variability. Orthostatic measures were equally unhelpful. Autonomic dysfunction must be infrequent in people exposed to these chemicals or in the organophosphate subgroup. In summary these exposures do not affect cholinergic vagal function that controls sinus arrhythmia nor do they alter adrenergic response to adjust or compensate for standing (orthostasis). Two years ago chlorine exposure reduced heart rate variability but did not select individuals as abnormal. We conclude that autonomic dysfunction does not occur from exposure to these chemicals.

## Objectives & Notes

**Dale W. Griffin, Ph.D.**

Date of talk: Thursday, June 7, 2007, 2:00pm

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### SPEECH TITLE: “Desert Dust and Respiratory Disease”

At the end of this Presentation, the participant should be able to:

1. Understand the potential impacts of soil particles associated with dust storms on public health.
2. Understand the variety and concentrations of microorganisms associated with dust storm clouds.
3. Understand the ability and frequency that large dust storms move through our atmosphere on a global scale.

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## Desert Dust and Respiratory Disease

Dale W. Griffin, Ph.D., MSPH, Environmental and Public Health Microbiologist

U.S. Geological Survey, Florida Integrated Science Center, Tallahassee, Florida 32310

Dust storms move an estimated three billion tons of soil some distance in Earth's atmosphere each year. Large dust events are capable of global dispersion and can load downwind atmospheres with significant quantities of respirable particulate matter, for extended periods of time. In addition to the approximately 1 million to 1 billion bacterial cells (pathogens and non-pathogens) that are present in each gram of desert topsoil, other constituents such as viruses, fungi, metals, radioisotopes, organic toxins, and inorganics are capable of causing morbidity and mortality in downwind environments. This presentation will cover our current state of knowledge in this emerging research field to include, desert dust and its ability to trigger immune response, and studies in desert dust related to silicosis/pulmonary fibrosis.

## Objectives & Notes

**Lisa Nagy, M.D.**

Date of talk: Thursday, June 7, 2007, 2:30pm

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Board Certifications:	Emergency Medicine
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### SPEECH TITLE: **“The Recognition of Dysautonomia and its relationship to Oral Galvanism”**

At the end of this Presentation, the participant should be able to:

1. Identify common signs of dysautonomia or ‘POTS’ on history and physical.
2. How to examine patients for sources of galvanic current.
3. Prioritize treatment based on dental assessment.

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## The Recognition of Dysautonomia and its Relationship to Oral Galvanism

In this talk I will review the common symptoms experienced by the average person with mild dysautonomia as well as the more severe symptoms experienced by the more damaged environmental patient. Postural Orthostatic Tachycardia Syndrome is one form of dysautonomia characterized by a positive tilt table with a heart rate elevation of 25 beats per minute when tide to a board and not moving the skeletal muscles compared to lying. Interestingly, many of the patients who are members of the dysautonomia society complain of electrical sensitivity and their hair standing on and when walking below chandeliers and the like! Some of these patients will have, as I did, a mouth battery worsening or causing this dysautonomia. It is easy and essential to identify these treatable cases of Oral Galvanism. Others may have no such galvanic occurrence but may none the less be helped by energetic therapies which help to correct their electrical irregularities.

Obviously, the Autonomic Nervous System is a part of the peripheral nervous system and uses electricity to conduct impulses down the axon until the impulse tells the neurotransmitters to be released. 80% of the fibers of the ANS are sensory and may conduct the sensation of pain. The dental pulp has it's own, mostly sympathetic, ANS fibers which travel along arteries, veins and lymphatics of the tooth pulp. Any dysfunction in a tooth can cause arousal of the sympathetic fibers and result in headache as well as other symptoms. The occurrence of 'The Mouth Battery' is a well described phenomenon since before 1931 which can disrupt, in some susceptible patients – but not all, the normal functioning of the ANS. (Dietrich Klinghardt).

Again it is my personal experience that has convinced of the importance of two things: 1. the prime role of electrical interference or dysfunction in the body leading to the symptoms of chemical sensitivity as well as electrical sensitivity and 2. the role of electrical currents (namely in the mouth) in the development of symptoms of dysautonomia. Both of these anecdotally observed connections helps me to understand the potential role for diagnosis and treatment of patients with chemical sensitivity and dysautonomia. There are millions of patients with dysautonomia and at NDRF.org you can read about different types of dysautonomia.

In my case, I had severe exacerbation of electrical sensitivity when I became overloaded on my way to Dallas for treatment. In order to cope with symptoms that I did not understand, I increased my cortisol from 5 mg to 10 mg then 20 mg every 4 to 6 hours. I was urinating too much and increased the florinef to .2 micrograms in a desperate effort to maintain intravascular volume. After shopping in stores with high VOC content I became more and more dysautonomic, volume depleted and eventually hypokalemic from the florinef. One evening (the third night in Dallas) while lying down I placed the base of the telephone on my chest and thereafter the world went black and my arms went blue. I told my husband I must be dying and hung up to call 911. I must say it was horrifying.

I was so allergic to paper that I couldn't write on it. Yet I didn't really understand the principals of environmental medicine yet – and here was about to die from electrical and chemical sensitivity! I wrote my will – as I was sure I was dying- on my sheet and explained at the top that I was doing it because I was allergic to paper. Actually paper burned me when I touched it or got even a few feet from it. I used a commode twice hourly for 6 hours. I experienced that 'understanding' of 'how the world works' (at least environmentally and medically) that manic patients have been said to do. But I knew I was sane and was just 'doing' things that manic patients do. I now understood that thousands of people had gone before me and presented this same way to emergency rooms all over the world. They were environmentally overloaded like me but everyone looked at them and said they were crazy. I had been guilty of treating many patients with Haldol who probably needed oxygen, fluids, trisalts, and Vitamin C.

My potassium was 2.4 and eventually after it was replaced I was able to leave the hospital AMA at 3 AM. It is now my impression that it was possibly the hypokalemia and the subsequent treatment of it that stabilized the electrical sensitivity to the point where I could leave the hospital. I, therefore, wonder if giving potassium can ameliorate the symptoms and is part of the reason trisalts may work.

Evidently psychiatry cleared me and amazingly (and rightly so) decided I was not insane! Once home, I had now developed severe electrical sensitivity to all neon signs, fluorescent lights, telephones, and cardiac monitors. The display on the dashboard of the car was so bright I couldn't look at it and I couldn't even sit in the car under those lights 30 feet overhead at the grocery store parking lot – and that lasted for 2 months!

The treatment with neutralization shots in Dallas and ALF, oxygen, sauna, and IV vitamins was very effective. I left being much improved but still wearing a mask when I occasionally entered a store or walked near traffic. I eventually moved to an Island to heal. I was significantly electrically sensitive so that the computer base was 50 feet away in another part of the house and I could not talk on the phone without a head set and at that for only a few minutes before becoming dysautonomic. My hand becomes numb and painful when touching the mouse, and I

was dependant on Midodrine (an alpha agonist) 10 mg 4 times a day to raise my BP and constrict the veins in the lower extremities. I became especially worse when using these electrical appliances and had to take a dose to counteract the contact with these items. I 'pretzeled' (Streeten) my legs and felt like falling asleep do to poor cerebral perfusion. The land line phone gave me chest pain and profuse axillary diaphoresis for a couple years before the crown removal.

While there I was treated by a dentist who measured the currents and voltages in each tooth and found that most had a reading of 1 microampere and 50 millivolts or less. One crown had a reading of 11 microamperes and he removed it that day and placed a temporary. I returned home to work on my computer and make phone calls using my headset as usual.

Immediately after returning from the dentist I realized I didn't want Midodrine as usual, that I could talk on the phone for hours instead of minutes, and that I could use the computer without fatigue. I later learned that I wasn't nearly as sensitive to smoke, diesel exhaust or perfume. I basically never wore a mask again after that point! My electrical sensitivity was much improved. Instantaneously I was 90% better.

My hand stills goes numb if I touch the mouse for long. And I must use a headset for the telephone and cell phone – which I rarely use. I think folks with peripheral neuropathy in the hands who type should consider the EMF effect from conduction through the keys and especially the mouse. A 'grounding glove' works exceedingly well for this even if not plugged in to the ground (obtainable at [lessEMF.com](http://lessEMF.com)).

I have only used Midodrine on rare occasion when my legs or low back aches from exposure to a carpeted hotel for days, or when shopping (which I rarely do). And always 2.5 mg. I find this incredible response to the removal of a single crown unbelievable. In this crown there was porcelain, metal and an underlying silver amalgam. The touching of these two metals led to this phenomenon of oral galvanism. I have reviewed the literature on this subject from 1930 and I will summarize the highlights of my readings. A wonderful compilation of articles was supplied courtesy of Doug Cook. Doug (at 920 842 2083) has made a meter called the Oral Potential Meter which I think every environmental doc should have and use to test for this phenomenon in his patients. Even if there are no crowns but just multiple amalgams there can still be electrical currents generated. The worst currents occur between gold and amalgams. In the Power Point that follows I will discuss a little about the history of Oral Galvanism as well as the variety of symptoms that can arise from it. Addressing the interaction of dissimilar dental metals in the EI patient is of prime importance and should not be left to the end of treatment.

Other Resources: IAOMT 863 420 6373

DAMS 800 311 62

Questions: [Lisa@nagy1.com](mailto:Lisa@nagy1.com), 508 696 6998, [www.environmentalmedicineinfo.com](http://www.environmentalmedicineinfo.com)



## Objectives & Notes

**Cyril W. Smith, Ph.D.**

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Date of talk: Thursday, June 7, 2007

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### Training:

Current Job Description:	Retired Physicist and author
Medical School/ University Attended	University of Exeter and University of London
Other Information: (including titles of books or articles you have recently written):	“Frölich’s Interpretation of Biology Through Theoretical Physics,” “ANS Involvement in Chemical and Electromagnetic Sensitivities,” “Quanta and Coherence Effects in Water and Living Systems” and “Watergates – Logic Operations in Water”

### SPEECH TITLE: “**Electromagnetics and the ANS**”

At the end of this Presentation, the participant should be able to:

1. Recognize electromagnetic hypersensitivity in patients
2. Understand relations between endogenous and exogenous frequencies and the ANS
3. Understand ANS interactions and stability requirements

*The American Environmental Health Foundation and the University of North Texas Health Science Center is not responsible for the contents of this presentation. AEHF has not altered or modified the contents of the information provided by this speaker.*

# **Electromagnetics and the ANS**

Cyril W. Smith, Ph.D.

## **Electromagnetic Sensitivity in Patients**

Electromagnetic Sensitivity is found in patients who already have an on-going chemical sensitivity. If an environmental frequency or pattern of frequencies matches that of some toxic chemical in the body H-bonded to water. The electrical and chemical triggered symptoms are identical. Once some patient specific field or intensity threshold has been exceeded, frequency becomes the relevant parameter.

Electromagnetic Hypersensitivity actually does exist and can be elicited under environmentally controlled double-blind conditions with 100% reactions to an active frequency and 0% to the placebos. See: Rea WJ. Pan Y. Fenyves EJ. Sujisawa I. Suyama H. Samadi N. and Ross GH. "Electromagnetic Field Sensitivity", Journal of Bioelectricity 10(1&2): 241-256 (1991).

The first body system to become compromised in chemical and electrical hypersensitivities is the ANS. Just a few of the many factors which can affect the ANS have been listed in this presentation. In health, the body will be aware but, not incapacitated by them.

## **Endogenous & Exogenous Frequencies, Acupuncture Meridians & the ANS**

Each acupuncture meridian and chakra point has a characteristic pair of frequencies. Where a meridian has a link to the ANS there is an additional frequency: ~ 3 mHz for sympathetic ANS~ 0.3 Hz for parasympathetic ANS.

Acupuncture Meridians could originate with coherence between embryonic ectoderm and endoderm cells with the link persisting as the organism develops; the ectoderm to the acupuncture points, the endoderm and mesoderm to the target organs.

At Yuan Source Points or Luo Connecting Points, the frequencies of both meridians occur.

Sources of exogenous frequencies affecting the ANS include the electromagnetic environment, frequency signatures of chemicals H-bonded to water, frequency imprints in water, homoeopathic potencies and allergen dilutions.

The relationship between the acupuncture meridians and the autonomic nervous system (ANS) comes from the work of Dr. Reinhardt Voll. In his work, cited in English by Kenyon (J.N. Kenyon, "Modern Techniques of Acupuncture" Vol. 3, Chapter 11 – Disordered Autonomic Steering), Voll identifies a complete system of acupuncture points which indicate the functioning of both branches of the autonomic nervous system.

Voll regards the 'Nerve Degeneration' meridian point ND1a as the Summation Point for entire ANS. In measurements using electroacupuncture apparatus, stress is indicated by a percentage change; the frequencies measured at this and other related Voll points show similar changes.

## **Conclusions**

The conclusions from the first of these presentations are that the ANS is linked to the acupuncture meridian system and its characteristic frequencies appear at linked points additional to the meridian frequencies and that:

Living systems use chemical and electrical pathways for ANS control to avoid positive feedback instability characteristic of electronic amplification.

Chemicals carry both chemical and frequency information (in trace water)

Homoeopathic potencies carry frequency information in water with little or no chemical information. Their frequencies are related to the mother tincture and the dilution ratio with certain integer exceptions.

Chemicals and frequencies in the body can affect proteins and enzymes, DNA translation, cell progression, compromise the immune system and destabilise the ANS. Frequency can effect L/D isomeric transitions and thence control enzyme reactions.

If endogenous frequencies are weak or missing, the body may seek bio-information from frequencies in the environment to control the ANS.

Exogenous frequency patterns can mimic the frequency patterns of toxic chemicals in the body thereby triggering panic reactions in the ANS.

**25th ANNUAL INTERNATIONAL SYMPOSIUM  
On Man and His Environment in Health and Disease  
June 7-10, 2007 Dallas, Texas**

Cyril W. Smith, Ph.D.

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**Handout**

**This Handout includes a number of the Tables to be shown in these presentations which may not reproduce clearly or contain too much detail for viewing briefly but which may be useful for future reference.**

**#1. Electromagnetics and the ANS**

**Electrical Sensitivity**

Found in patients who already have an on-going chemical sensitivity. If an environmental frequency or pattern of frequencies matches that of some toxic chemical in the body H-bonded to water. The electrical and chemical triggered symptoms are identical. Once some patient specific field or intensity threshold has been exceeded, frequency becomes the relevant parameter.

**Press Call – 1984**

Hypersensitive patient with strong reaction to power lines; sensitivity a million times normal Reacting 200 yards from power lines. Enough field penetrates vehicle driving under power lines to trigger a reaction

**Electromagnetic Hypersensitivity**

Actually does exist and can be elicited under environmentally controlled double-blind conditions with 100% reactions to an active frequency and 0% to the placebos.

See: Rea WJ. Pan Y. Fenyves EJ. Sujisawa I. Suyama H. Samadi N. and Ross GH. “Electromagnetic Field Sensitivity”, Journal of Bioelectricity 10(1&2): 241-256 (1991).

## Case Summary for Patient on Slides –1984

Age 37 - married with 2 children

### **Main Complaint:**

Headache for past 22 years - right-sided with deep pain behind eyes radiating to ear.

Nausea – no vomiting, worse in last 2 years. Each attack lasts 1 week – unable to move about, must lie in bed.

No relief from many migraine tablets, even more than prescribed.

### **Other complaints:**

Blocked nose –last 10 years, operation gave no relief.

Cough – Feb-Mar and Nov-Dec.

Thunderstorms – unwell, drowsy, migraine before storm.

Allergies:

1 glass of wine – intoxicates,

coffee - oesophagitis,

perfume – rash,

onions – sore eyes.

### **Electrical testing - 12 October 1984:**

Testing started at 2 Hz - patient's eyes closed & went into second stage anaesthesia which persisted at frequencies between the symptom variations listed below:

50 & 70 Hz	Slight convulsions
4 kHz	Eyes open, fingers & toes not working
25 kHz	Eyes open, but not speaking
40-50 & 70 kHz	Slight convulsions
100 kHz	Severe whole body convulsions
240-360 kHz	Eyes open, but not speaking
3-4 MHz	Slight convulsions
12 & 21 MHz	Eyes open, but not speaking
30, 42 & 70 MHz	Slight convulsions
281-284 MHz	Eyes remained open for 10 min (osc. off)
310 MHz	Slight convulsions
340, 360, 380 MHz	Walked with assistance
<b>512 ± 3 MHz</b>	<b>Best frequency, patient almost back to normal</b>
920 & 1010 MHz	Slight convulsions
1040 & 1080 MHz	Mentally limp, weak fingers, headach.

## **Endogenous & Exogenous Frequencies & the ANS**

### **Endogenous Frequencies**

Each acupuncture meridian and chakra point has a characteristic pair of frequencies.

Where a meridian has a link to the ANS there is an additional frequency: ~ 3 mHz for sympathetic ANS~ 0.3 Hz for parasympathetic ANS.

Acupuncture Meridians may originate with coherence between embryonic ectoderm and endoderm cells persisting as the organism develops with the ectoderm forming the acupuncture points, the endoderm and mesoderm the target organs.

The normal condition is for the acupuncture meridian frequencies to fluctuate in a quasi-periodic manner over about a one hour period. If the meridian is synchronized to a stimulatory phase frequency, its rate increases ten-fold. If it is synchronized to a depressive phase frequency, all fluctuation ceases, which is analogous to the effect of a toxic chemical.

At Yuan Source Points or Luo Connecting Points, frequencies of both meridians occur.

### **Sources of Exogenous Frequencies Affecting the ANS**

Electromagnetic Environment. Chemicals H-bonded to water.

Frequency imprints in water. Homoeopathic potencies. Allergen dilutions

**Endogenous Frequencies on “Classical” Acupuncture Points**  
**(ANS related meridians in bold)**

‘Classical’ Acupuncture Meridians	Point Measured	Low Band Frequency Hz	High Band Frequency MHz
Lung	Lu1	0.48	24
Large Intestine	LI1	0.055	2.7
<b>Stomach</b>	<b>St45 / right</b>	<b>0.044</b>	<b>22</b>
<b>Stomach</b>	<b>St45 / left</b>	<b>0.44</b>	<b>2.2</b>
Spleen	Pn1	0.055	2.7
<b>Heart</b>	<b>He9</b>	<b>7.8</b>	<b>380</b>
Small Intestine	SI1	0.025	1.2
<b>Urinary Bladder</b>	<b>BL67</b>	<b>5.5</b>	<b>270</b>
<b>Kidney</b>	<b>Ki1</b>	<b>0.00095</b>	<b>0.047</b>
Pericardium	Pe9	0.25	13
<b>Sanjiao (TW)</b>	<b>TW1</b>	<b>6000</b>	<b>300,000</b>
<b>Gall Bladder</b>	<b>GB44</b>	<b>0.05</b>	<b>2.46</b>
Liver	Liv1	4.8	240
Du Mai (GV)	GV14	4.3	149
Ren Mai (CV)	Ren24	14	730

**Endogenous Frequencies on Additional “Ting” Points**  
**(ANS related points in bold)**

Voll’s Additional Acupuncture ‘Ting’ Points	Point Measured	Low Band Frequency Hz	High Band Frequency MHz
Lymphatics	Ly1	0.06	2.95
Nerve Degeneration	ND1	0.00055	0.027
Allergy	AD1	2	98.4
Organ Degeneration	Or1	0.078	3.85
Fatty Degeneration	FatD1	0.74	36
Skin Degeneration	Sk1	0.0035	0.172
Joint Degeneration	JD1	0.3	148
Fibroid Degeneration	FibD1	800	39,400
Circulation, pericardium	Ci9	0.05	2.46

## Voll's Electroacupuncture Points Linking to the ANS

### Nerve Degeneration Meridian (ND1a) Summation Point for Entire ANS

St10a →	<u>Summation Point for Parasympathetic ANS</u>	<u>Summation Point for Sympathetic ANS</u>	← <u>GB20</u>
GB11b	Vagus nerve nucleus in medulla	Sympathetic nerve - cranial	GB19a
St8c	Vagus nerve -cervical	Sympathetic nerve - cervical	GV16
St8d	Pharangeal plexus	Cervical ganglion	TW1a
St16	Vagus nerve -thoracic	Sympathetic trunk – thoracic	BL16*
St15	Oesophageal plexus	Sympathetic trunk – abdominal	BL24*
St18	Pulmonary plexus	Coeliac plexus	St44c
St20 L/R	Gastric plexus – anterior/posterior	Sympathetic - Pelvic	BL33
Ki20	Vagus nerve - coeliac	Inferior hypogastric plexus	BL63*
Ki21	Vagus nerve - hepatic		* Summation points
Ki 19	Vagus nerve - renal		
BL35	Sacral preganglion fibres		
BL34	Pelvic plexus		
BL32	Pelvic splanchnic nerves		

## **Conclusions**

The ANS is linked to the acupuncture meridian system and its characteristic frequencies appear at linked points additional to the meridian frequencies.

1. Living systems use chemical and electrical pathways for control to avoid positive feedback instability
2. Chemicals carry chemical and frequency information (in trace water)
3. Homoeopathic potencies carry frequency information in water with little or no chemical information
4. Chemicals in the body can destroy proteins and enzymes, prevent DNA translation cell progression errors and compromise the immune system and destabilise the ANS
5. If endogenous frequencies are weak or missing, the body may seek its control information from frequencies in the environment with nonsensical results
6. External frequency patterns can mimic frequency patterns of toxic chemicals in the body triggering panic reactions

## Objectives & Notes

**Stephen T. Sinatra, M.D.**

Date of talk: Thursday, June 7, 2007, 4:00pm

Optimum Health  
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### Training:

Current Job Description:	Cardiologist, Lecturer, writer
Current Faculty Appointments:	Assistant Clinical Professor of Medicine, University of Connecticut School of Medicine
Medical School/ University Attended	Albany Medical School
Internship:	Albany Medical Center
Residency:	St. Francis Hospital
Board Certifications:	FACC, CNS, CRT
Other Information: (including titles of books or articles you have recently written):	Reverse Heart Disease Now; The Fact Food Diet; The Sinatra Solution

### SPEECH TITLE: “Metabolic and Mitochondrial Defense, The Missing Link in Heart Disease”

At the end of this Presentation, the participant should be able to:

1. Define the complex role of energy and the heart.
2. Learn how the new triad of bioenergetic energy in cardiac health, i.e., coenzyme Q10, L-carnitine and D-ribose, can help prevent and overcome heart disease and the important contribution these energy-supporting nutrients make in people’s lives.
3. Discover how ATP supporting nutrients will improve symptoms of fibromyalgia and chronic fatigue.

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**Metabolic and Mitochondrial Defense**  
**The Missing Link in Heart Disease**  
*The Future of Medicine*

**Stephen T. Sinatra, M.D., F.A.C.C.**

Central to our understanding of cellular bioenergetics is the distinction between the concentration of ATP in the cell and the efficiency of ATP turnover. In ischemic or hypoxic hearts the cell's ability to match ATP supply and demand is disrupted, causing both depletion of the adenine nucleotide pool and dysfunction in mitochondrial ATP turnover mechanisms. Metabolic therapies that help cardiomyocytes meet their absolute need for ATP fulfill a major clinical challenge of preserving pulsatile cardiac function while maintaining cell and tissue viability. D-Ribose, L-carnitine, and coenzyme Q<sub>10</sub> work in synergy to help the ischemic or hypoxic heart preserve its energy charge in support of energy consuming biochemical reactions that maintain cardiac work, allow the heart to grow and repair itself, and to survive.

The metabolic factors associated with myocardial ischemia or hypoxia are profound and have direct impact on disease pathology. Paramount in this myriad of metabolic challenges is the effect of acute or chronic hypoxia on cellular bioenergetic pathways. In ischemic disease, mitochondrial dysfunction and disruption of cardiac energy metabolism depletes cellular energy reserves, with adenine nucleotide levels declining more than 30% in congestive heart failure and 40% or more in coronary artery disease. Tissue biopsy and NMR findings confirm these conclusions.

The acute or chronic loss of energy substrates, mitochondrial damage, and disruption of normal energy utilization and supply create conditions of irreversible changes in the cell's biochemical state. Four theories have been advanced that relate to the biochemical changes contributing to the pathology of cardiac disease: i) a critical energy loss; ii) a critical accumulation of cellular calcium; iii) the effects of free radical formation; and iv) injurious effects of the accumulation of long-chain acyl compounds.

**Conclusion**

The energy-starved heart is poorly understood by physicians who treat cardiac disease on a day-to-day basis. Metabolic support with D-ribose, L-carnitine, and coenzyme Q<sub>10</sub> is critical for the maintenance of cellular energy charge in minimally oxidative ischemic or hypoxic hearts. Preservation of cellular energy charge provides the chemical driving force required to complete ATPase reactions needed to maintain cell and tissue viability and function. A new, emerging field in metabolic cardiology will be realized as clinicians choose to treat the energy-starved heart at the mitochondrial level.

***Goals and Objectives:***

1. Define the complex role of energy and the heart.
2. Learn how the new triad of bioenergetic energy in cardiac health, i.e., coenzyme Q<sub>10</sub>, L-carnitine and D-ribose, can help prevent and overcome heart disease and the important contribution these energy-supporting nutrients make in people's lives.
3. Discover how ATP supporting nutrients will improve symptoms of fibromyalgia and chronic fatigue.
4. Learn how mitochondrial defense is a cardinal factor to understanding the nature of heart disease.
5. Explore how targeted nutraceuticals can help people survive heart disease.

***References:***

1. Chopra RK, Goldman R, Sinatra ST, Bhagavan HN. Relative bioavailability of coenzyme Q<sub>10</sub> formulations in human subjects. *Int J Vitam Nutr Res.* 1998;68(2):109-13.
2. Berman M, A Erman, T Ben-Gal, et al. Coenzyme Q<sub>10</sub> in patients with end-stage heart failure awaiting cardiac transplantation: A randomized, placebo- controlled study. *Clin Cardiol* 2004;27:295-299.

3. Geleijnse JM, Vermeer C, Grobbee DE, et al. Dietary intake of menaquinone is associated with a reduced risk of coronary heart disease: the Rotterdam study. *J Nutr.* 2004;134(11):3100-3105.
4. Ingwall JS, RG Weiss. Is the failing heart energy starved? On using chemical energy to support cardiac function. *Circ Res* 2004;95(2):135-45.
5. Kaneki M, Hosoi T, Ouchi Y, et al. Pleiotropic actions of vitamin K: protector of bone health and beyond? *Nutrition.* 2006;22(7-8):845-852.
6. Kidd PM, et al. Coenzyme Q<sub>10</sub>: Essential energy carrier and antioxidant. *HK Biomed Consult* 1998;1-8.
7. Koos R, Mahnken AH, Muehlenbruch G, et al. Relation of oral anti-coagulation to cardiac valvular and coronary calcium assessed by multislice spiral computer tomography. *Am J Cardiol.* 2005;96(6):747-749.
8. Luo G, Ducy P, McKee M, et al. Spontaneous calcification of arteries and cartilage in mice lacking matrix GLA protein. *Nature.* 1997;386:78-81.
9. Omran H, D MacCarter, JA St. Cyr, B Luderitz. D-Ribose aids congestive heart failure patients. *Exp Clin Cardiol* 2004;9(2):117-118.
10. Omran H, et al. D-Ribose improves diastolic function and quality of life in congestive heart failure patients: A prospective feasibility study. *Eur J Heart Failure* 2003;5:615-619.
11. Pauly D, C Johnson, JA St. Cyr. The benefits of ribose in cardiovascular disease. *Med Hypoth* 2003;60(2):149-151.
12. Pauly D, C Pepine. D-Ribose as a supplement for cardiac energy metabolism. *J Cardiovasc Pharmacol Ther* 2000;5(4):249-258.
13. Schurgers LJ, Aebert H, Vermeer C, et al. Oral anticoagulant treatment: friend or foe in cardiovascular disease? *Blood.* 2004;104(10):3231-3232.
14. Schurgers LJ, et al. Regression of warfarin-induced medial elastocalcinosis by high intake of vitamin K in rats. *Blood.* 2006 Nov; [Epub ahead of print].
15. Schurgers LJ, et al. Vitamin K-containing dietary supplements: comparison of synthetic vitamin K1 and natto-derived menaquinone-7. *Blood.* 2006 Dec; [Epub ahead of print].
16. Schurgers LJ, Teunissen K, Knapen M, et al. Novel conformation-specific antibodies against matrix  $\gamma$ -carboxyglutamic acid (GLA) protein: undercarboxylated matrix GLA protein as a marker for vascular calcification. *Arterioscler Thromb Vasc Biol.* 2005;25(8):1629-1633.
17. Sinatra ST. Alternative medicine for the conventional cardiologist. *Heart Dis.* 2000 Jan-Feb;2(1):16-30.
18. Sinatra ST. Coenzyme Q10 and congestive heart failure. *Ann Intern Med.* 2000 Nov 7;133(9):745-6.
19. Sinatra ST. Coenzyme Q10: a vital therapeutic nutrient for the heart with special application in congestive heart failure. *Conn Med.* 1997 Nov;61(11):707-11.
20. Sinatra ST. Refractory congestive heart failure successfully managed with high dose coenzyme Q10 administration. *Mol Aspects Med.* 1997;18 Suppl:S299-305.
21. Singh RB, et al. A randomized, double-blind, placebo-controlled trial of L-carnitine in suspected myocardial infarction. *Postgrad Med* 1996;72:45-50.
22. Singh RB, et al. Effect of coenzyme Q<sub>10</sub> on risk of atherosclerosis in patients with myocardial infarction. *Mol Cell Biochem* 2003;246:75-82.
23. Zimmer HG. Regulation of and intervention into the oxidative pentose phosphate pathway and adenine nucleotide metabolism in the heart. *Mol Cell Biochem* 1996;160-161:101-109.



## Objectives & Notes

**Allan D. Lieberman, M.D.**

Date of talk: Thursday, June 7, 2007, 4:30pm

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### Training:

Current Job Description:	Medical Director – Center for Occupational and Environmental Medicine
Medical School/ University Attended	Chicago Med School
Internship:	Mt. Sinai Hospital – Chicago
Residency:	Children’s Memorial Hospital
Board Certifications:	American Board of Environmental Medicine

### CASE STUDY: “Epidemic of Aflatoxin Poisoning in Contaminated Pet Food”

At the end of this Presentation, the participant should be able to:

1. Be aware of presence of Aflatoxins in pet food derived from grains.
2. Recognize that if animals can get sick from exposures to contaminated foods, so can workers processing the feed.
3. Recognize manifestations of mycotoxins exposure.

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## **MEDICAL REPORT ON M.R.**

M.R., a 37 year-old male, was initially seen at the Center for Occupational and Environmental Medicine on November 14, 2006. The medical evaluation and the derived opinion is based upon:

1. A complete life history including:
  - a. Medical history
  - b. Developmental history
  - c. Personal and social history; drug, alcohol and tobacco history
  - d. Environmental history
  - e. Occupational history
  - f. Dietary history
  - g. Family history
2. Physical/neurological examinations
3. Laboratory studies
4. Previous medical records
5. My education, training and experience in the practice of Occupational and Environmental Medicine, 1979 to present; Diplomate, American Board of Environmental Medicine; Fellow, American Academy of Environmental Medicine; member of the American College of Occupational and Environmental Medicine.

### **PERTINENT FINDING OF MEDICAL HISTORY:**

M.R. came to the Center for Occupational & Environmental Medicine referred by Dr. Jack Thrasher and his attorney, requesting evaluation of his multiple problems associated with exposures to his work place. He has specifically asked for help with treating his condition.

1. Born 1969, Massachusetts, USA, 7 lbs, bottle-fed, 3rd of 4 children
2. Thrush
3. Lives in rural area of New York
4. Worked after school from age 16
5. US Army 19 years of age, began smoking ½ pack of cigarettes a day
6. Germany. Transported GB and VX (Poison gases)
7. Georgia age 21. Infectious Mono. Lost 33 pounds
8. Florida. US army reserves- 3 years
9. Security Department for University of Florida- 6 months. Had pre-employment physical and chest x-ray

10. Works for PCR- Chemical manufacturing- exposure to TCE, ammonia, silicone. Wore protective gear and forced air mask. Exposed to a chemical spill
11. Moves to horse farm- age 27
12. Warehouse work for KMART age 30-31
13. Diamond Pet Food- Gaston, SC. January 29, 2005. Has pre-employment physical. Loading and shipping.
14. 320,000 pounds of corn arrive.
15. December 21, 2005. Discover aflatoxin (1800-2000 ppb) standard 20 ppb. Works 15 hours a day through January 09. 2006. No protective gear is used.
16. Sick: Bronchitis, sinusitis, high fever and nosebleeds
17. 116 dogs reported dead from New York to Florida from eating contaminated dog food.
18. January 09, 2006- changes buildings. Walls and floors covered with BLACK MOLD. Handling open and loose dog food.
19. Dry scaly rashes on elbows and knees. Has persistent cough
20. January 17, 2006. Plant meeting called by management. “No health ramifications from workers exposure to contaminated corn”.
21. March 2006. Extreme fatigue, headaches, anorexia, vomiting, nosebleeds.
22. April 2006: Large lump in axilla. April 10 – May 31, back to main plant. May 31, 2006, abrupt onset of high fevers to 104 degrees, back soreness, chest tightness, “wheezing”, shortness of breath, dry cough, fatigue, headaches, myalgia and sinus drainage. Sick for 2 weeks.
23. Sees Doctor for the first time. Dr. Mayson Diagnosed with a virus. Employer sends him to occupational doctor. Receives chest x-ray – initially read as normal. TNF blood test- normal  
 June 02, 2006 Dr. Wingo PPD negative  
 June 05, 2006: third visit in 5 days. Prescribed Tylenol

June 06, 2006	June 09, 2006:	June 17, 2006
	AST 107	AST 50
	ALT 153	ALT 97
	ALKP04Tase 198	ALKP04Tase 158
	GGT 265	
	June 09, 2006	June 17, 2006
	Glucose 261 mg/dl	191 mg/dl
WBC 2600	WBC 4400	Albumin decrease 3.2
Hb 14.2	Hb 13.2	
Platelets 105	Platelets decrease 120	
	TNF alpha 7.3 pg/ml (normal)	

CT Scan lungs  
 Numerous tiny pulmonary parenchymal nodule borderline to enlarged mediastinal and hilar nodes.

Urine: Amber

Bilirubin- small  
Urobilinogen 4.0 (normal range 0.1- 1.0)  
Angiotensin Converting enzyme- 61 (Normal range 9-67)

24. All of June 2006- Urine dark red, thick and syrupy. “Muscles melting”, dehydrated, low back pain, headaches, confused, myalgia, chest pain, sudden onset difficulty in breathing, night sweats. Shortness of breath, dizziness, dry hacking cough, weight loss of 23 pounds.

25. Cat Scan of chest. Cluster of grapes- nodules through out lung fields. Bronchoscopy “clear airways”, needle aspiration. Washings, negative for virus and malignancy.

26. June 27, 2006 Laparoscopy-Biopsy of mediastinal node shows fibrotic, numerous non-caseating granulomas through out node. Compatible with sarcoidosis.

27. Histology of mediastinal nodes: 54% lymphocytes, majority B-Cells

June 14, 2006: Hepatitis profile- negative for A, B, C

June 30, 2006: No yeast, fungi isolated at 2 weeks.

July 05, 2006: Surgeon diagnosed Sarcoidosis

July 13, 2006: White blood cells increase 5.5

Hemoglobin increase 14

Platelet count increase 206

Sed rate 17 (less than 15)

Glucose 123 (2 pm)

Liver enzymes are all normal

Pulmonary function test greater than 100%

28. Out of work until July 24, 2006

29. Returns to work on July 24, 2006. When re-exposed to contaminated corn, develops shortness of breath and panic attacks when loading feed into 55-meter containers.

30. August 28,2006: Stops work at Diamond. Denied workers compensation from Travelers Insurance.

31. Goes to work for Target, unloading containers (1 month)

32. Begins working for Pepsi Bottling Co. in a clean environment. October 24, 2006

33. Referred to Center for Occupational and Environmental Medicine November 14, 2006 for comprehensive evaluation. Sequelae: Fatigue, profound memory loss, shortness of breath, dry cough, chest tightness, myalgia, weakness, depression, confusion and anxiety.

## **MEDICAL DIAGNOSIS**

History of exposure to moldy corn grain contaminated with aflatoxins (1800-2000 ppb) and other contaminants and to black mold on floor and walls of plant building (January 09- May 31, 2006) resulting in injury to and dysfunction of multiple organs and systems including:

I. Respiratory System

- a. Rhinitis, Sinusitis, Bronchitis, nose bleeds
- b. Interstitial lung disease
  - 1) Hypersensitivity Pneumonitis
  - 2) Sarcoidosis
- c. Normal Pulmonary function testing
- d. Dyspnea, chest tightness, cough

II. Gastrointestinal Systems

- a. Anorexia
- b. Nausea
- c. Vomiting
- d. Liver dysfunction

1. Elevated enzymes, GGT, ALT, AST, ALK Phosphate, elevated urobilinogen  
Negative for Hepatitis A, B and C.

III. Constitutional Signs and Symptoms

- a. Fever
- b. Headaches
- c. Weight Loss (23 pounds)

IV. Musculo-Skeletal Systems

- a. Arthralgia
- b. Myalgia
- c. Muscle weakness, atrophy

V. Blood and Lymph

- a. Elevated sedimentation rate
- b. Anemia
- c. Thrombocytopenia (105,000)
- d. Angiotensin converting enzyme- normal
- e. Leucopenia (2600)
- f. Lymphadenopathy- hilar, axillary. Fibrosis, numerous non-caseating granulomas. Cells predominately B-cells.

VI. Metabolism

- a. Diabetes type II.

VII. Nervous System- Neurotoxicity

- a. Memory loss
- b. Confusion
- c. Anxiety/ panic disorder
- d. Depression
- e. Irritability

**DISCUSSION**

The question must be asked: What disease or injury did this worker develop or sustain?

This worker unequivocally had an interstitial lung disease consistent with known causes but also of unknown etiology. A review of table 16-5 from Robbins textbook of pathology outlines the known and unknown causes of interstitial lung disease. (See attached pdf document of table 16-5) Organic dusts from grains, moldy grains are documented causes and this worker was unequivocally exposed to moldy corn and becomes acutely ill after these exposures. His acute clinical presentation is consistent with hypersensitivity pneumonitis as well as pulmonary mycotoxicosis an atypical form of FARMERS LUNG DISEASE. But because of the presence of diffuse lymph node involvement demonstrating non-caseous granulomas, Sarcoidosis was also considered as consistent with his disease. Sarcoidosis also causes interstitial lung disease but no absolute cause has so far been identified.

Aflatoxicosis was identified as the dominant cause of death in the 116 dogs evaluated. The pathway of aflatoxins into the body is primarily from eating moldy grains. But the medical literature also documents an airborne pathway. It is more likely that the pathway of antigen irritants into the workers body was through breathing. Also, aflatoxins may only be one of several hazardous agents capable of causing his injuries and disease. The moldy corn could also have thermophilic bacteria, another major cause of hypersensitivity pneumonitis.

Temporality is the single most important criterion for establishing causation. The onset of this workers illness clearly begins after his workplace exposures. The BLACK MOLD on walls and floor of alternative work site beginning January 09, 2006 is also a factor. He becomes abruptly ill on May 31, 2006 after having been relocated back to the main plant on April 10, 2006.

The acute onset of fever and respiratory distress is classical of hypersensitivity pneumonitis. As is recurrence when re-exposed to the provoking antigens in his contaminated work place. Dr. Mayson documents a two-week history of fever (104 degrees), chills, and chest tightness with some sneezing, dyspnea, dry cough, fatigue, myalgia, headaches and sinus drainage. The “cluster of grapes” nodules throughout the lung fields represent the interstitial lung disease.

The presence of abnormal liver function noted on June 09, 2006 suggests a more causative role for the known aflatoxin. This is further supported by return to normal liver functions when away from the work place from may 31, 2006 to July 24, 2006.

M.R. returns to work on July 24, 2006. OSHA recommends the contaminated corn be incinerated but the company elects to bury it in 55-meter containers in the county landfill. When he is re-exposed to the contaminated corn, he develops shortness of breath and panic attacks. He stops work on August 28, 2006 never to return to Diamond’s employment.

He describes the sequelae of his “toxic” exposures to include:

“Profound memory loss, shortness of breath, cough, chest tightness, muscle pain, weakness, nervousness, anxiety, panic and confusion”.

In establishing causation, 7 criteria modified from Sir Bradford Hill are used. These criteria ask:

1. *Is the agent implicated known to be able to cause the injury or disease in question?*

Applying these accepted criteria for establishing causation it is this consultants opinion that M.R. did suffer work related injuries from his exposures at Diamond Pet Food because:

1. The alleged hazardous agents, grain dusts and their contaminants are capable of causing interstitial lung disease. As demonstrated in table 16-5 from Robbins Pathologic Basis of Disease. P790 4<sup>th</sup> edition 1989

Corn is a source of multiple mycotoxins in addition to aflatoxins, which are capable of causing neurotoxicity, immunotoxicity and hepatotoxicity. See table 24-3 P795 (see attached pdf document for table)

These mycotoxins including aflatoxins can be inhaled.

1. Sorenson WG et al  
Aflatoxin in respirable corn dust particles. *J. Toxicol Env. Health* 1981. 7:669-72
2. Burg WA et al  
Measurement of airborne aflatoxins during handling of contaminated corn. *Am. Ind Hyg Asso J.* 1981-42: 1-11
3. Dutkiewicz J et al  
Levels of bacteria, fungi and endotoxin in bulb and aerosolized corn silage. *Appl environ Microbial* 1989 55: 1093-9
4. Curtis L, Lieberman A. et. Al  
Adverse Health effects of indoor mold. *J. Nutrition and Environ. Med* 2004, 14: 261-74

Thus, the medical experience and literature documents the ability of the multiple work place exposures to cause the injuries and disease suffered by this worker.



# 25<sup>th</sup> ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT Schedule

## Friday, June 8, 2007

8:00 **ANNOUNCEMENTS/MODERATOR:** Stephanie McCarter, M.D.

8:05 Russel J. Reiter, Ph.D., "Light Pollution: Physiological Consequences"

8:25 Q & A

8:35 Doris Rapp, M.D., "Political Aspects of the Good, Bad, and Ugly in Medicine"

8:55 Q & A

9:05 Stephen T. Sinatra, M.D., "Plaque Reversal, the Solution"

9:25 Q & A

9:35 Martha Stark, M.D., "The Wisdom of the Matrix: Regulation, Balance, and Harmony"

9:55 Q & A

### 10:05 BREAK WITH EXHIBITORS

10:50 Tang G. Lee, Ph.D., "Attenuating Electromagnetic Radiation in Buildings"

11:10 Q & A

11:20 Riki Ott, Ph.D., "Toxicological Paradigm Shifts after the Exxon Valdez Oil Spill"

11:40 Q & A

### 11:50 OPEN LUNCH

1:00 **MODERATOR:** Doris Rapp, M.D.

1:00 Jon Pangborn, Ph.D., "Perpetuation of Inflammation by Epigenetic Influences"

1:20 Q & A

1:30 Paul R. Cheney, M.D., Ph.D., "Cardiac Diastolic Dysfunction in Chronic Fatigue Syndrome"

1:50 Q & A

2:00 Mohamed B. Abou-Donia, Ph.D., "Organophosphorus Compound-Induced Neurotoxicity"

2:20 Q & A

2:30 Richard Weisler, M.D., "Elevated Rates of Suicides and Cancers in a Downwind Neighborhood: Why?"

2:50 Q & A

### 3:00 BREAK WITH EXHIBITORS

3:45 Divakar Dalela, M.D., "Benign Prostatic Hyperplasia - Can Dietary Modulations Prevent it?"

4:05 Q & A

4:15 Ron Overberg, Ph.D., C.C.N., R.D., "Organic Acid Analysis and Chemical Sensitivity"

4:35 Q & A

4:45 Stephanie McCarter, M.D., "Building Considerations for a Low EMF Home"

5:05 Q & A

5:15 CASE STUDY: Gerald H. Ross, M.D., "Toxic Buildings From One Simple Common Procedure"

Panel: Doctors Reiter, Rapp, Sinatra, Stark, Lee, Ott, Pangborn, Abou-Donia, Weisler, Dalela, Overberg, and McCarter

### 6:00 RECEPTION IN EXHIBIT ROOM

**FRIDAY, JUNE 8, 2007**

**ABSTRACTS**

**AND**

**HANDOUTS**

## Objectives & Notes

**Russel J. Reiter, Ph.D.**

Date of talk: Friday, June 8, 2007, 8:05am

University of Texas Health Science Center  
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### Training:

Current Job Description:	Research Scientist
Current Faculty Appointments:	Professor University of Texas Health Science Center
Other Information: (including titles of books or articles you have recently written):	Authored in excess of 1,100 articles in medical journals and has written/edited 36 books. Received (awarded) 4 honorary doctor of medicine degrees on the list of the most "highly cited" scientists in the world.

### SPEECH TITLE: "Light Pollution: Physiological Consequences"

At the end of this Presentation, the participant should be able to:

1. Realize the pervasiveness and potential health effects of light pollution
2. Determine the negative chronobiotic and tumor stimulating effects of melatonin suppression
3. Understand the potential benefits of melatonin treatment

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## **Light pollution: physiological consequences.**

Russel J. Reiter, Department of Cellular and Structural Biology, The University of Texas Health Science Center, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, E-mail: Reiter@uthscsa.edu

The goals of this presentation are a), to document how common light pollution at night has become and b), to enumerate the real and potential pathophysiological consequences of light pollution at night. The interruption of the daily dark period suppresses circulating melatonin levels which are important for optimal health.

Pineal melatonin production and release occurs primarily during the daily dark period. During human evolution, the light/dark environment to which individuals were exposed was determined primarily by the rising and setting of the sun. With the advent of the invention of artificial sources of light, the daily duration of darkness was abbreviated and, furthermore, the shorten night could be interrupted by brief or prolonged periods of light. In some cases, individuals are inadvertently exposed to light at night, e.g., by what is referred to a “transpass light” (light over which an individual has no control), which also suppressed circulating melatonin concentrations.

The consequences of excessive light pollution in terms of melatonin are two-fold. Since the duration of nocturnally elevated melatonin is proportional to the duration of darkness, the extension of light into the normal dark period and/or the exposure to light in the morning (when it is still night) truncates melatonin production. Secondly, interruption of the dark period with acute artificial light exposure causes a precipitous drop in pineal melatonin production and, as a result, blood melatonin concentrations plummet to low daytime values. The net result of these two scenarios is a reduction in the total amount of melatonin the pineal gland produces during a 24 hour period.

This loss of melatonin due to light pollution is not trivial. Epidemiological studies have documented an increased breast cancer incidence in female night shift workers while the frequency of colorectal cancer is reportedly elevated in both men and women who regularly work at night. It is estimated that, world wide, perhaps 25% of the work force normally does so at night.

Melatonin is known to be an important endogenous oncostatic agent. As a result, reducing circulating levels of this indole due to light exposure at night would be expected to aggravate cancer incidence. Melatonin has a variety of actions on both initiation of cancer and on neoplastic cell growth. As a potent free radical scavenger and indirect antioxidant, melatonin prevents damage to DNA which is a result of free radicals. Since it is estimated that 75% of the cancer that develops in humans is believed to be, initially, due to free radical mutilation of DNA, the loss of the highly protective melatonin is significant. Many experimental studies have confirmed that melatonin reduces DNA damage that is mediated by free radical-generating agents/processes, e.g., ionizing radiation.

Once initiated, melatonin also is a highly effective inhibitor of neoplastic cell proliferation and tumor enlargement. The mechanisms by which melatonin attenuates the growth of cancer cells seem to be numerous. Since many tumor cells possess membrane receptors for melatonin, some of the indole’s inhibitory effects are receptor mediated while others may be receptor independent. Via its receptors, melatonin inhibits the uptake of growth factors, e.g., fatty acids and especially linoleic acid (LA), by neoplastic cells. This reduction in intracellular LA reduces formation of 13-hydroxyoctadecadienoic acid (13-HODE) which activates transcription factors that mediate tumor cell proliferation. The levels of melatonin in human blood at night are sufficient to inhibit tumor cell (particularly hepatoma) proliferation via this means. Since this is the case, the loss of melatonin due to light pollution may have substantial consequences in terms of cancer.

In addition to limiting fatty acid uptake by cancer cells, melatonin also inhibits the enzyme, telomerase, which maintains the length of the telomeres on linear chromosomes. In normal cells, telomeres shorten at each cell division which makes the chromosomes fragile and predisposes the cells to death. Many cancer cells activate telomerase activity which maintains the length of the telomeres rendering the chromosomes more stable and the cells more resistant to death. Melatonin inhibits telomerase in MCF-7 human breast cancer cells causing them to die.

Beyond these two oncostatic mechanisms, melatonin also inhibits endothelin-1 synthesis. Endothelin-1 is an important angiogenic factor for growing tumors. By inhibiting the rate limiting enzyme in endothelin-1 production, i.e., endothelin-1 synthesizing enzyme, melatonin “starves” tumors of important nutrients and growth factors and, as a consequence, the cells die and the tumors grow less rapidly.

Finally, melatonin limits hormone-dependent cancer cell growth by interfering with the action of steroids, e.g., estrogen or testosterone, at the level of the cancer. Also, because free radicals promote tumor cell proliferation, as a radical scavenger melatonin can curtail proliferation of cancer cells. These multiple oncostatic actions of melatonin are obviously compromised when light at night interrupts endogenous melatonin production.

Key references:

Blask DE, Dauchy RT, Sauer LA. Putting cancer to sleep at night: the neuroendocrine 2005; 2:179-188.

Jasser SA, Blask DE, Brainard GC. Light during darkness and cancer relationships in circadian photoreception and tumor biology. *Cancer Causes Control* 2006; 17:515-523.

Reiter RJ. Potential biological consequences of excessive light exposure; melatonin suppression, DNA damage, cancer and neurodegenerative diseases. *Neuroendocrinol Lett* 2002; 23 (Suppl 2): 118-121.

Reiter RJ, Gultekin F, Manchester LC, Tan DX. Light pollution, melatonin suppression and cancer cell growth. *J Pineal Res* 2006; 40:357-358.

Sainz RM, Mayo JC, Rodriguez C, Tan DX, Lopez-Burillo S, Reiter RJ. Melatonin and cell death: differential actions on apoptosis in normal and cancer cells. *Cell Mol Life Sci* 2003; 60:1407-1426.

Sanchez-Barcelo EJ, Cos S, Mediavilla D, Martinez-Campa C, Gonzalez A, Alonso-Gonzalez C. Melatonin-estrogen interactions in breast cancer. *J Pineal Res* 2005; 38:217-222.

Schernhammer ES, Kroenke GH, Laden F, Hankinson SE. Night shift work and risk of breast cancer. *Epidemiology* 2006; 17:108-111.



## Objectives & Notes

### Doris Rapp, M.D.

8179 E. Del Cuarzo Dr.  
Scottsdale, AZ 85258

Date of talk: Friday, June 8, 2007, 8:35am

Phone: 480/905-9195  
Fax: 480/659-9500  
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### Training:

Current Job Description:	Writer, still trying to get integrated medical centers going
Current Faculty Appointments:	Clinical Assistant Professor emeritus at SUNYAB State University of New York at Buffalo
Medical School/ University Attended	New York University, Bellevue Medical College
Internship:	Buffalo Children's Hospital
Residency:	Buffalo Children's Hospital
Board Certifications:	Pediatrics, Pediatric Allergy and Environmental Medical
Other Information: (including titles of books or articles you have recently written):	Latest not in print yet is "Can Chemicals Cause Epidemics" and working on revised smaller edition of "Our Toxic World, A Wake Up Call"

### SPEECH TITLE: "Political Aspects of the Good, Bad and Ugly in Medicine"

At the end of this Presentation, the participant should be able to:

1. Know what is good and bad about recent and past EPA and FDA environmental decisions.
2. Know more of the good concerning the rise of "Green" in our world.
3. Know more about choices in relation to genetically engineered foods, dry cleaning, fish, and pets.

*The American Environmental Health Foundation and the University of North Texas Health Science Center is not responsible for the contents of this presentation. AEHF has not altered or modified the contents of the information provided by this speaker.*

## **Political Aspects of the Good, Bad and Ugly in Medicine**

**Doris J Rapp, MD**

Medicine today is a major challenge because of the extent of present day chemical pollution. An update on some of the recent and past EPA decisions that will help or harm the public is needed. The reasons why some of these decisions that can critically affect our well being, especially in relation to Genetic Engineered foods. "Green" has become an "in" term and the extent of this increasing awareness will be discussed. Choices regarding dry cleaning, fish, and pet dangers concerns also will be addressed.

## Objectives & Notes

**Stephen T. Sinatra, M.D.**

Date of talk: Friday, June 8, 2007, 9:05am

Optimum Health  
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### Training:

Current Job Description:	Cardiologist, Lecturer, writer
Current Faculty Appointments:	Assistant Clinical Professor of Medicine, University of Connecticut School of Medicine
Medical School/ University Attended	Albany Medical School
Internship:	Albany Medical Center
Residency:	St. Francis Hospital
Board Certifications:	FACC, CNS, CRT
Other Information: (including titles of books or articles you have recently written):	Reverse Heart Disease Now; The Fact Food Diet; The Sinatra Solution

### SPEECH TITLE: **“Plaque Reversal, the Solution”**

At the end of this Presentation, the participant should be able to:

1. Explore the nature of hypercoaguability and why it is often necessary to know one’s “thrombogenic potential.”
2. Discover how dietary cox -2 inhibitors can naturally inhibit plaque deposition.
3. Explore how target nutraceuticals especially vitamin K2 and other supporting nutrients can help prevent plaque deposition.

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# **Metabolic and Mitochondrial Defense**

## **The Missing Link in Heart Disease**

### **The Future of Medicine**

**Stephen T. Sinatra, M.D., F.A.C.C.**

Central to our understanding of cellular bioenergetics is the distinction between the concentration of ATP in the cell and the efficiency of ATP turnover. In ischemic or hypoxic hearts the cell's ability to match ATP supply and demand is disrupted, causing both depletion of the adenine nucleotide pool and dysfunction in mitochondrial ATP turnover mechanisms. Metabolic therapies that help cardiomyocytes meet their absolute need for ATP fulfill a major clinical challenge of preserving pulsatile cardiac function while maintaining cell and tissue viability. D-Ribose, L-carnitine, and coenzyme Q<sub>10</sub> work in synergy to help the ischemic or hypoxic heart preserve its energy charge in support of energy consuming biochemical reactions that maintain cardiac work, allow the heart to grow and repair itself, and to survive.

The metabolic factors associated with myocardial ischemia or hypoxia are profound and have direct impact on disease pathology. Paramount in this myriad of metabolic challenges is the effect of acute or chronic hypoxia on cellular bioenergetic pathways. In ischemic disease, mitochondrial dysfunction and disruption of cardiac energy metabolism depletes cellular energy reserves, with adenine nucleotide levels declining more than 30% in congestive heart failure and 40% or more in coronary artery disease. Tissue biopsy and NMR findings confirm these conclusions.

The acute or chronic loss of energy substrates, mitochondrial damage, and disruption of normal energy utilization and supply create conditions of irreversible changes in the cell's biochemical state. Four theories have been advanced that relate to the biochemical changes contributing to the pathology of cardiac disease: i) a critical energy loss; ii) a critical accumulation of cellular calcium; iii) the effects of free radical formation; and iv) injurious effects of the accumulation of long-chain acyl compounds.

### **Conclusion**

The energy-starved heart is poorly understood by physicians who treat cardiac disease on a day-to-day basis. Metabolic support with D-ribose, L-carnitine, and coenzyme Q<sub>10</sub> is critical for the maintenance of cellular energy charge in minimally oxidative ischemic or hypoxic hearts. Preservation of cellular energy charge provides the chemical driving force required to complete ATPase reactions needed to maintain cell and tissue viability and function. A new, emerging field in metabolic cardiology will be realized as clinicians choose to treat the energy-starved heart at the mitochondrial level.

### ***Goals and Objectives:***

6. Define the complex role of energy and the heart.
7. Learn how the new triad of bioenergetic energy in cardiac health, i.e., coenzyme Q<sub>10</sub>, L-carnitine and D-ribose, can help prevent and overcome heart disease and the important contribution these energy-supporting nutrients make in people's lives.
8. Discover how ATP supporting nutrients will improve symptoms of fibromyalgia and chronic fatigue.
9. Learn how mitochondrial defense is a cardinal factor to understanding the nature of heart disease.
10. Explore how targeted nutraceuticals can help people survive heart disease.

### ***References:***

24. Chopra RK, Goldman R, Sinatra ST, Bhagavan HN. Relative bioavailability of coenzyme Q<sub>10</sub> formulations in human subjects. *Int J Vitam Nutr Res.* 1998;68(2):109-13.
25. Berman M, A Erman, T Ben-Gal, et al. Coenzyme Q<sub>10</sub> in patients with end-stage heart failure awaiting cardiac transplantation: A randomized, placebo- controlled study. *Clin Cardiol* 2004;27:295-299.

26. Geleijnse JM, Vermeer C, Grobbee DE, et al. Dietary intake of menaquinone is associated with a reduced risk of coronary heart disease: the Rotterdam study. *J Nutr.* 2004;134(11):3100-3105.
27. Ingwall JS, RG Weiss. Is the failing heart energy starved? On using chemical energy to support cardiac function. *Circ Res* 2004;95(2):135-45.
28. Kaneki M, Hosoi T, Ouchi Y, et al. Pleiotropic actions of vitamin K: protector of bone health and beyond? *Nutrition.* 2006;22(7-8):845-852.
29. Kidd PM, et al. Coenzyme Q<sub>10</sub>: Essential energy carrier and antioxidant. *HK Biomed Consult* 1998;1-8.
30. Koos R, Mahnken AH, Muehlenbruch G, et al. Relation of oral anti-coagulation to cardiac valvular and coronary calcium assessed by multislice spiral computer tomography. *Am J Cardiol.* 2005;96(6):747-749.
31. Luo G, Ducy P, McKee M, et al. Spontaneous calcification of arteries and cartilage in mice lacking matrix GLA protein. *Nature.* 1997;386:78-81.
32. Omran H, D MacCarter, JA St. Cyr, B Luderitz. D-Ribose aids congestive heart failure patients. *Exp Clin Cardiol* 2004;9(2):117-118.
33. Omran H, et al. D-Ribose improves diastolic function and quality of life in congestive heart failure patients: A prospective feasibility study. *Eur J Heart Failure* 2003;5:615-619.
34. Pauly D, C Johnson, JA St. Cyr. The benefits of ribose in cardiovascular disease. *Med Hypoth* 2003;60(2):149-151.
35. Pauly D, C Pepine. D-Ribose as a supplement for cardiac energy metabolism. *J Cardiovasc Pharmacol Ther* 2000;5(4):249-258.
36. Schurgers LJ, Aebert H, Vermeer C, et al. Oral anticoagulant treatment: friend or foe in cardiovascular disease? *Blood.* 2004;104(10):3231-3232.
37. Schurgers LJ, et al. Regression of warfarin-induced medial elastocalcinosis by high intake of vitamin K in rats. *Blood.* 2006 Nov; [Epub ahead of print].
38. Schurgers LJ, et al. Vitamin K-containing dietary supplements: comparison of synthetic vitamin K1 and natto-derived menaquinone-7. *Blood.* 2006 Dec; [Epub ahead of print].
39. Schurgers LJ, Teunissen K, Knapen M, et al. Novel conformation-specific antibodies against matrix  $\gamma$ -carboxyglutamic acid (GLA) protein: undercarboxylated matrix GLA protein as a marker for vascular calcification. *Arterioscler Thromb Vasc Biol.* 2005;25(8):1629-1633.
40. Sinatra ST. Alternative medicine for the conventional cardiologist. *Heart Dis.* 2000 Jan-Feb;2(1):16-30.
41. Sinatra ST. Coenzyme Q10 and congestive heart failure. *Ann Intern Med.* 2000 Nov 7;133(9):745-6.
42. Sinatra ST. Coenzyme Q10: a vital therapeutic nutrient for the heart with special application in congestive heart failure. *Conn Med.* 1997 Nov;61(11):707-11.
43. Sinatra ST. Refractory congestive heart failure successfully managed with high dose coenzyme Q10 administration. *Mol Aspects Med.* 1997;18 Suppl:S299-305.
44. Singh RB, et al. A randomized, double-blind, placebo-controlled trial of L-carnitine in suspected myocardial infarction. *Postgrad Med* 1996;72:45-50.
45. Singh RB, et al. Effect of coenzyme Q<sub>10</sub> on risk of atherosclerosis in patients with myocardial infarction. *Mol Cell Biochem* 2003;246:75-82.
46. Zimmer HG. Regulation of and intervention into the oxidative pentose phosphate pathway and adenine nucleotide metabolism in the heart. *Mol Cell Biochem* 1996;160-161:101-109.



## Objectives & Notes

### Martha Stark, M.D.

Harvard Medical School  
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Boston, MA 02115

Date of talk: Friday, June 8, 2007, 9:35am

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### Training:

Current Job Description:	Teaching/lecture circuit and full-time private practice in psychiatric medicine and psychoanalysis
Current Faculty Appointments:	Faculty, Center for Psychoanalytic Studies, Massachusetts General Hospital, Harvard Medical School; Massachusetts Mental Health Center, Harvard Medical School; Boston Psychoanalytic Institute; and Massachusetts Institute for Psychoanalysis
Medical School/ University Attended	Harvard Medical School
Adult Psychiatry Residency:	The Cambridge Hospital, Cambridge, MA
Child Psychiatry Fellowship:	Massachusetts Mental Health Center, Boston, MA
Psychoanalytic Training:	Boston Psychonalytic Institute, Boston, MA
Board Certifications:	American Association of Psychiatric Medicine
Other Information: (including titles of books or articles you have recently written):	Stark M (1994). Working with Resistance. Northvale, NJ: Jason Aronson; Stark M (1994). A Primer on Working with Resistance. Northvale, NJ: Jason Aronson; Stark M (1999). Modes of Therapeutic Action: Enhancement of Knowledge, Provision of Experience, and Engagement in Relationship. Northvale, NJ: Jason Aronson.

### SPEECH TITLE: “The Wisdom of the Matrix: Regulation, Balance, and Harmony”

At the end of this Presentation, the participant should be able to:

1. Recognize the significance of the fact that all living tissues are liquid crystals
2. Appreciate the importance of the interconnectedness and interdependence of the different components of the living matrix
3. Understand the concept of resilience and the body’s ability to adapt to environmental stressors at ever newer homeostatic set points and ever higher levels of order and complexity
4. Recognize the costliness of physiological adaptation in terms of depletion of the body’s nutrient and energetic reserves
5. Appreciate the significance of the living matrix as a regulatory system, which makes possible the maintenance (or, if lost, the recovery) of the body’s balance, order and harmony

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**The Wisdom of the Matrix: Regulation, Balance, and Harmony**  
**Martha Stark, M.D. - June 7 - 10, 2007**  
**25th Annual International Symposium**  
**on Man and His Environment in Health and Disease**

important to consider the impact of the myriad interlinking factors responsible for overall health and vitality  
a systemic (or holistic) approach speaks to the interdependence of the following components --

- (1) the body's various regulatory systems (including, but not limited to, the endocrine, nervous, and immune systems);
- (2) the body's numerous physiological processes (such as those responsible for maintenance of body temperature; achievement of acid-base balance; regulation of blood sugar levels; production of energy; handling of oxidative stress; detoxification; renewal, repair, and defense against disease);
- (3) genetic uniqueness and biochemical individuality;
- (4) lifestyle choices (including, but not limited to, diet, nutrition, exercise, and sleep);
- (5) environmental stressors;

psychological stressors - involve both the "presence of bad" and the "absence of good" in the early-on parent-child relationship, both "too much that was bad" between parent and child and "not enough that was good" - psychological trauma and abuse on the one hand, emotional deprivation and neglect on the other

environmental stressors - involve, more generally, both "too much bad" (that is, too much that is disease-promoting) and "not enough good" (that is, not enough that is health-promoting), too many free radicals and not enough antioxidants, too many antibiotics and not enough probiotics, too many pollutants and not enough nutrients

the cumulative impact of which constitutes stress and must ultimately be corrected for through both the elimination of bad in order to lighten the total body load and the supplementation with good in order to replenish the total body reserves

- (6) integration of mind, body, and spirit.

a systemic (or holistic) approach takes into consideration not just the individual parts but also the system as a whole

whereas reductionism involves analysis of the system's individual components, a systems perspective appreciates the interdependence of the system's individual components (that is, appreciates the system's complexity) and focuses attention on the properties of the system that emerge as a result of the synergistic interaction of the system's various components

the aim of a systemic (or holistic) approach is to complement, not replace, one that is more reductionist

to demonstrate the limitations of a reductionist approach:

most psychopharmacologists believe that aberrant feelings speak to abnormal or imbalanced levels of neurotransmitters in the brain

regulation of mood can therefore be achieved, they reason, by specifically targeting, with drugs, the levels of these chemical mediators. Indeed, psychotropic medications (from antidepressants to antipsychotics) do just this

but is it any wonder, then, that so many psychiatric patients fail to benefit from psychopharmacologic intervention?

does not so-called "treatment resistance" speak to this reductionism -- this too-narrow-a-focus on imbalanced neurotransmitter levels and this too-limited-a-perspective that fails to consider not just the underlying causes for these imbalances but also the contributions of the myriad other factors (including the environmental, genetic, and lifestyle ones mentioned earlier) constituting the overall clinical picture?

only a small percentage of state-of-the-art psychopharmacologists are beginning to espouse a more holistic approach, one that is both broader-based and more medicalized, one that takes into account the various components of a systemic approach, appreciating the multiplicity of factors involved in the regulation of mood

in contradistinction to the reductionism of most conventional health care professionals, environmental medicine practitioners are interested in what causes the symptom (what causes the cough, the fever, the inflammation, the depressed mood)

whereas in traditional medicine the primary concern of the health care practitioner is to alleviate the symptom by way of such anti-symptom medications as antitussives, antipyretics, anti-inflammatories, and antidepressants (the patient's mother has just died, give her a pill so she won't feel so bad about it), the primary concern in environmental medicine is to ferret out the sources of the symptom so that the deeper issues can be addressed

as Jonathan Wright is wont to say, attempting to eliminate a symptom instead of dealing with its underlying causes is like putting the lid on a boiling pot of water to keep it from spilling over instead of turning off the stove

in environmental medicine, it is understood that symptoms are the way the body signals that there is underlying dysfunction, imbalance

symptoms, then, are a manifestation of the body's wisdom

but not only do symptoms "alert" the practitioner to the existence of more fundamental problems but they are also sometimes part of the solution

if a patient is coughing or vomiting, as long as it's not life-threatening, it may well be better to allow the body these opportunities to rid itself of whatever noxious agents are prompting the discharges than to attempt elimination of the symptoms with drugs

or if a patient, new to contact lenses, finds that they are irritating her eyes, better that she listen to the wisdom of her inflamed eyes protesting "no" than that she attempt to override this alarm signal

in other words, symptoms can be adaptive and part of the body's efforts to heal itself

as such, they indicate that the body is mobilizing its own resources...

by way of another example, when the body encounters a foreign invader, the body may well respond by developing a fever to kill off the unwanted microorganisms

the fever, a symptom, is therefore part of the body's self-protective response to the presence of an environmental stressor and part of what enables the body to recover

a study done in 1988 on children with chicken pox not surprisingly found that those children treated with antipyretics to reduce their fevers fared less well than those whose fevers went untreated

in other words, those children who were allowed to heal naturally had an accelerated rate of recovery, an instance, obviously, of the symptom (the fever) being part of the cure

on a somewhat more pedestrian note -

when the body perspires, it releases chemical pollutants (many of which are cancer-producing) that have accumulated in the sweat glands

the use of antiperspirants will obviously interfere with the release of these toxins

is it any wonder that breast cancer occurs most often in the upper outer quadrant of the breast, in the area just below the armpit?

it would seem that, here too, the symptom (the perspiring) is part of what promotes health and the symptom-relieving treatment (the antiperspirant) is part of what promotes disease

parenthetically, it is important to recognize that when a drug causes side effects, this is one of the ways the body is signaling that it wants to be rid of the drug

to think that there was a time when I would say to my psychiatric patients that, although they might initially experience some uncomfortable side effects from the drugs I was prescribing, their bodies would eventually rebalance and the "adverse reactions" would disappear

little did I realize that I was advising my patients to override the message being delivered by their body and to rely instead upon their body's ability to adapt, over time, to chronic stress

I was not appreciating the cost to their systems of such adaptations

to return to the idea of a systemic (or holistic) approach to treatment and the body as a complex network of interlocking components, regulatory processes, and negative and positive feedback loops

this conceptualization of the "system" affects the way in which the environmental medicine practitioner positions herself in relation to the breathtakingly complicated, highly textured, supremely unique individual who presents to her office in search of balance, harmony, and optimal health

to summarize -

mainstream medicine is a downstream approach - the patient comes in sick and in search of relief for her symptoms

but environmental medicine advocates an upstream approach - if underlying dysfunction and imbalance in the patient can be proactively detected and addressed, the development of illness may well be prevented and symptoms will then be unnecessary

again, environmental medicine focuses attention upon the complex interplay of a whole host of factors and their synergistic impact on an individual's mental, physical, and spiritual wellbeing

in the language of complexity theory, a person's overall health and vitality can be conceived of as a supersystem, comprised of the various interdependent regulatory subsystems mentioned at the beginning of my talk, subsystems that are in intimate and precise relationship with one another and able to exchange information and energy amongst themselves and with the environment

and, to anticipate, it is this very exchange of information and energy (amongst and between) that will enable the system to maintain its dynamic balance by evolving to ever higher levels of order and complexity in the face of challenge and in defiance of the second law of thermodynamics, which has it that ordered systems will tend to "run down" over time, that is, will tend toward "disorder" and randomness (or increasing entropy) -- unless they are open to regulatory input from the outside

use of the word "disorder" here is an auspicious term, inasmuch as it speaks not only to the idea of randomness (the less-than-optimally-ordered distribution of molecules) but also to the idea of medical disease

medical disorders are indeed about disordered distribution of molecules and therefore disruption to the ease of flow of information and energy -- "dis-ease" if you will -- dis-order, dis-ease

before I move on, I would like to offer a brief synopsis of complexity theory (also known as general systems or chaos theory) and a few definitions:

open systems are (1) chaotic; (2) complex adaptive; (3) nonlinear dynamic; and (4) self-organizing with emergent (or collective) properties

chaotic means seeming randomness but underlying order that emerges, over time, as the system evolves;

complex speaks to the multiplicity of interlinking components with underlying, though not always readily apparent, order;

adaptive means responsive to regulatory input from the environment, whether that input be toxic or therapeutic;

nonlinear, whereby the evolution of the system is subject to the laws of deterministic chaos, the behavior of the system in part predictable, in part random;

dynamic, in the sense of evolving to novel configurations, over time, as a result of interactions with the environment;

self-organizing, whereby the system spontaneously evolves, with time, to ever increasing order and complexity; and, finally,

emergent, which suggests the arising of essential or collective properties, during the process of self-organization, that are not predetermined

a prime example of a complex or chaotic system in the realm of the non-living is, of course, the stock market  
what's predictable are the rallies and the corrections; what's unknowable in advance is their timing...

in any event, all complex systems have a broad-based dispersal of power, no one component more fundamental than another

so, too, the body, wherein the control of the system (the body's health and vitality) is highly dispersed and its proper functioning dependent on the integrated activities of every component...

in 1932, Walter B. Cannon, in his groundbreaking volume entitled **The Wisdom of the Body**, introduced the concept of homeostasis to describe the various regulatory mechanisms utilized by the body to maintain or, if lost, to restore the constancy of its internal environment

Cannon captured the wondrousness of the body's ability to preserve its balance in the face of ongoing challenge with the following: "...the system is open, engaging in free exchange with the outer world..." and, later, "...the structure itself is not permanent but is being continuously broken down by the wear and tear of action... and as continuously built up again by processes of repair"

maintenance of a steady internal biochemical, physiological, and energetic balance (in the face of environmental perturbation) - an ongoing, dynamic, regulatory process of compensatory microadjustments, the body, in its infinite wisdom, somehow preconsciously "knowing" to institute regulatory measures to "correct for" potential or actual disruption to its equilibrium

Hans Selye, author of the 1956 classic **The Stress of Life**, took up where Cannon had left off

long intrigued by the body's nonspecific response to generic unpleasantness, Selye eventually developed the idea of a general adaptation (or stress) syndrome, a three-stage model that addressed not just the impact of the "usual and customary" stresses of everyday life with which Cannon had been primarily concerned but also the cumulative impact over time of unusual and extreme stress

Selye's stress response speaks directly to the body's ongoing efforts to restore its balance in the face of both acute and chronic stress

drawing upon the seminal ideas of Cannon, Selye, and others who have followed in their steps, we now understand the three stages of the stress response to be as follows --

first is the alarm stage of the acute stress response, characterized by heightened arousal and a mobilization of the body's defenses in the interest of self-protection

but if the stress persists, then the body, in response to the cumulative impact of both too much bad and not enough good, will transition into the second stage of the stress response, namely, resistance and adaptation, characterized by an intensification of the body's defensive efforts (for example, through upregulation of its enzyme detoxification systems or overactivation of its sympathetic nervous system), all with an eye either to fending off, that is, resisting, the stressor and/or to making whatever internal adjustments are necessary in order to live with, that is, adapt to, the stressor

in other words, if you can't beat it, then join it; if you can't resist it, then adapt to it

the concept of adaptation therefore encompasses the variety of regulatory, compensatory, and defensive mechanisms employed by the body to maintain its balance and harmony in the face of ongoing stressors

upregulation and downregulation; activation and deactivation

in essence, adaptation prompts reconstitution at ever newer homeostatic set points -- but each time at some cost to the system in terms of its adaptation reserves (that is, its nutrient and energetic resources). The body, in its infinite

wisdom, adapts -- necessary for short-term survival but costly to the system over the long haul and certainly a major contributor to the body's accelerated aging

adaptation (as is true for every defense, whether physiological or psychological) involves both "gain" to the system and "pain"; it is both beneficial to the individual and, ultimately, detrimental, both protective and, ultimately, damaging

with the concepts of allostasis and allostatic load, Bruce McEwen, Robert Sapolsky, and Peter Sterling speak to this paradoxical impact on the body of the various adaptations it utilizes to cope with chronic stress

the term allostasis is used to emphasize the adaptive value of these compensatory adjustments, allostatic load to emphasize the price paid

but we are also reminded by those who encourage us to think in terms of allostasis, that a state of dynamic balance is rarely achieved by way of single-point tuning and local adjustment (as had been implied by Cannon) but more often involves body-wide regulatory changes and multiple adjustments through an elaborate system of checks and balances

finally, this broader-based allostatic model highlights the limitations of the homeostatic model, this latter a somewhat outdated, though time-honored, model that implies the existence of a single optimal steady internal state for the body and pays short shrift to the multitude of continuously shifting dynamic balances and multiple set points in the body resulting from constantly fluctuating environmental conditions

the holism of allostasis should complement, not replace, the reductionism of homeostasis

the allostatic model appreciates that the body is a complex adaptive system, a nonlinear dynamic system in a constant state of flux

the emergence of this new paradigm is therefore a manifestation of how the field of functional medicine has evolved, over the years, to accommodate ever increasing order and complexity...

parenthetically, psychoanalysis has also evolved, over the decades, from the more traditional Freudian model (a 1-person psychology focusing upon the internal dynamics of the individual patient) to the more contemporary intersubjective perspective (a 2-person psychology that focuses upon the relational dynamics between patient and therapist and upon negotiating at the intimate edge of that engagement). ...this newer systems approach complementing the older depth psychology

but returning now to the concept of adaptation as the body's attempt to restore its balance:

a few brief examples of single-point tuning mechanisms (with the understanding that these are but the tip of the iceberg) --

(1) When the thyroid is poisoned by environmental pollutants and becomes compromised in its functioning, one of the ways in which the body adapts is to redistribute the flow of blood, reducing blood supply to the skin and other nonessential areas in favor of the body's more essential systems -- thus the dry brittle hair (and tell-tale loss of the outer third of the eyebrows) and the dry scaly skin so characteristic of hypothyroidism.

(2) When the body is on overload from too much bad and not enough good and the HPA stress axis has become activated, the excess levels of cortisol so produced will be fed back to both the hypothalamus and the anterior pituitary to decrease the output of cortisol by the adrenals -- negative stabilizing feedback loops.

(3) When the body is exposed to endocrine disrupting, neurotoxic, and carcinogenic toxins, the body sequesters these lipophilic xenobiotics in its fat cells, the better to reduce oxidative stress by keeping these electron-scavenging free radicals out of circulation.

(4) When there is caloric restriction (whether intentional or not), the body slows its metabolism, slows the rate at which it burns its fuel, in order to conserve its energy.

(5) During intense exercise, when aerobic respiration has depleted the oxygen supply, the body resorts to anaerobic respiration, which, although not as efficient as aerobic respiration in producing ATP, does at least allow the cells continue functioning, even if suboptimally.

(6) When the internal environment becomes too acidic (perhaps secondary to the accumulation of metabolic waste products and toxicant pollutants), the body may compensate by leaching calcium from its bones in an effort to buffer the acidity. As with many of the body's compensatory mechanisms, the good news is that the acid-base balance will have been restored but the bad news is the potential for demineralization of the bones and osteoporosis.

and, finally, (7) when the body is sleep deprived, the body may activate the sympathetic nervous system in an attempt to compensate for its fatigue. The result: wired-but-tired, the plight of so many of us in these modern stressful times. Here the good news is the body's ability to continue functioning, but the bad news is the price paid in terms of the wear-and-tear on the system and the accelerated aging -- allostatic load, depletion of adaptation reserves.

with respect to the third and final stage of the stress response: Selye posited that if stress continues indefinitely, the body begins to break down

no longer able to resist or adapt, no longer able to maintain its internal constancy, the exhausted body, overloaded from the cumulative impact of too much bad and depleted from the cumulative impact of not enough good, will collapse, accompanied by progressive deterioration in structure and function

in fact, loss of the body's adaptability and resilience ushers in this final stage of maladaptation, dyshomeostasis, and chronic illness

### **Rea's Rain Barrel / Stark's Sandpile**

some 30 years ago, William Rea (in the tradition of Theron Randolph) introduced the model of a rain barrel gradually filling with drops of rain to represent visually the cumulative impact, over time, of stress on the body

the "capacity" of the barrel speaks to the body's capacity to tolerate environmental pollutants, overt clinical symptoms manifesting only once the "point of toxic accumulation" has been reached and the barrel has become filled to overflowing

but even prior to this "critical" point of overflow, as the total body load is increasing, there will be subclinical impairment

over the course of the past 10 to 15 years, Dr. Rea has been formulating some very compelling ideas about the theoretical underpinnings for the body's ability to reverse structural damage and functional impairment

inspired by Dr. Rea's state-of-the-art formulations, I have come up with a second simulation model, one intended to complement, not replace, the model of the rain barrel

but instead of a rain barrel to which drops of rain are being added, it is a sandpile to which grains of sand are being added

both models offer a visual representation of the cumulative impact of stress on a system over time, but whereas the focus of the rain barrel model is on one critical moment in time, the focus of the sandpile model is on a series of such moments over the course of time

in other words, the rain barrel model speaks to what happens when one specific critical threshold is reached; the sandpile model, on the other hand, speaks to what happens over and over again, each time a critical threshold is reached, namely, partial collapse of the sandpile, then its partial recovery, partial collapse, then its partial recovery, periodic cycles of disruption and repair, disruption and repair -- until such time as a final critical threshold is reached and the sandpile collapses entirely

in essence, the sandpile model offers a dramatic depiction of the reversibility of dysfunction and the resilience of an open system that is able to evolve by virtue of its receptivity to regulatory input from the environment

as grains of sand are being added, the continuously evolving sandpile will be able, at least for a while, to maintain its basic cone shape

in other words, environmental stress will prompt ongoing compensatory microadjustments in the sandpile's structure, enabling its integrity and balance to be preserved

eventually, however, the cumulative impact of the additional grains will be such that the sandpile will be no longer able to maintain its basic shape

a minor avalanche will be precipitated, and the sandpile's structure will become partially compromised

but then an extraordinary thing will happen: as more and more grains of sand are being added to the now partially collapsed sandpile, these additional stressors not only will provide the impetus for a partial reorganization of the sandpile's structure but will be the means by which such a reorganization will take place

the partially collapsed sandpile will reconstitute itself and "something new" will emerge, a regrouping, a new order, a realignment, characterized by metastability and precarious balance

adaptive reconstitution at this new allostatic set point will enable partial recovery of the sandpile's structural integrity and partial reversal of its underlying disorder and dysfunction

but this structural reorganization will only give the appearance of a "return to normal," because, in truth, the reorganization will have been accomplished at the expense of the system's overall stability and balance

nonetheless, iterative cycles of partial collapse, then partial recovery, partial collapse, and then partial adaptive reconstitution at ever higher levels of order and complexity will continue indefinitely, with the sandpile growing ever bigger, even as it is becoming ever more compromised in its structure

chaos theory speaks of these increasingly unstable and imbalanced sandpiles as being poised at the edge of chaos, the boundary between order and true chaos

so, as stress to the system continues (in the form of ongoing addition of these grains of sand), the sandpile will teeter at this edge of chaos -- until some indeterminate point in time, when a final critical threshold will be reached and a devastating, cataclysmic collapse of the entire sandpile will be triggered

a major avalanche, total breakdown, irretrievable structural damage to the sandpile

in summary and with respect to the body: recurrent cycles of partial collapse, partial adaptive reconstitution at ever newer allostatic set points and ever higher levels of order and complexity, until the critical moment in time when the cumulative impact of stress (in the form of both presence of bad and absence of good) will be such that the entire system (the matrix) collapses and chronic progressive irreversible deterioration in structure and function ensues, with irreparable disorder replacing the erstwhile order. Loss of adaptability, loss of resilience, loss of regulatory capacity, the onset of chronic illness, positive destabilizing feedback loops now replacing the negative ones that had once served to stabilize the system and maintain the constancy of its internal environment

but, as environmental medicine practitioners, we have at least a fighting chance to combat this tendency of all dissipative systems to run down over time, if we can but (1) appreciate the wisdom and the restitutive power of the matrix; (2) understand the complex interplay of its various components; (3) recognize the importance of restoring regulatory capacity and reinstating balance and harmony; (4) appreciate the fine line between input that is good or nontraumatic stress and prompts adaptive reconstitution (though at some cost to the system) and input that is bad or traumatic stress and prompts further decline; and (5) utilize the concept of the matrix to inform upstream intervention so as to forestall the development of downstream disorder, imbalance, and dysfunction (or, if it is too late for that, at least appreciate the potential for reversibility of disease if its underlying causes can be ferreted out)

but with this said, as is true for all nonlinear dynamic, complex adaptive systems (be they sandpiles or living systems) exposed to environmental stress (be it good or bad stress), one can never know in advance exactly when the little bangs (the minor avalanches) will occur nor exactly when the final big bang (the catastrophic major avalanche) will occur, only that such events will inevitably -- and necessarily -- come to pass

**THE WISDOM OF THE MATRIX: REGULATION, BALANCE, AND HARMONY**  
**Martha Stark, M.D.**

**25th Annual International Symposium  
on Man and His Environment in Health and Disease**

**June 7 - 10, 2007**

**REFERENCES**

- Bak P. **How Nature Works: The Science of Self-Organized Criticality**. Baden-Baden, Germany: Springer-Verlag Telos, 1996.
- Bak P, Tang C, Wiesenfeld K. Self-organized criticality: An explanation of 1/f noise. **Physical Review Letters** 1987;59(4).
- Becker R, Selden G. **The Body Electric: Electromagnetism and the Foundation of Life**. New York, NY: William Morrow, 1985.
- Buchanan M. **Ubiquity: Why Catastrophes Happen**. New York, NY: Three Rivers Press, 2000.
- Cannon WB. **The Wisdom of the Body**. New York, NY: WW Norton & Company, 1932.
- Darbre PD. Recorded quadrant incidence of female breast cancer in Great Britain suggests a disproportionate increase in the upper outer quadrant of the breast. **Anticancer Res** 2005 May-Jun;25(3c):2543-50.
- Emoto M. **The Hidden Messages in Water**. Hillsboro, OR: Beyond Words Publishing, 2004.
- Gerber, R. **Vibrational Medicine: The #1 Handbook of Subtle-Energy Therapies**. Rochester, VT: Bear & Co, 2001.
- Gladwell M. **Blink: The Power of Thinking Without Thinking**. New York, NY: Little, Brown and Company, 2005.
- Gleick J. **Chaos: Making a New Science**. New York, NY: Penguin, 1987.
- Goldberger AL, Rigney DR, West BJ. Chaos and fractals in human physiology. **Scientific American** Feb 1990:43-49.
- Heine H. **Homotoxicology and Ground Regulation System (GRS)**. Baden-Baden, Germany: Aurelia-Verlag, 2000.
- Ho MW. **The Rainbow and the Worm: The Physics of Organisms**. River Edge, NJ: World Scientific, 1998.
- Kauffman, S. **At Home in the Universe: The Search for the Laws of Self-Organization and Complexity**. New York, NY: Oxford University, 1995.
- Levine SA, Kidd PM. **Antioxidant Adaptation: Its Role in Free Radical Pathology**. Hayward, CA: Allergy Research Group, 1985.
- Mackowiak PA. Fever: blessing or curse? A unifying hypothesis. **Ann Intern Med** 1994;120:1037-40.
- Maes M, Smith RS. Fatty acids, cytokines, and major depression. **Biol Psychiatry** 1998;43(5):313-14.
- McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. **Ann N Y Acad Sci** 1998;840:33-44.

- McEwen BS. **The End of Stress As We Know It.** Washington, DC: National Academies Press, 2004.
- Oschman J. **Energy Medicine: The Scientific Basis.** New York, NY: Churchill Livingstone, 2000.
- Oschman J. **Energy Medicine in Therapeutics and Human Performance.** New York, NY: Butterworth Heinemann, 2003.
- Pert C. **Molecules of Emotion: Why You Feel the Way You Do.** New York, NY: Scribner, 1997.
- Pischinger A. **Matrix and Matrix Regulation: Basis for a Holistic Theory in Medicine.** Brussels, Belgium: Haug International, 1991.
- Randolph T, Moss R. **An Alternative Approach to Allergies: The New Field of Clinical Ecology Unravels the Environmental Causes of Mental and Physical Ills.** New York, NY: Harper Perennial, 1990.
- Rea W. **Four Volume Set on Chemical Sensitivity.** Boca Raton, Florida: Lewis Publishers, 1992, 1994, 1996, 1997.
- Sapolsky RM. **Why Zabrabs Don't Get Ulcers.** New York, NY: WH Freeman & Company, 1998.
- Schwartz G, Russek L. **The Living Energy Universe.** Charlottesville, VA: Hampton Roads Publishing Company, 1999.
- Selye H. **The Stress of Life.** New York, NY: McGraw-Hill Book Co., 1978.
- Stark M. **Modes of Therapeutic Action: Enhancement of Knowledge, Provision of Experience, and Engagement in Relationship.** Northvale, NJ: Jason Aronson, 1999.
- Sterling P. Principles of allostasis: Optimal design, predictive regulation, pathophysiology, and rational therapeutics. In: **Allostasis, Homeostasis, and the Costs of Physiological Adaptation** (ed. J Schulkin), pp. 17-64. New York, NY: Cambridge University Press, 2004.
- Sterling P, Eyer J. Allostasis: A new paradigm to explain arousal pathology. In: **Handbook of Life Stress, Cognition, and Health** (eds. S Fisher, J Reason). New York: John Wiley & Sons, 1988.
- Strogatz S. **Sync: How Order Emerges from Chaos in the Universe, Nature, and Daily Life.** New York, NY: Hyperion, 2003.
- Szent-Gyorgyi A. **Introduction to a Submolecular Biology.** New York, NY: Academic Press, 1960.
- Wallenstein G. **Mind, Stress, and Emotions.** Boston, MA: Commonwealth Press, 2003.
- Williams R. **Biochemical Individuality: The Basis for the Genetotropic Concept.** New Canaan, CT: Keats Publishing, 1956.
- Wright HW. **A More Excellent Way -- Be In Health.** Thomaston, GA: Pleasant Valley Publications, 2003.

## Objectives & Notes

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### Training:

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### SPEECH TITLE: “Attenuating Electromagnetic Radiation in Buildings”

At the end of this Presentation, the participant should be able to:

1. Understand the sources of EMF radiation in buildings.
2. Determine the potential health impacts of EMF radiation.
3. Methods to attenuate EMF exposure in buildings.

*The American Environmental Health Foundation and the University of North Texas Health Science Center is not responsible for the contents of this presentation. AEHF has not altered or modified the contents of the information provided by this speaker.*

## **Attenuating electromagnetic radiation in buildings.**

Robin, Ashton, Tang Lee\*, Karen Taylor  
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### **Goals and objectives**

To understand that along with advancements in technology and the rate at which technology inundates everyday lives, the exposure from electromagnetic field and low dose radio-frequencies, has greatly increased and can be expected to continue increasing.

To understand that with more exposure there is an expectation of increasing health risks and disease, affecting the total quality of human life

To recognize that common building materials provide minimal attenuation to these electromagnetic fields and radio frequencies. We must examine other methods of reducing our exposure intensity and exposure time. Limiting exposure intensity and duration is particularly important for young children, babies and pregnant women, and those suffering from electrical hypersensitivity.

There are several devices and materials purported to attenuate certain frequencies but with varying effectiveness, and may produce other problems. For example, variations caused by electrical interference called electro-smog can be filtered to provide a smoother current but it also creates other emissions. Special fabrics and coatings such as paints can also reduce RF transmission due to its base material and orientation.

This paper will examine the many types and intensities of RF that is in our midst and the potential health hazards. It will then summarize the many types of devices and encapsulation materials that are used to reduce RF exposures. Finally radio-frequency attenuation is difficult to achieve and complex requiring considerably more research.

### **1.0 Identifying human radio-frequency exposure**

Exposure to electromagnetic fields (EM) and radio-frequency comes from many sources. Low-level chronic exposure produces escalating and worsening of symptom presentation. Most exposure goes undetected and persons are not aware of total body exposure until symptoms become persistent and difficult to manage even though EMF sensitivity has been documented. (Rea, WJ, et al, 1991)

### **2.0 Early Symptoms of Chronic Exposure**

The early symptoms of chronic EM field and radio-frequency exposure are diverse. There is complaint of general malaise, a sense of fatigue/lethargy and “feeling tired”, with little energy. Complaint of headache is common so are complaints of light and heat sensitivities, especially with the teeth. (Beale I L, Pearce N E, Booth R J, 1997)

Often, inhalation and ingestion exposure produces symptoms similar to a cold or flu with inflammation in the ears, throat, and sinus. Chronic exposure effects the sensory perception of the ears and eyes causing blurred vision, ringing in the ears, TMJ, teeth grinding, and stress along the gums. In some instances, there can be swelling of the tongue. (Royal Society of Canada, 1999)

Low-level chronic exposure causes a loss of taste and smell, with a metallic taste often occurring. The tongue appears “coated” with red blisters appearing on the inner lip and cheek. There can also be reticular nodule formation in soft tissue of the gums and cheek. Hard lump-like formations also occur on the fingertips and knuckles.

Small children express symptoms of lethargy, colic, indigestion, and failure to thrive with poor weight gain, appetites, and mental interest.(Bonhomme-Faivre L, Marion S, Bezie Y, et al. 1998) Older children express poor attention, have difficulty sleeping or have excessive fatigue. Dark circles (purpura) appear around the eyes, loss of coordination, headaches, teeth sensitivity, rashes, night sweats, asthma, and difficulty learning, with delayed development. (Akerstedt T, Bengt A, Gianluca F, et al. 1999)

When chronic exposure is not mitigated and remediated, symptoms of exposure worsen and there is biological decline in major system organs such as the heart, kidney, lungs, thyroid, pineal, thalamus, renal gland, and pituitary. There is delay in puberty, menstruation, and fertility. In addition, thermo-physiological changes impede bone function and diminish connective tissue structure and elasticity. The spine is particularly affected with characteristic impediment occurring at the cervical spine C6, C7 and Thoracic T1 vertebra resulting in spine curvature, with shoulder (rotator cuff) deterioration.

The pelvis structure from the lumbar spin, sacrum, coccyx, hips and joints, particularly the right hip and joint are affected by softening, with dehydration of synovial membranes.

### **3.0 Medical Determination of Exposure**

Currently, there are no clinical or laboratory tests that can determine radio-frequency exposure. Multiple testing and studies have been performed to determine the thermophysiological consequences on human tissue exposed to both ionizing and non-ionizing radio frequencies, but there are no standard medical tests that can determine human exposure in clinical practice.

Clinical testing can be performed to determine the health of people exposed. These include: liver enzyme (elevated GGH levels), cholesterol HDL elevation, or blood test for chloroform levels.

Many cases of chronic exposure express heavy metal toxicity. Since demineralization and oxidation work on all elements and systems of human physiology, testing can be performed to determine elemental copper, zinc, and chromium levels. Iodine and thyroid testing is not a clear determinant to assessing exposure. Blood thyroid test can show thyroid gland function within normal/low range at moderate levels of exposure. Tests do not indicate iodine trapping capability and bioavailability of iodine present in the metabolism of T3 and T4 thyroid factors.

Irrespective of exposure route, dehydration and inflammation are characteristic presentations. In addition, the breakdown of  $\text{CHO}_3$  (hydrogen carbonate) causes over-saturation of oxygen with presentations of chest heaviness and difficulty breathing.

### **4.0 Attenuating Radio-frequency Exposure**

The advancement and deployment of technology makes attenuation of exposure a difficult and complex problem to solve.

Attenuation of EM fields and radio-frequency becomes an effort directed toward reducing and eliminating environmental exposure and the length of exposure, by reducing the amount of technology and appliances used. Attention needs to be given to reducing this EM field and radio-frequency pollution by filtering, reflection, redirection, neutralization, and wherever possible, elimination. This includes non-use of unnecessary medical tests such as x-ray, CAT scan, and ultrasound.

According to the United States Department of Energy through the Low Dose Radiation Research Program, “Radiation exposures associated with human activity are expected to be low dose and low dose-rate radiation from medical tests, waste clean up, terrorism events (e.g., dirty bombs) and environmental isolation of materials associated with nuclear weapons and nuclear power production. The major type of radiation exposures will be low Linear Energy Transfer (LET) ionizing radiation (primarily X- and gamma-radiation) from fission products.” (US Department of Energy: Low Dose Radiation Research Program)

### **5.0 Reducing Environmental Exposure**

Environmental pollution is a key contributor to the total amount of human exposure to EM fields and radio-frequency. Stringent industry regulations are necessary to reduce the emissions from all sources of industry, including telecommunications. Environmental protection measures must be initiated and implemented for our personal and corporate safety. Standards and guidelines greatly reducing man-made electromagnetic pollution, which inadvertently contributes to global warming through heat generation, must be established.

All industries must become inherently interested in the safe construction of all electrical equipment, residential appliances, and their applications and usage. Interest driving standards and guidelines must be on an International scale.

### ***5.1 Reducing Exposure Time in the Home and Workplace***

Individuals and families must become aware and educated about complications and concerns with the use of technology and products, both in the home and workplace. Conscious effort and education is needed to become aware of electrical products and their emissions. Further education is needed to inform the public about the use of chemicals and petroleum products in the home and environment.

Environmental Protection standards and guidelines need be developed to outline the routes and sources of contamination by radio-frequency. Reducing dependence on technology should be encouraged.

### ***5.2 Shielding and Reflection***

Total protection from EM fields and radio-frequency radiation in this day and age is a virtual impossibility. The most effective way to mitigate exposure to radio-frequencies is to reduce or cease the transmission of radio-frequencies, but this would severely curtail the efficiency of our communication systems, lifestyle, and transportation.

There are, however, many products commercially available purportedly for shielding and protection. Some of these products have application in the home and workplace, and made for products such as cellular telephones, computer monitors, televisions and stereo equipment; others are for automotive and personal wear.

Reducing dependence on EM field producing technology is also encouraged. The most significant effort we can make to attenuating radio frequency exposure is through education, continuing research, and implementing higher safety policies, guidelines and standards.

Education is required to teach the public the cost technology is having on human health and quality of life. Society needs to understand that the conveniences of modern technology become inconvenient to health and living. The best current way to attenuate the effects of radio frequency is to limit improper production from all sources. This includes the electrochemical pollution from electrical energy produced by burning coal and petroleum, from nuclear power plants, from factories producing so-called energy saving products like compact fluorescent bulbs; and refineries making processed foods that deplete the body's minerals, thereby reducing the natural biological electromagnetic field.

Investigation and research is underway by Professor Tang Lee (University of Calgary, Calgary, Canada) to determine the efficacy of shielding products in building design. In development are the design and application of products such as Mu-Metals and clays in the application of shielding radio-frequency coming into residential housing. Other strategies include indoor heating methods using solar and silicon dioxide heating protocols and convection that does not emit EMF or dangerous radio frequencies.

### ***5.3 Attenuation of EMF and radio frequencies is complex and difficult to achieve.***

The difficulty with attenuating radio-frequency lies in the principal of energy, that being, matter is neither created or destroyed it only transforms from one state to another. All matter is in a state of transformation. The process of oxidation-reduction affects matter as it moves through time.

Attenuating radio-frequency exposure then becomes a "constant capture" problem. As one filter captures let's say electrical energy, it transforms the accelerated charged particle into in a static one thereby creating heat. The filter meant to capture electrical frequency emission has created a problem by generating a magnetic frequency emission, heat.

Similarly, capturing radio frequency through shielding systems such as Mu-Metal sheets and containment architectures would result in heat generated that would need to be addressed through another filtering or shielding

system. Certain materials may attenuate some frequencies, but our society is exposed to radio frequencies from multiple sources

Radio-frequency capture through filters and shields needs to be dispersed. Each filter or shielding system claiming radio-frequency attenuation must be certified for heat dispensation guidelines and standards. Serious hazard could result from the implementation of a filter or shield where the heat dispensation is not adhered. Over time, filtered captures of electric or magnetic field can reach critical mass resulting in fire and explosion. Simply, the device for filtering, alerting, and alarming for EM field, including gasses, and radio frequency over heats.

Also, consideration must be given to the dangerous emissions from the capture or filter system and their effect and exposure potential.

Even if a shielding method is possible, it is only likely to be effective at certain frequencies and transparent to most of the others. The radio frequency spectrum spans from 3 Hz to 300 GHz that includes radio waves, microwaves, infrared, light, ultraviolet, x-rays and gamma rays (see the 88 page listing of the US frequency allocation chart).<sup>1</sup> Thus there is considerably more research required before we can achieve any effective radio frequency attenuation methods.

### **Conclusion**

The exposure from electromagnetic field and low dose radio-frequencies, both ionizing and non-ionizing, has greatly increased and can be expected to continue increasing. With more exposure there is an expectation of increasing health risks and disease, affecting the total quality of human life

Common building materials provide minimal attenuation of these electromagnetic field and radio frequencies. Commercial devices and materials purported to attenuate certain frequencies achieve varying degree of effectiveness, but can produce other problems.

It must be understood that EMF and radio frequency attenuation is difficult and complex requiring considerably more research.

### **References:**

United States Department of Energy; Office of Science, Low Dose Radiation Research Program, <http://www.lowdose.energy.gov/index.htm> [http://www.lowdose.energy.gov/about\\_main.htm](http://www.lowdose.energy.gov/about_main.htm)

Beale I L, Pearce N E, Booth R J "Association of Health Problems with 50-Hertz Magnetic Fields in Human Adults Living Near Power Transmission Lines." *Second World Congress for Electricity and Magnetism in Biology and Medicine*, Bologna Italy, June 1997

Akerstedt T, Bengt A, Gianluca F, et al. A 50-Hz electromagnetic fields impairs sleep. *J Sleep Res* 1999; 8:77 81.

Bonhomme-Faivre L, Marion S, Bezie Y, et al. Study of human neurovegetative and hematologic effects of environmental low frequency (50 Hz) electromagnetic fields produced by transformers. *Arch Environ Health* 1998; 53: 87-92.

Rea, WJ, Pan, Y, Yenyves, EJ, Sujisawa, N, Ross, GH, Electromagnetic Field Sensitivity, *Journal of Bioelectricity*, 10(1&2), 241-256, 1991.

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<sup>1</sup> <http://www.ntia.doc.gov/osmhome/Chp04Chart.pdf>

A Review of the Potential Health Risks of Radiofrequency Fields from Wireless Telecommunication Devices. An Expert Panel Report prepared at the request of the Royal Society of Canada for Health Canada, RSC.EPR 99-1, Ottawa, Canada, March 1999.

Reeben M, Arbatova J, Palgi J, Miettinen R, Halmekyto M, Alhonen, Riekkinen P Sr, Saarma M. (1996) Induced expression of neurotrophins in transgenic mice overexpressing ornithine decarboxylase and overproducing putrescine. *J. Neurosci. Res.* 45: 542-548.

## Objectives & Notes

### Riki Ott, Ph.D.

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### Training:

Current Job Description:	Independent researcher, lecturer, and author
Medical School/ University Attended	University of Washington (Ph.D.), University of South Carolina (Ms)
Internship:	Commercial fisherm'am in Cordova, Alaska, before, during and after the oil spill
Residency:	21 years in Cordova, Alaska, case study site for the sociology studies
Other Information: (including titles of books or articles you have recently written):	Sound Truth and Corporate Myth\$: The Legacy of the Exxon Valdez Oil Spill (Dragonfly sisters Press, 2005); Not One Drop: The True Story of Promises, Betrayal, and Courage in the Wake of the Exxon Valdez (Chelsea Green Publishing, 2007 fall); Resume posted on <a href="http://www.soundtruth.info">www.soundtruth.info</a>

### SPEECH TITLE: "Toxicological Pardigm Shifts after the Exxon Valdez Oil Spill"

At the end of this Presentation, the participant should be able to:

1. Understand the causes and mechanisms of lingering harm to wildlife from the oil spill
2. Understand the likely causes of lingering harm to farmer cleanup workers from the spill
3. Advocate increased protection from PAH exposure for workers, public health and the environment.

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# Toxicological Paradigm Shifts after the Exxon Valdez Oil Spill

Riki Ott, PhD

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## Abstract

Eighteen years ago, on March 24, 1989, the *Exxon Valdez* spilled between 11 to 30 million gallons oil in Prince William Sound, Alaska—and unleashed a cascade of events that would ultimately change scientists’ understanding of oil toxicity and lead to regulatory action to reduce human exposure to airborne oil particulates.

In 1989, the general consensus among scientists was that the oil spill’s effects on sealife would be short-term and deadly. They predicted:

- Birds and marine mammals that use the sea surface would suffer and die through hypothermia, drowning, and ingestion through preening and grooming.
- Fish and other sealife in the water column would take a toxic hit from the water soluble fraction (WSF), composed mostly of benzene—a single aromatic ring hydrocarbon and its derivatives. Death through narcosis would occur at concentrations in mere parts of oil per million parts of seawater—seemingly low levels.
- Oil stranded on beaches would rapidly “weather” or degrade, leaving an asphalt-like substrate that was “environmentally benign” or harmless to sealife. Wildlife would recover rapidly.

Scientists were partially right: the *Exxon Valdez* killed more wildlife than any other spill—before or since—in history. But the killing did not stop in 1989. Unrelated studies found, for example:

- Pink salmon eggs, developing in oiled beaches, died in ever increasing numbers in 1990, 1991, and 1992.
- Young sea otter pups, first weaned from their mothers, died by the score on oiled beaches.
- Over-wintering harlequin ducks died in higher numbers on oiled beaches.

In 1993, the Pacific herring stocks crashed totally unexpectedly in April, followed by the collapse of pink salmon stocks in August—for the second consecutive year. In response to public pressure, public-trust (non-industry funded) scientists conducted four seminal “ecosystem studies.” The standard 4-day bioassays of the 1970s typically exposed adult organisms of a single species to a range of concentrations of the WSF in seawater. In contrast, the ecosystem studies looked at generations of fish, birds, and mammals in relation to each other and the environment—and the studies focused on PAHs or polycyclic aromatic hydrocarbons—3-5 ring benzene compounds—the supposedly “environmentally benign” oil buried on beaches in the Sound.

By 1999, the 30-year old oil toxicity paradigm had shattered. Over 400 peer-reviewed papers were published during the early 2000s. The emerging understanding held that oil has both short-term direct effects on sealife—predicted by the old paradigm, as well as long-term direct, indirect, and delayed effects in the low parts per billion. This new paradigm shifts from a simplistic understanding, based largely on short-term toxicity tests with single species, to a more refined understanding based on a synthesis of ecosystem studies over time.

Under the new paradigm, oil causes short-term effects and long-term effects. PAHs are slow-acting poisons that kill sensitive eggs and embryos, stunt growth of juveniles, and sicken and cause functional sterility in adults. By compromising health of individuals, oil can cause population-level harm to species—and delayed and indirect effects in ecosystems. Further, oil is harmful to fish and wildlife at extraordinarily low levels—in the low parts per billion range or 1,000 times lower than levels thought to be “safe” for wildlife under the old paradigm.

The ecosystem studies proved to be a tipping point. During the 1980s and 1990s, a growing body of sophisticated medical studies had found similar persistent and deadly effects of PAHs on humans. In 1999, with evidence of long-term effects of PAHs in both humans and the ecosystem, the U.S. EPA added twenty-two PAHs to its list of persistent, bioaccumulative, and toxic pollutants.

In light of the documented long-term harm to wildlife from the EVOS and the accumulated evidence that PAHs are human health hazards, an investigation and separate pilot study were conducted in 2003 to determine if this spill and subsequent 1989 cleanup affected the health of cleanup workers. Results of the investigation found that workers were likely exposed to dangerous levels of dangerous chemicals, largely because OSHA safety standards and Exxon's worker safety program failed to adequately protect worker health. Exxon's injury and illness data show that 6,722 workers reported upper respiratory problems or over half of the total workforce. Exxon's air quality monitoring data show workers were likely overexposed to benzene, oil mist and PAH aerosols. Workers reported a suite of acute and chronic symptoms that matched the symptoms listed on the material safety data sheet for crude oil overexposure. However, Exxon did not conduct long-term health monitoring on its workers. Instead, Exxon declared the respiratory illnesses were "infections"—colds and flu—which are exempt from OSHA reporting requirements. A 2003 survey of former EVOS cleanup workers, conducted independently through the Yale Medical School, found significantly higher self-reported chronic symptoms, including respiratory problems, CNS problems, and chemical sensitivities. Efforts are underway to initiate a cohort epidemiology study on the former cleanup workers.

PAHs levels in air and water have risen to where they pose a serious threat to public health and the environment. Medical doctors have linked low levels of PAHs with respiratory problems, like asthma and bronchitis, cancer and other alterations in the DNA code, and aggravation of heart attacks and arrhythmias. Indoor air in heavy traffic areas or busy seaports can be just as deadly as outdoor air. People at risk include commuters, those who live, work and play in high traffic areas, kids on diesel school buses, and soccer moms—a lot of everyday people.

It's not just people. According to the National Research Council, the average PAH level in ten rivers in North America is within 10 percent of the range now known to sicken and kill wildlife and diminish entire populations of species.

Federal laws and regulations have not kept up with the new science on PAHs and do not adequately protect public health, workers, and the environment from all-too-often-common low levels of PAH exposure.

However, other actions may reduce exposure. For example, on March 29, 2007, the EPA finalized the Clean Air Fine Particle Implementation Rule to reduce levels of small particulates. This is also known as the "Soot Rule" as PAHs bind to the surface of microscopic dust particles.

Further, in 2006, shippers, unions, state officials and environmental groups took steps to reduce PAH pollution in the port of Los Angeles/Long Beach. This is the third largest port in the world and it has long been recognized as the single largest source of air pollution in the LA basin. Also, the EPA's Clean School Bus USA initiative, launched in 2003, aims to upgrade the nation's entire school bus fleet to low emission buses by 2010.

Instead of waiting for the cumbersome federal process to set adequate protective standards, environmental medical doctors—who have long recognized the "petrochemical problem"—could actively educate public health officials, the media, and the general public about the health effects from exposure to low levels of PAHs and opportunities for change.



## Objectives & Notes

### Jon Pangborn, Ph.D.

Date of talk: Friday, June 8, 2007, 1:00pm

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### Training:

Current Job Description:	President Bionostics, Inc.
Current Faculty Appointments:	Adjunct Prof. Nutritional Biochem. Union Institute, Cincinnati, OH
Medical School/ University Attended	Ph.D. from Syracuse University
Board Certifications:	Fellow, Am. Institute of Chemicals
Other Information: (including titles of books or articles you have recently written):	Autism: Effective Biomedical Treatments, J. Pangborn, and S. Beker; Co-Founder: Defeat Autism Now! (DAN!) Organization

### SPEECH TITLE: “Perpetuation of Inflammation by Epigenetic Influences”

At the end of this Presentation, the participant should be able to:

1. Know that inflammation/oxidant stress can alter gene expression which can alter metabolism
2. Appreciate the benefits and drawbacks to altered methionine metabolism as an adaptive response to inflammation.
3. Understand that certain nutritional deficits, toxic overloads and other factors can cause persistence of inflammation.

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PERPETUATION OF INFLAMMATION BY EPIGENETIC INFLUENCES  
ABSTRACT

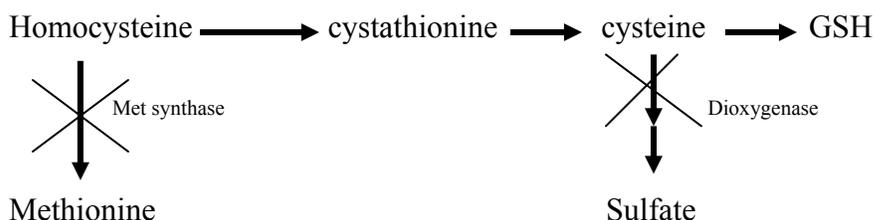
Jon B. Pangborn, Ph.D., F.A.I.C.

Epigenetics is the process of influencing tissue and cell functions via levels of gene expression rather than by an individual's (fixed) gene sequence. Some genes can change from being silent (methylated) to being expressed (unmethylated) in response to environmental exposures that challenge metabolic or immunologic capabilities. Methylation occurs on the cytosine part of cytosine-guanosine nucleotide pairs in DNA, and S-adenosylmethionine ("SAM") is the methyl donor.

Normally, epigenetic changes succeed in coping with environmental challenges. For example, this might be accomplished by changing enzyme activities, molecular transport, or upregulating detoxication and antioxidant capacities. Typically, epigenetic changes are eventually reversed to regain the original, stable metabolic and immunologic status.

Unfortunately, epigenetic demands for improving detoxication, antioxidant capacity, immune competence and specificity, etc. are not always met. One factor contributing to unfulfilled demands is variance or weakness (genetic or acquired) in metabolism. Those individuals with deficient P450 (e.g. CYP2D6) activity have difficulty detoxifying many xenobiotics and drugs. Those with both zinc deficiency and the adenosine deaminase type 2 variant (~4% of Caucasians) have lessened ability to process adenosine and are at risk for lessened immune response capabilities. Those with a cysteine or magnesium deficiency may not be able to muster up enough glutathione regardless of orders from headquarters. Those with folate or cobalamin supply or metabolism problems may even have dysregulated methylation. When such weakness or faults are present, cells may become deadlocked in an adaptive state of illness. Epigenetic changes insist on a metabolic or immunologic response that can't be met by the biochemistry or immune factors at hand. Without intervention that remedies the physiologic or metabolic faults, a perpetually inflammatory condition could result.

This can be illustrated by examining how methionine metabolism responds to oxidant stress and inflammation. Suppose an environmental exposure results in increased hydrogen peroxide,  $H_2O_2$ , in a cell, and suppose there isn't enough glutathione, GSH, on hand to reduce the  $H_2O_2$  to  $H_2O$ . One response is that of  $H_2O_2$  initiating a call for increased expression of ubiquitin ligase (by a gene on chromosome 15). Ubiquitin, which is in every cell in the body, finds the cystathionine enzyme system, binds to it and marks part of it for cleavage. The conjugation of ubiquitin and enzyme protein is catalyzed by the ligase expressed by a gene on 15q. Enzymatic cleavage occurs, which in this case makes processing of homocysteine to cysteine more rapid. At the same time, cysteine dioxygenase is similarly altered by the ubiquitin process, and possibly methionine synthase is also downregulated.



In essence, a change in genetic expression has then "fixed it" so that more GSH than before will be made from available homocysteine. The additional GSH reduces the excess  $H_2O_2$ , the oxidant stress is relieved, and the cell-gene systems are once more at peace with their environment.

But, what happens if something goes wrong with GSH synthesis? (These somethings include: deficient homocysteine, deficient vitamin B<sub>6</sub> as P5P-cystathionine enzyme cofactor, deficient magnesium – required for GSH synthesis.) Elevated adenosine, from whatever cause, increases homocysteine's precursor, inhibits methylation by SAM, and can be the reason behind deficient homocysteine, as can deficient methionine in the diet. If any of these circumstances are present, then the consequences of what the gene and ubiquitin have done include: decreased detoxication by sulfation, methylation, and taurine conjugation, including less taurocholate for biliary function. Reduced or deficient taurine means less quenching of OCl<sup>-</sup> in the cell, and the whole process hasn't relieved the GSH shortage. The cell is then in an adaptive state that perpetuates inflammation and is more handicapped with detoxication than before.

While anti-inflammatory medications may relieve this adaptive process, the real solution is to find and correct (if possible) the weakness or fault that didn't allow the body to heal itself.

## Bibliography

Epigenetics – a convenient article that's also online (Medscape) is: Diabetes 2005;54(7) 1899-1906 Am. Diabetes Assn.

How ubiquitin works in oxidative stress, see: Shang, Gong and Taylor, Am. Soc. Biochem. Molec. Biology 1997; 272(37) 23086-23093

For ubiquitin and cysteine dioxygenase, see Stipanuk, Hirshberger, et al. Am.J. Physiology – Endo and Metab 2004; 286; 439-448 or download from [ajpendo.physiology.org](http://ajpendo.physiology.org) on August 17, 2005.

The following PMID Abstracts and articles are pertinent.

PMID 12631446, Vitvitsky V, Mosharov E.. “Redox regulation of homocysteine-dependent glutathione synthesis” Redox Rep 2003;8(1):57-63

PMID 11041866, Mosharov E, Crandford MR, Banerjee R “The qualitatively important relationship between homocysteine metabolism and glutathione synthesis by the transsulfuration pathway and its regulation by redox changes” Biochemistry 2000 Oct 24;39(42):13005-11

PMID 15502870, Hondorp ER, Matthews RG “Oxidative stress inactivates cobalamin-independent methionine synthase in Escherichia coli PLoS Biol.2004 Nov;2(11);e335

PMID 15561573, Banerjee R, Zou CG “Redox regulation and reaction mechanism of human cystathionine beta-synthase: a PLP-dependent hemesensor protein” Arch Biochem Biophys 2005 Jan 1; 443(1):144-56

Olteanu H, Banerjee R “Redundancy in the pathway for redox regulation of mammalian methionine synthase” J Biol Chem 278(40) 38310-38314 Oct 3, 2003



## Objectives & Notes

**Paul R. Cheney, M.D., Ph.D.**

Date of talk: Friday, June 8, 2007, 1:30pm

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### Training:

Current Job Description:	Private Practice: The Cheney Clinic
Current Faculty Appointments:	None
Medical School/ University Attended	Emory University, M.D.; Duke University, Ph.D.
Internship:	Emory University affiliated hospitals
Residency:	Emory University affiliated hospitals
Board Certifications:	Internal Medicine
Other Information: (including titles of books or articles you have recently written):	Written and presented over 25 publications

### SPEECH TITLE: “Cardiac Diastolic Dysfunction in Chronic Fatigue Syndrome”

At the end of this Presentation, the participant should be able to:

1. The pathophysiology at Diastolic Dysfunction
2. The link between cellular energy deficiency and Diastolic Dysfunction
3. The implications at Diastolic Dysfunction in CFS

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## Abstract

### **Evidence for Diastolic Dysfunction in the Chronic Fatigue Syndrome enhanced by Tilt-Echocardiography: A study of ninety consecutive cases.**

Cheney PR and Lucki NC

**Background:** The Chronic Fatigue Syndrome (CFS) is a disabling disorder characterized by fatigue, which is prominently post-exertional, orthostatic intolerance and recently described low cardiac output (2003). The incidence of preserved ejection fraction heart failure in women is epidemic, unexplained and deadly (2006). The hypothesis that CFS patients have a disorder of cellular energy production might, if it affected the heart, present as energy dependent cardiac dysfunction such as diastolic dysfunction with preserved ejection fraction and might help explain the rise in diastolic heart failure which is now above 54% of all heart failure admissions (2006).

**Methods:** A preliminary age and sex-matched case-control study using 10 CFS cases and 10 normal controls was used to assess the sensitivity and specificity of a large set of echo parameters, especially diastolic parameters. A tilt-echo protocol using a standard tilt-table was used with echo interrogations in the supine, 30-degree and 70-degree head-up tilt with five minutes of equilibration before each echo interrogation. A Vivid-7 GE echo platform was used and the studies were performed by two alternating RDCS sonographers. Following the preliminary study, a larger study of ninety consecutive patients from 29 states and four countries were evaluated in a national referral practice for CFS. The average age was 48.9 yrs with a range from 14-74 years. Only 11 cases or 12% were over sixty. A history of hypertension was rare and only four were diabetic with only one type I diabetic. The average functional capacity was an AHA functional class III. Over 90% were disabled with CFS with many homebound and an average length of illness of 17 years (range 2-28 yrs). The ninety CFS patients were predominantly female (71%).

**Results:** The preliminary and on-going age and sex matched case-control study revealed that the most sensitive single echo parameter was the E/e' ratio or the ratio of the mitral in-flow E-velocity to the tissue doppler (TDI) e-velocity both measured at 70-degrees head-up tilt (90% above 8.0 for cases vs. 20% for controls). The most specific single echo parameter was left atrial (LA) collapse (cavitation) below 23 mm in diameter on head-up tilt (50% below 23 mm for cases vs. 0% for controls). Mitral in-flow E/A or TDI e'/a' ratios below 1.1 were 60% in the cases vs. 20% for controls in the case-control study.

Results on 70 consecutive patients with all three types of in-flow velocities measured revealed that 50 of 70 or 71% had either mitral in-flow E/A or TDI e'/a' ratios  $\leq 1.1$  in the supine position. Pulmonary vein D/S reversal ( $<1.1$ ) in the supine position was observed in 67 of 70 or 96% of cases. LA cavitation ( $\leq 25$ mm) at 70 degrees head-up tilt was observed in 48 of 90 or 53% but was observed in 17/20 or 85% of those under 40 years of age. In contrast to the age sensitivity of mitral in-flow velocity reversal (older cases) and LA cavitation (younger cases), E/e' ratios above 8.0 at 70-degree head-up tilt and pulmonary vein D/S reversal ( $<1.1$ ) in the supine position were both far less age sensitive.

**Conclusions:** Chronic fatigue syndrome patients exhibit evidence of diastolic dysfunction at a level well above that reported for control populations of the same age. Energy dependent diastolic dysfunction would appear to be a hallmark of CFS and supports the hypothesis that CFS is a syndrome of cellular energy deficiency. Tilt-echo protocols provide an amplification of often masked diastolic dysfunction (ie pseudonormalization) which increases sensitivity with little loss in specificity and reveals previously unreported data on a patient population known to be sensitive to head-up tilt.

## Objectives & Notes

**Mohamed B. Abou-Donia, Ph.D.**

Date of talk: Friday, June 8, 2007, 2:00pm

Duke University Medical Center  
Laboratory of Neurotoxicology  
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Email: donia@acpub.duke.edu

### Training:

Current Job Description:	Research and Teaching
Current Faculty Appointments:	Professor of Pharmacology and Cancer Biology, Duke University Medical Center
Medical School/ University Attended	University of California, Berkeley
Board Certifications:	American Board of Toxicology, Academy of Toxicological Sciences
Other Information: (including titles of books or articles you have recently written):	Editor of the book "Neurotoxicology," published more than 320 papers.

### SPEECH TITLE: "Organophosphorus Compound-Induced Neurotoxicity"

At the end of this Presentation, the participant should be able to:

- 1.
- 2.
- 3.

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**Organophosphorus Compound-Induced Neurotoxicity. Mohamed B. Abou-Donia, Department of Pharmacology and Cancer Biology, Duke University Medical Center, Durham, North Carolina 27710**

Organophosphorus compounds are derivatives of phosphoric, phosphorus or phosphonic and phosphinic acids. Organophosphorus compounds are an economically important chemical class with numerous uses as pesticides, industrial fluids, flame retardants, therapeutics, and nerve gas agents. The action of organophosphorus compounds is related to their phosphorylating abilities of target proteins. This is dependent on the electrophilicity (positive character) of the phosphorus atom, which is determined by its substituent groups. Steric factors of substituents also play a major role in determining the biological activity of these chemicals. Lipid solubility is important, because it enhances their ability to cross biological membranes and the blood brain barrier (BBB), leading to increased biological activity. Biologically, organophosphorus compounds are neurotoxic, both to humans and other animals. Synthetic organophosphorus compounds are tailor-made to inhibit acetylcholinesterase (AChE), an enzyme essential for life in humans and other animal species. During World War II, organophosphorus compounds were developed primarily as agricultural insecticides and later as chemical warfare agents. These chemicals produce three distinct neurotoxic actions: 1) cholinergic neurotoxicity resulting from inhibition of AChE; 2) organophosphorus ester-induced delayed neurotoxicity (OPIDN) resulting in axonal and myelin degeneration of the central and peripheral nervous systems; and 3) organophosphorus ester-induced chronic neurotoxicity (OPICN) leading to neurobehavioral alterations and neuropathological changes of the nervous system. Studies in the past half-century have documented the development of chronic neurotoxicity symptoms in humans, resulting from acute exposure to high doses that cause acute cholinergic toxicity, and from long-term, low-level, sub-clinical doses of these chemicals. Furthermore, epidemiological studies have reported an association between exposure to organophosphate insecticides and the development of Parkinson's Disease. Other studies have implicated exposure of the Gulf War veterans to low-level exposure to the organophosphorus nerve agent sarin, and developing Amyotrophic Lateral Sclerosis (ALS).

## Objectives & Notes

**Richard Weisler, M.D.**

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Date of talk: Friday, June 8, 2007, 2:30pm

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### Training:

Current Job Description:	Principal Clinical Investigator in trials, private practice, teaching, and consulting with industry and governmental agencies
Current Faculty Appointments:	Adjunct Professor of Psychiatry at University of North Carolina at Chapel Hill and Adjunct Associate Professor of Psychiatry at Duke University Medical Center
Medical School/ University Attended	University of North Carolina at Chapel Hill
Internship:	Medicine-University of North Carolina at Chapel Hill and Dorothea Dix Hospital
Residency:	Psychiatry-University of North Carolina at Chapel Hill/North Carolina Memorial Hospital
Board Certifications:	American Board of Psychiatry and Neurology
Other Information: (including titles of books or articles you have recently written):	Over 20 articles published.

### SPEECH TITLE: "Elevated Rates of Suicides, Cancers and Respiratory Related Deaths in a Downwind Neighborhood: Why?"

At the end of this Presentation, the participant should be able to:

1. Explore the possible relationship between a NC neighborhood's proximity to past potentially hazardous chemical air emissions and groundwater contamination from nearby industrial facilities, and the 3 fold statistically significant increased rate of suicides and ATSDR/CDC confirmed statistically significant 6 fold increased rate of primary brain cancers, 5 fold increased rate of blood cancers, and 32% increased rate for all cancers. Further explore the possible role of potential past hazardous chemical exposures in the marked elevations in rates of pancreatic, colon, and lung cancers that were only documented by a more complete data set than the NC Central Cancer Registry provided to ATSDR/CDC at the time of their 2/2006 review of 1990-2000 neighborhood cancer incidence.
2. Appreciate some of the challenges and joys of doing community based environmental health research. Discuss the environmentally impacted community's interactions with us, and their reactions to the contamination and health research. Describe some interactions with health, environmental, and transportation agencies at both the state and federal level as well as politicians, city, and county officials.
3. Discuss the needs for potential remedies and future environmental and health studies, possible future health monitoring needs of area residents, workers and Army Reservist past and present, and the future environmental risk reduction strategies for this Salisbury neighborhood. Begin to explore the potential for similar adverse health problems in other communities near related industrial facilities and hazardous chemical exposure prevention strategies.

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Title: Elevated Cancer Rates and Suicides in Downwind Residential Neighborhoods and Chemical Releases from Nearby Industries: A Potential Link?

Authors: Weisler, RH, Davidson, JRT, Crosby, L, Zeller, L, Taylor-Guevara, H, Singleton, SS, Fiffer, MR, Tsougas, SM, Turner, LM

Submitted by: Dr. Richard Weisler, Blue Ridge Environmental Defense League (BREDL), Adjunct Professor of Psychiatry at University of North Carolina at Chapel Hill, Adjunct Associate Professor, Duke University Medical Center, NC Bipolar Depression Support Alliance (NC BDSA)

Affiliations: Duke University Medical Center Department of Psychiatry (Davidson), BREDL (Crosby and Zeller), Clean Water for NC (Taylor-Guevara), NC BDSA (Singleton), Duke University Nicholas School of the Environment and BREDL (Fiffer, Tsougas, Turner)

Two Salisbury, NC residential neighborhoods (entire Census Tract Block Groups 513031, Milford Hills, and 513032, Meadowbrook totaling about 1561 residents) exhibited statistically significant elevated rates in primary brain cancers, lymphomas, and all cancers from 1990-2000. Pancreatic cancers also appear to be significantly increased as were lung and colon cancers. The authors previously reported in their retrospective health study a three-fold increase in death by suicide and about 6 fold increase in primary brain cancers in the same two Salisbury largely middle-class neighborhoods, situated immediately downwind from two asphalt facilities, a chlorinated solvent contaminated former NC Department of Transportation asphalt testing laboratory, and two petroleum contaminated remediation sites. In this BREDL community study, using data from death certificates, calls by area families, and community volunteer reports, the authors found significantly elevated brain and blood cancer rates. From 1988-2000, Milford Hills and Meadowbrook experienced 8 primary brain cancers, representing over six times the brain cancer risk for the state of North Carolina as a whole. While these two neighborhoods represent about only 1.2% of the Rowan County population, they appear to represent about 7 % of the county pancreatic cancer cases from 1996-2004. The Rowan County Public Health Director independently verified the brain, colon, lung, and pancreatic cancers cases as well as suicides. Based on past NC Cancer Registry statements, our findings in these two neighborhoods, coupled with the recent Agency for Toxic Substances and Disease Registry (ATSDR)/Centers for Disease Control and Prevention (CDC) confirmatory review, should represent a significant area for concern in North Carolina. The NC State Center for Health Statistics and NC Cancer Registry previously reported a “lower than normal rate of lymphomas” and only “a slight elevation in brain cancers” in the same Salisbury neighborhood area during this time-frame, and failed to appreciate the statistically significant increased rate represented by the six deaths by suicide from 1994-2003. The NC Central Cancer Registry appear to have missed locating or capturing significant numbers of colon and lung cancers cases based on our verified death certificate data comparisons with the registry tabulations of cancer incidence for the census tract block groups.

Some of the elevated cancer rates that we found in Milford Hills and Meadowbrook have been confirmed by a recently released ATSDR study of the roughly 1430 residents of census tract block groups 513031 and 513032 who lived within their predefined 1 mile radius of the Salisbury asphalt facilities. The ATSDR/CDC analysis of NC Cancer Registry data found statistically significantly elevated rates of brain cancers, lymphoma, and all cancers overall in the two census tract block groups, compared to Rowan County as a whole from 1990-2000 (Table 1). ATSDR noted that the elevated cancer rates were not found in the larger one-mile radius, which we feel is potentially due to past exposure dilution effects. Block groups 513031 and 513032 together represent slightly more than the northeast quadrant of the one mile radius ATSDR studied. The cancer and suicide case locations BREDL identified suggest that downwind proximity to potentially hazardous emissions may represent a surrogate exposure dose-response relationship.

Lymphomas, brain, lung, and pancreatic cancers have been associated with exposure to benzene, polyaromatic hydrocarbons, and/or asphalt fumes in occupational studies. Occupational studies have also reported elevated suicide rates in asphalt workers and in workers exposed to sulfur compounds like carbon disulfide. Chlorinated solvents have also been associated with brain and/or other cancers. All of these known or suspected carcinogens and toxins along with other chemicals have been released immediately upwind of these Salisbury neighborhoods in unknown quantities. Hydrogen sulfide, benzene, asphalt fumes, polyaromatic hydrocarbons, particulates, and chlorinated

solvents, were released in unknown quantities by the liquid asphalt terminal and the hot mix asphalt plant. The liquid asphalt terminal for many manufactured cutback asphalt for decades made with a recipe of up to 45% solvents(naphtha, benzene, nitrobenzene, or other petroleum solvents) mixed with 325 °F liquid asphalt without any or adequate environmental controls. Chlorinated solvents were released in unknown quantities by volatilization during both the decades of spraying of solvent-contaminated water, and from solvent-contaminated holding ponds at the former asphalt testing laboratory and the liquid asphalt tank farm. The former petroleum distribution bulk tank farm released benzene and other neurotoxic petroleum solvents into the air during their earlier operation, and during their untreated aggressive fluid-vapor and solid-vapor extraction cleanup of groundwater contamination.

Given the large amount of uncertainty in other emissions and the lack of retrospective air modeling, the authors suggest that hydrogen sulfide can serve as a surrogate marker for other air toxins in Salisbury. The NC Department of Environment and Natural Resources (NCDENR) conducted an air modeling study in 2001 on benzene and hydrogen sulfide releases in Salisbury. The study estimated the average maximum benzene level at 123,333% of the NC acceptable ambient air level on an annual basis primarily at the former petroleum tank farm remediation site. Hydrogen sulfide levels in a large part of the same affected neighborhoods were modeled at 215 parts per billion (ppb), with averages at 860 ppb in a few residences, compared to the North Carolina health-based 24-hour standard of 86.2 ppb. NCDENR's 2002 air monitoring study which ATSDR used for a review underestimates past hazardous exposures for residents and workers, as it came only after carbon filters were installed at the asphalt terminal, asphalt storage tank exhaust fans were removed, manufacturing of cutback asphalt (up to 45% naphtha or other petroleum solvents mixed with liquid asphalt) at the liquid asphalt terminal was discontinued, spraying of chlorinated solvent contaminated water was halted, and the former petroleum tank farm's untreated air-sparging and soil vapor extraction remediation ceased. Many of these changes occurred just prior to the NCDENR air monitoring study that followed our complaints and urging, and we believe these filters and process changes significantly reduced future public health exposures to hazardous chemical compounds.

Since 1999 the City of Salisbury has received over 600 official odor and respiratory problem complaints from residents of Census tract block group 513031, resulting in 29 citations to the asphalt facilities. The frequency of air complaints diminished after the 2001 and 2002 installation of carbon filters at the liquid asphalt terminal and the removal of the liquid asphalt storage tank exhaust fans. Retrospective air-modeling has been hampered by the asphalt terminal as well as NCDENR's refusal to heed written and oral requests by the City of Salisbury, Rowan County Health Department, NC Department of Health and Human Services (NCDHHS), citizens, and ATSDR/CDC for a more complete characterization of liquid asphalt stack emissions. NCDENR and the polluting companies/NC Department of Transportation have also refused to complete a retrospective groundwater modeling study to help estimate past solvent exposures for residents despite a formal request being made in November 2002 by the NC Department of Health and Human Services Occupational and Environmental Epidemiology Branch. Adequate determination of the horizontal and vertical extent of area groundwater contamination and safe clean-up of the contaminated soil and groundwater has yet to be completed. The ATSDR's recommendation in 2002 for carbon filter installation to control emissions from about 1300 ninety ton liquid asphalt railroad tank cars during unloading has not occurred, and NCDENR has ignored similar requests to require additional emission controls from residents, the City of Salisbury, and the Rowan County Health Department.

Conclusion: Further study of the neighborhood cancers and suicide cases are needed to better characterize other potential risk factors and the length of residence. Better characterization of hazardous emissions and retrospective air and groundwater contamination is needed to better estimate past and present chemical and particulate exposures, and investigate possible etiologies for these adverse health outcomes. Area residents both past and present should be educated about cancer and suicide prevention approaches. Additional health monitoring for some residents may be advisable, Area health providers should be informed about the findings. Additional efforts to further reduce the possibility of hazardous chemical exposures for area residents and workers should be made, Safely remediation of the soil and groundwater contamination in the area should be completed in a timely fashion. The etiologies for these neighborhood residents statistically significant serious adverse health findings needs further systematic exploration.

Seeing statistically significant elevations of brain cancer, lymphomas, all cancers, and it now appears pancreatic, colon, and lung cancers in the same residential neighborhoods along with the first literature report of adult male suicide cluster raises significant concerns about the past impact of potentially hazardous environmental exposures on area residents.

Table 1. Age-Adjusted Standardized Cancer Incidence Rates in the One-Mile Area Surrounding Industrial Facilities on Jake Alexander Boulevard, Salisbury, North Carolina and in Two Specific Census Block Groups, 1990-2000

Site	SIR (1 mile)	95% Confidence Interval	SIR (Block Groups 513.031 & 513.032)	95% Confidence Interval
Brain	1.70	(0.45, 4.33)	6.16	(1.66, 15.78)*
Lymphoma	1.37	(0.55, 2.83)	4.94	(1.80, 10.76)*
All Cancers	0.77	(0.62, 0.93)*	1.32	(1.07, 1.60)*

\* Statistically significant results ATSDR/ CDC February 2006 Final Report for their Salisbury, NC cancer study  
Henderson, A, Foster, S

The authors wish to thank Alden Henderson, PhD, MPH, Pam Tucker, MD, Carl Blair, and Stephanie Foster, MPH with ATSDR/CDC, Leonard Wood, MPH the Rowan County Public Health Director, and Steve Wing, PhD with the UNC School of Public Health for their efforts and technical advice in this project.

No direct funding was received by any of the authors for this work. Melissa Fiffer, Lisa Turner, and Stacy Tsougas all were given Duke University Nicholas School of Environment Stanback Internships to support their undergraduate work with the non-profit Blue Ridge Environmental Defense League. Melissa Fiffer did some of this work during her undergraduate independent study with Dr. Weisler, and she currently has a Stanback Internship to support further efforts on this project as she pursues a Masters at the Duke School of Environment. Dr. Weisler began this work after his mother who lived in the neighborhood became ill and later died from lung cancer, while living in the impacted neighborhoods. His mother's estate is pursuing legal recourse at this time. In part to reduce potential conflicts, the cancer and suicide cases and health findings in these Salisbury neighborhoods were independently confirmed by non-involved parties.

## Objectives & Notes

**Divakar Dalela, M.D.**

Date of talk: Friday, June 8, 2007, 3:45pm

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Medical University  
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India

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### Training:

Current Job Description:	Full time Consultant Urologist & Training Postgraduates for M.Ch. Urology
Current Faculty Appointments:	Professor & Head, Dept. of Urology, K.G. Medical University, Lucknow.
Medical School/ University Attended	K.G. Medical College, Lucknow -1979-1987, SGPGIMS, Lucknow – 1988-1992
Internship:	K.D. Medical College, Lucknow – 1985-86
Residency:	S.R. Surgery, K.G. Medical College, Lucknow – 1986-1988, S.R. Urology, SGPGIMS -1989-1992
Board Certifications:	M.B.B.S.; M.S. (Surgery); M.Ch. (Urology)
Other Information: (including titles of books or articles you have recently written):	Publication details available on website: kgmcindia.edu (Urology Department); drdivakardalela.com

### SPEECH TITLE: “Benign Prostatic Hyperplasia - Can Dietary Modulations Prevent it?”

At the end of this Presentation, the participant should be able to:

1. Learn epidemiologic basis for nutritional link
2. Understand molecular mechanisms of dietary influences
3. Gain knowledge about constituents of diet implicated currently.

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## Benign Prostatic Hyperplasia – Can Dietary Modulation Prevent it?

Divakar Dalela<sup>1</sup>, Manish Ahuja<sup>2</sup>

<sup>1</sup>Professor & Head

<sup>2</sup>Senior Resident

Department of Urology, King George's Medical University, Lucknow (India)

### **Abstract**

Benign prostatic hyperplasia (BPH) represents a non cancerous enlargement of the prostate gland. This is considered to be a part of natural ageing process. The underlying etiology behind this disease is the continuous growth of cells under the influence of androgens, especially testosterone. The enlarged prostate obstructs the urinary flow and produces symptoms. Although effective treatment of BPH is available, prevention appears to be an attractive option. Diet seems to have a very important role in prostate cell growth as evident by the yawning gap in incidence of BPH between the East and the West. Vegetarian diet containing phytoestrogens, notably flavonoids and lignans, have been seen to prevent cell multiplication in prostate. Soy, a rich source of genistein, has also been found to have anti-proliferative activity. Phytoestrogens act by decreasing availability of testosterone to the prostate cells and by blocking the enzyme 5-alpha-reductase that converts testosterone to DHT, A MUCH MORE ACTIVE FORM. Lycopene is the second major player in prevention of BPH. Possibly acts by inhibition of growth factors (IGF-1 and IL-6) and androgenic signaling. Omega-3 fatty acids are present in high amount in Flaxseed. Its consumption has also been found to lower incidence of BPH. Green tea intake is associated with lower risk. Nonvegetarian diet (beef, poultry, eggs, etc) is associated with increased incidence of BPH. Role of alcohol, coffee, milk, zinc and vitamin D is under evaluation.

## Objectives & Notes

**Ron Overberg, Ph.D., C.C.N., R.D.**

Date of talk: Friday, June 7, 2007, 4:15pm

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### Training:

Current Job Description:	Nutritionist at Environmental Health Center - Dallas and at Nutri Wellness
Medical School/ University Attended	University of Texas at Dallas (Ph.D.)
Internship:	Texas Women's University (R.D.)
Board Certifications:	Board Certified Clinical Nutritionist by C.N.C.B.
Other Information: (including titles of books or articles you have recently written):	2005 Revision of the "Rotational Bon Appetit Cookbook;" 2005 "EHC-D Food Rotation and Supplement Guide" Handout, Registered Dietitian, Texas Women's University in Denton

### SPEECH TITLE: "Organic Acid Analysis and Chemical Sensitivity"

At the end of this Presentation, the participant should be able to:

1. Understand the benefit of doing an organic acid analysis of a chemically sensitive patient.
2. Utilize organic acid analysis to determine the adequacy of a current nutritional support program and the need for additional supplementation
3. To assess nutrition related dysfunctions in energy production, detoxification, and other pathways in chemical sensitive patients.

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## Organic Acid Analysis and Chemical Sensitivity

Ron Overberg, Ph.D, C.C.N, R.D.  
Environmental Health Center-Dallas

### Abstract:

An analysis is presented on data collected from 472 urine organic acid analysis panels done over a 5 year span at the Environmental Health Center in Dallas. The patient population included 126 men and 346 women, ages 13 to 84, receiving treatment of chemical sensitivities and chronic disease. The data was compared to the ninety-fifth percentile reference values provided by the laboratory (Metametrix Laboratories).

Urine organic acid testing provides a convenient non-invasive way to measure functional markers for: Vitamin or mineral deficiencies, energy production dysfunction, need for anti-oxidants, toxic exposure, detoxification deficiencies, neuroendocrine function and intestinal bacterial and yeast overgrowth.

The metabolism of fats, carbohydrates and amino acids to acetyl-CoA is the first step in energy production. It is analyzed by looking at the representative fat metabolism analytes: Adipate, 15.6%, Suberate, 9.1% and Ethylmalonate 14.1%. They were all well above the expected 95 %, indicating a need for carnitine and riboflavin. The analytes for carbohydrate metabolism (Pyruvate, 18.2%, Lactate, 26% and  $\beta$ -Hydroxybutyrate, 12.7%) were also elevated, indicating a need for thiamin, riboflavin, niacin, panthothenic acid, Lipoic acid, CoQ10, chromium and vanadium. Protein metabolism is analyzed by looking for a build up of the alpha-ketoacids from the branch chain amino acids: Valine, Leucine and Isoleucine (13.7%, 2.1%. and 12.7%, respectively). These indicate a need for thiamin, riboflavin, niacin, panthothenic acid, and Lipoic acid.

Acetyl-CoA then enters the Citric Acid Cycle where it combines with oxaloacetate to become citrate. Citrate is the first of a series of Citric Acid Cycle intermediates that can be measured. The following values are the sum of the high and low values outside the reference ranges: Citrate 26.7%, Cis-Aconitate 17.4%, Isocitrate 28.5%, Alpha-ketoglutarate 22% Succinate 21.6%, Fumarate 18.6%, and Malate 18.8%. Each analyte value can indicate the need for various B-vitamins, minerals and CoQ10.

In the third step of energy production NADH and FADH<sub>2</sub> enter the Electron Transport pathway to ultimately produce water and energy. Hydroxymethylglutarate, the precursor of CoQ10, is a marker for blockage of the biosynthetic pathway. The results are significantly higher (19.2%) than the expected reference value. This indicates the need for CoQ10 supplementation or that the patient was taking a statin class drug that blocks formation both ubiquinone and cholesterol.

Methylation markers: 13.7% of the patients had an elevated Methylmalonate, a functional marker for B12 need. 17.2% had an elevated Formiminoglutamate, indicating the need for more Folic acid.

Biotin and B6: The need for Biotin is determined by the level of  $\beta$ -hydroxyisovalerate (12.3%) and an elevated Xanthurenate (19.4%) is one of the markers indicating a need for pyridoxine.

Neurological markers: Vanilmandelate (20.1%) and Homovanillate ((10.1%) are the metabolites of Epinephrine and Norepinephrine and of Dopamine respectively in indicate a need d for additional Tyrosine. 5-Hydroxyindoleacetate (26.8%) is the breakdown product of serotonin and indicates that this population needs more L-tryptophan. Products of interferon gamma-stimulated macrophages, astrocytes and microglial cells, Kynurenate (17.2%) and Quinolate (16.7%) both have effects on the central nervous system. The first antagonistic and the second is agonistic for the NMDA receptor activation. An elevated Kynurenate indicates a need for more pyridoxine and an elevated Quinolate indicates a need for more magnesium and anti-oxidants (plus investigation of the origin of inflammation).

Oxidative damage and anti-oxidant markers: p-Hydroxyphenyllactate at 18.6 % indicates a significantly higher need for Vitamin C. 8-Hydroxy-2-deoxyguanosine is a product of oxidative damage to DNA, at 18.9% indicates a need for increased anti-oxidant support

Detoxification markers: 2-Methylhippurate is a by-product from the detoxification of Xylene, and is high for 16.7% of this patient population. Orotate, a marker for the ammonia detoxification capacity of the urea cycle is elevated at 12.7%. Glucarate, a marker for overall hepatic detoxification is 25.2%. Alpha-Hydroxybutyrate levels at 10.8% indicate that the rate of glutathione production is higher than average. Pyroglutamate (9.7%) is generated when Glutathione is sacrificed to salvage amino acids. Sulfate is an indicator of the amount of sulfur available for detoxification and a marker of total body sulfur compound (esp. glutathione) status. 9.7% of the patients had low body stores and 5.3% had high sulfur excretion consistent with up-regulated detoxification.

Bacterial and yeast overgrowth markers. Benzoate 12.5%, Hippurate 14.6%, Phenylacetate 25.2, Phenylpropionate 4.7%, p-Hydroxybenzoate 16.3, p-Hydroxyphenylacetate 10.4%, Indican 19.1%, and Tricarballoylate 9.3% are used for general bacteria. D-lactate 7.7% is for a marker of general bacterial overgrowth that, in some patients, can be antecedent to a special situation of toxemia from dietary carbohydrate-induced overgrowth of *L. acidophilus*. 3,4-Dihydroxyphenylpropionate 13.1% is used for the clostridia type bacteria. D-Arabinitol 7.7% is used as a yeast marker.

In conclusion: Urine Organic analysis gives clinically significant data to guide the treatment of chemically sensitive and chronic disease patients.

#### References:

1. Bralley, J.A., Lord, R.S. p175-208, "Laboratory Evaluations in Molecular Medicine" Institute for Advances in Molecular Medicine ISBN 0-9673949-1-0
2. Guyton, A. C., Hall, J.E., Textbook of Medical Physiology, 9<sup>th</sup> ed. W.B. Saunders Co. ISBN 0-7216-5944-6
3. Lehninger, A. L., Biochemistry 2nd ed. Worth Publishers, Inc. ISBN 0-8790-047-9



## Objectives & Notes

**Stephanie McCarter, M.D.**

Date of talk: Friday, June 8, 2007, 4:45pm

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Point, TX 75472

### Training:

Medical School/ University Attended	Indiana University School of Medicine
Internship:	Indiana University School of Medicine
Residency:	Indiana University School of Medicine
Board Certifications:	Internal Medicine

### SPEECH TITLE: **“Building Considerations for a Low EMF Home”**

At the end of this Presentation, the participant should be able to:

1. Identify Electromagnetic Field (EMF) sensitivity in patients and how to treat such patients.
2. To understand the differences between electrical and magnetic fields found in the home and the environment.
3. To understand sources of EMF fields in the home and surrounding environment and how to minimize these fields in home construction.

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## Building Considerations for a Low EMF Home

By  
Stephanie McCarter, M.D.

Chemical Sensitivity is based on histochemical and biochemical disturbances of function which includes electrolability and oscillating capacity of the ground regulating system (GRS). Due to this capacity of the GRS structures, they are sensitive to electrostatic and electromagnetic environmental influences such as 1) static fields, 2) air electrical charges and 3) electromagnetic impulse fields.

The lability of the GRS to electromagnetism is manifested in chemically sensitive (CS) patients. CS patients often have specific frequency sensitivities from TV and radio frequencies, to cell phone and computer frequencies.

To build a low EMF home in this modern world, many factors must be considered. Location is of the utmost importance and often difficult to optimize given the increasing amount of electromagnetic radiation present worldwide. Cell phone towers, TV and radio transmitters, large power lines and airplane flight paths must be avoided. Underground sources must be considered as well, as underground water and high mineral content of soil have been known to generate magnetic fields.

Electric and magnetic fields differ distinctly and must be lowered in different ways. Magnetic fields are loop lines around current (electron) flow and difficult to shield. Magnetic fields are mitigated by controlling flow paths and shutting off appliances. Electric fields are produced by electron separation (voltage) and connect as lines between voltage differences which can be shielded. They are mitigated by grounding and shutting off circuits.

Lowering magnetic fields involves specific guidelines for electrical wiring installation, most importantly keeping the supply (hot wires) and return (white wires) from being separated by distance and keeping the wiring equal in length so hot and neutral fields cancel out. Main electrical service panels and subpanels can create magnetic fields and also need hot and neutral fields cancelled. Switches can also generate magnetic fields if improperly wired.

Proper bonding and grounding of utilities is important as electrical current can be carried on return neutral pathways (connected to house grounding system) in utility multi grounded neutral systems. Steps can be taken to prevent and /or minimize this electric current.

Lowering electrical fields involves keeping electrical wiring in MX, MC, or rigid metal conduit and electrical boxes in metal to shield electric fields. A demand or "kill" switch for the bedroom eliminates electric and magnetic fields while a person sleeps. Shield electric fields emitted from refrigerators by placing them on a dedicated circuit with wiring in metal conduit. Placing large appliances away from living and sleeping places lowers exposure to both magnetic and electrical fields.

Location of electric service panels and utilities should be at approximately the same location and furthest away from living and sleeping quarters.

When finished, use a gaussmeter to test the house with power turned off to help detect any problem areas.

## References for "Building Considerations for Low EMF Home"

1. Chemical Sensitivity Vol. 4 by Dr. William Rea, CRC Press, Inc., pp. 2017-2046.
2. The Health House, 4th Edition, by John Bower, Healthy House Institute, 2001, pp.304-322.
3. Prescriptions for a Healthy House by Paula Baker-Laporte, Erica Elliot, and John Banta, New Society Publishers, 2001, pp. 269-284
4. Interview and frequency chart per Joel-Anthony Gray, Director of ScanTech and IT Consulting EMF - RF - Radon - Radiation - DC Geomagnetic Scans & Surveys  
Technical Consulting  
www.scantech7.com  
scantech@warppmail.net  
Cell 214.912.4691  
Fax 214.343.9027
5. "Investigating and minimizing AC Magnetic and Electric Field Exposure in Existing Buildings" by Spark Burmaster, 2/1998.
6. "Effects of Being Connected to a Utility Multi Grounded Neutral System" by Spark Burmaster, 8/2006
7. "Electromagnetic Man" by Dr. Cyril Smith and Simon Best, The Bath Press, Avon for J.M. Dent and Sons Ltd., 1989, pp. 86-103, 158-161, 200, 239-240, 264-265.



## Objectives & Notes

**Gerald H. Ross, M.D.**

Date of talk: Friday, June 8, 2007, 5:15pm

Bountiful, UT

Phone: N/A

### Training:

Current Job Description:	Semi-Retired
Current Faculty Appointments:	N/A
Medical School/ University Attended	Dalhousie University School of Medicine, Halifax, Nova Scotia
Internship:	Dalhousie University School of Medicine, Halifax, Nova Scotia
Residency:	Through the College of Family Physicians of Canada
Board Certifications:	Family Medicine (Canada), Environmental Medicine (USA)

### CASE STUDY: “Toxic Buildings from one Simple Common Procedure”

At the end of this Presentation, the participant should be able to:

1. Demonstrate the common method of building humidification in North America
2. Identify the class of common boiler additives used in buildings
3. Describe common symptoms resulting from boiler additives.

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## ***TOXIC BUILDINGS FROM ONE SIMPLE COMMON PROCEDURE:***

### ***A CASE PRESENTATION***

Gerald H. Ross, MD

#### **Goals and objectives:**

*At the end of this presentation, participants should be able to:*

- 1) *Describe the common method of humidification of indoor air in North America.*
- 2) *Identify the usual class of boiler additives used in buildings.*
- 3) *Describe common symptoms resulting from exposure to boiler additives.*

**Abstract:** This case presentation involves the 53 year old woman who had worked inside a certain building for many years. She has the gradual development of a number of health problems over several years, but in the fall of 1989, she and about 20 other occupants of the building had an significant intensification of symptoms.

Her principal symptoms were significant mucous membrane irritation, such as:

itching, irritated eyes; blurry vision; tearing of the eyes; photophobia; blisters or cysts at the inside corners of her eyes and under eyelids; painful eyes, burning and 'raw' feeling inside her nose; burning of lips; swelling of face; hoarseness or loss of voice; heavy feeling in lungs; continual clearing of throat mucus; breathing distress; wheezing requiring inhalers.

The patient was seen by a significant number of physicians before being referred to an Environmental Medicine physician by an ophthalmologist, because of long-standing 'toxic keratitis' that was unresponsive to treatment.

Extensive investigation followed, and eventually the building was found to be widely contaminated with the aliphatic amine *cyclohexylamine*, which is commonly used as a boiler additive in heating plants.

The attendees will be instructed in the recognition of this common practice, in spite of NIOSH issuing calls for its cessation, because it is too dangerous. The characteristics of symptoms produced by cyclohexylamine will be reviewed and its common effects on humans reviewed.

**Conclusions:** Cyclohexylamine and other aliphatic amines are commonly added to boilers in heating plants for buildings, and the chemical can significantly contaminate the interior of large buildings. This may be one of the reasons who chemically sensitive patients do singularly poorly when they spend time inside larger public buildings.

#### **References:**

American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I, II, III. Cincinnati, OH: ACGIH, 1991. 365

NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Washington, D.C.: U.S. Government Printing Office, June 1994. 84

NIOSH; National Occupational Exposure Survey (NOES) (1983)

Edgerton SA et al; Environ Sci Technol. 23: 484 - 488 (1989)

# 25<sup>th</sup> ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT

## Schedule

### Saturday, June 9, 2007

8:00 **ANNOUNCEMENTS/MODERATOR:** Kaye H. Kilburn, M.D.

8:05 Jean Monro, M.D., "Man's Sense of Awareness as Illustrated by Autonomic Dysfunction"

8:25 Q & A

8:35 William J. Meggs, M.D., Ph.D., "Neurotoxicity of Autonomic Nervous System II: Diagnosis & Treatment"

8:55 Q & A

9:05 William J. Rea, M.D., "Treatment of Autonomic Dysfunction"

9:25 Q & A

9:35 Paul R. Cheney, M.D., Ph.D., "High Patent Foramen Ovale (PFO) Frequency in Chronic Fatigue Syndrome"

9:55 Q & A

### 10:05 BREAK WITH EXHIBITORS

10:50 Monica Sandstrom, Ph.D., "Provocation with Mobil Phone Signals"

11:10 Q & A

11:20 Dale Griffin, Ph.D., "Desert Dust Microbiology and Human Health"

11:40 Q & A

### 11:50 BUFFET LUNCH WITH THE EXHIBITORS

1:00 **MODERATOR:** Jean Monro, M.D.

1:00 Theodore R. Simon, M.D., "Functional Imaging in 2007"

1:20 Q & A

1:30 Amer Suleman, M.D., "Postural Orthostatic Tachycardia Syndrome"

1:50 Q & A

2:00 Divakar Dalela, M.D., "Diet & Cancer Prostate: Interrelationships & Implications"

2:20 Q & A

2:30 Riki Ott, Ph.D., "Sociological Paradigm Shifts after the Exxon Valdez Oil Spill"

2:50 Q & A

### 3:00 BREAK WITH EXHIBITORS

3:45 Klaus-Dietrich Runow, M.D., "Poisoned Children - Toxic Metal Intoxication in Kosovo"

4:05 Q & A

4:15 Cyril W. Smith, Ph.D., "The ANS - a Unified Approach"

4:35 Q & A

4:45 Tang G. Lee, Ph.D.: "Beyond 60 Hz EMF to Radio-Frequencies"

5:05 Q & A

5:15 Donald Hillman, Ph.D.: "EMF in Homes, Workplaces, and Schools, Part 2"

5:35 Q & A

5:45 CASE STUDY: Amy Dean, D.O., "Vasculitis Responsive to Environmental & Osteopathic Medical Intervention"

Panel: Doctors Monro, Meggs, Rea, Cheney, Sandstrom, Griffin, Simon, Suleman, Dalela, Ott, Runow, Smith and Lee

6:00 **AJOURN**

**SATURDAY, JUNE 9, 2007**

**ABSTRACTS**

**AND**

**HANDOUTS**

## Objectives & Notes

### Jean Monro, M.D.

Breakspear Hospital  
Hertfordshire House  
Wood Lane, Paradise Estate  
Hemel Hempstead, Herts HP2 4FD  
England

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### Training:

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Medical School/ University Attended	London Hospital Medical School, England
Residency:	London Hospital
Board Certifications:	MB, BS, MRCS, LRCP, FAAEM, DipIBEM, MACOEM
Other Information: (including titles of books or articles you have recently written):	Treatment of cancer with mushroom products. Arch Environ Health 2003;58:533-7 Coriolus. Available from www.jintmed.com

### SPEECH TITLE: "Man's Sense of Awareness as Illustrated by Autonomic Dysfunction"

At the end of this Presentation, the participant should be able to:

1. Quantitative organ-specific tests are available for Chronic Fatigue Syndrome.
2. Cardiovascular regulation is a target of environmental toxicity.
3. Brainstem activity is a good index of autonomic function in patients suffering from the effects of environmental toxicity.

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# Breakspear Hospital



Title: **MAN'S SENSE OF AWARENESS AS ILLUSTRATED BY AUTONOMIC DYSFUNCTION**

Venue: **25<sup>TH</sup> ANNUAL INTERNATIONAL SYMPOSIUM ON MAN AND HIS ENVIRONMENT, DALLAS, 7<sup>TH</sup>-10<sup>TH</sup> JUNE 2007**

Author: **JEAN A MONRO**  
**MB, BS, MRCS, LRCP, FAAEM, DIBEM, MACOEM**  
**Medical Director, Breakspear Hospital, Wood Lane, Hemel Hempstead, Herts, HP2 4FD, UK**

In Association with: **DR PETER JULU**  
**MBChB, MSc, PhD**  
**Autonomic Neurophysiologist/Senior Research Fellow, Breakspear Hospital, Wood Lane, Hemel Hempstead, Herts, HP2 4FD, UK**

## ABSTRACT

### *Dysfunctional Brainstem Autonomic Activity and Inotropic Fatigability in Chronic Fatigue Syndrome*

Chronic Fatigue Syndrome (CFS) is characterised by long-term fatigue, impairment of short-term memory and musculoskeletal pain (Fukuda et al., 1994). The cause of CFS is not known. Pain is an important reason for the disability in CFS. However, although patients with CFS experience muscle pain, they also have severe post-exertion malaise following sustained exercises. The mechanism of increased muscle pain after exercise in CFS is not known but is considered to be an important reason for low compliance with graded exercise therapy (Chaudhuri and Behan, 2004). The mechanism of the malaise and the disproportionate exhaustion following sustained exercise is also not known.

Our hypothesis is that cardiovascular regulation by the autonomic nervous system is abnormal in CFS and fails to match the cardiac output with the demands in muscles during a sustained exercise. A mismatch between tissue perfusion and metabolism can explain the increased muscle pain following a sustained exercise. We have investigated the possibility of autonomic involvement in this post-exertion malaise in patients with CFS diagnosed using the criteria proposed by Fukuda et al. (1994).

Fifteen patients with CFS had target-organ orientated examination of the autonomic nervous system done using the NeuroScope system (MediFit Instruments Ltd, London, UK) as described by Julu et al. (1997). These were 13 women aged between 12-73 (mean age 46) years and two teenage boys (mean age 15.5 years) attending routine clinics at Breakspear Hospital, London. Since we were interested in the autonomic regulation of the cardiac output, we examined in details the cardioaccelerator, inotropic and baroreflex functions of the cardiovascular system. Cardioaccelerator and inotropic function were measured during a sustained isometric exercise of the dominant hand as previously described (Julu et al., 2000). We used the VaguSoft software (MediFit Instruments Ltd, London, UK) to

measure the gradient of the arterial pressure rise during the ejection period of the cardiac cycle to serve as a real-time index of inotropic function of the heart. We also measured the cardiac vagal tone (CVT) and the cardiac sensitivity to baroreflex (CSB) continuously at rest in the supine position and during the isometric exercise in a sitting position to monitor in real-time the baroreflex regulation of the heart at the brainstem level using the NeuroScope system as previously described (Julu et al., 2003). The autonomic function in our patients with CFS was compared with the laboratory standards we use in routine diagnostics.

Eight patients (53%) had Abnormal Spontaneous Brainstem Activations (ASBA) in which there were large, repetitive and spontaneous increases in CVT, CSB and blood pressure for no apparent reasons. All but two patients had measurable fatigability of the inotropic function of the heart precipitated either by orthostasis or by a sustained isometric contraction of the forearm muscles.

We conclude that a large proportion of patients with CFS have detectable irritability of the brainstem, a sign of neuronal hypersensitivity with such low thresholds to have spontaneous activations. This could explain abnormal sensory awareness in these patients. We also have presented here evidence that suggests inadequate tissue perfusion in nearly all patients with CFS caused by cardiac inotropic fatigue. The inotropic fatigue is precipitated by contraction of postural skeletal muscles, which are known to be rich in mitochondria and myoglobin and would use far more resources required for ATP production compared with myocardium. Therefore, it is likely that a yet unidentified resource(s) for ATP production has a limited supply in the body and can be depleted in CFS. This is the first quantifiable evidence of fatigability of cardiac inotropic function in CFS. We suggest that the autonomic dysfunctions we have shown here can explain most of the unique sense of environmental awareness in our patients.

#### Reference List

1. Chaudhuri A, Behan PO (2004) Fatigue in neurological disorders. *Lancet* 363: 978-988.
2. Fukuda K, Straus SE, Hickie I, Sharpe MC, Dobbins JG, Komaroff A (1994) The chronic fatigue syndrome: a comprehensive approach to its definition and study. International Chronic Fatigue Syndrome Study Group. *Ann Intern Med* 121: 953-959.
3. Julu PO, Cooper VL, Hansen S, Hainsworth R (2003) Cardiovascular regulation in the period preceding vasovagal syncope in conscious humans. *J Physiol* 549: 299-311.
4. Julu PO, McCarron MO, Hansen S, Barnes A, Jamal GA, Ballantyne JP (1997) Selective defect of baroreflex blood pressure buffering with intact cardioinhibition in a woman with familial aniridia. *Neurology* 49: 1705-1708.
5. Julu PO, McCarron MO, Hansen S, Job H, Jamal GA, Ballantyne JP (2000) Complex regional pain syndrome with selective emotional sudomotor failure. *Eur J Neurol* 7: 351-354.



## Objectives & Notes

**William J. Meggs, M.D., Ph.D.**

Date of talk: Saturday, June 9, 2007, 8:35pm

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Current Job Description:	Physician and Research Scientist
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Internship:	University of Rochester
Residency:	University of Rochester
Board Certifications:	Medical Toxicology, Allergy and Immunology, Internal Medicine, Emergency Medicine
Other Information: (including titles of books or articles you have recently written):	Book: The Inflammation Cure, McGraw-Hill (2003)/ Fallacies in Refutation of Causality, published in Clinical Toxicology, 2005. Sustained oxygenation without ventilation, published in Amer. Journal Emergency Medicine in 2005. Pressure Immobilization Bandages delay of toxicity of coral snake bites, published in Annals of Emergency Medicine, 2005.

### SPEECH TITLE: "Neurotoxicity of the Autonomic Nervous System II: Diagnosis and Treatment"

At the end of this Presentation, the participant should be able to:

1. To know how to diagnosis autonomic dysfunction using analytical tools such as heart rate variability and pupillography.
2. To know the literature using these tools to diagnosis autonomic dysfunction for specific toxins, including occupational exposure to lead, the nerve gas sarin, and occupational and acute poisons with organophosphate insecticides.
3. To know modalities for the treatment of autonomic dysfunction.

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## Neurotoxicity of the Autonomic Nervous System II: Diagnosis and Treatment

### Goals and Objectives

- To know how to diagnosis autonomic dysfunction using analytical tools such as heart rate variability and pupillography.
- To know the literature using these tools to diagnosis autonomic dysfunction for specific toxins, including occupational exposure to lead, the nerve gas sarin, and occupational and acute poisons with organophosphate insecticides.
- To know modalities for the treatment of autonomic dysfunction.

### Conclusions

Autonomic dysfunction can result from exposures to a number of toxins and can persist long after the exposure. Bedside examination can be used to diagnosis autonomic dysfunction when it is grossly apparent, but sometimes the findings are subtle. For example, using bedside diagnosis, the clinician shines a light into a patient's eye and notes that the pupil constricts. The examination will be described as normal. Using the technique of pupillography, in which the latency period before constriction, the rate of constriction, the change in area, and the recovery time are measured, allows the detection of subtle abnormalities that are not obvious on bedside examination. These abnormalities are evidence of brain damage. An important principle is that autonomic dysfunction is seldom an isolated finding. This documentation of brain damage can be important objective evidence of a poisoning with effects that persist long after direct detection of the poison is no longer possible. Heart rate variability is another parameter that can be used to detect autonomic dysfunction. The literature supporting the use of these techniques in specific poisonings, including lead, organophosphate insecticides, the nerve gas sarin, road dust, carbon disulfide, and organic mercury poisoning, will be reviewed. Approaches to treating autonomic dysfunction will be discussed.

### References

Bremner, F. Smith, S. Pupil findings in a consecutive series of 150 patients with generalised autonomic neuropathy. *Journal of Neurology, Neurosurgery & Psychiatry*. 77(10):1163-8, 2006 Oct.

*Circulation*. 1996;93:1043-1065  
<http://circ.ahajournals.org/cgi/content/full/93/5/1043>

Filippov VL. Shumakova KM. Tsimbal FA. [Use of pupillometry in the diagnosis of neurologic disorders caused by organophosphorus compound poisoning]. [Russian] *Meditina Truda i Promyshlennaia Ekologiia*. (6):11-6, 1997.

Haley RW. Vongpatanasin W. Wolfe GI. Bryan WW. Armitage R. Hoffmann RF. Petty F. Callahan TS. Charuvastra E. Shell WE. Marshall WW. Victor RG. Blunted circadian variation in autonomic regulation of sinus node function in veterans with Gulf War syndrome. *American Journal of Medicine*. 117(7):469-78, 2004 Oct 1.

Oka T. Matsukura M. Okamoto M. Harada N. Kitano T. Miike T. Futatsuka M. Autonomic nervous functions in fetal type Minamata disease patients: assessment of heart rate variability. *Tohoku Journal of Experimental Medicine*. 198(4):215-21, 2002 Dec.

Sanner BM. Rawert B. Henning B. Zidek W. Ciguatera fish poisoning following travel to the tropics. *Zeitschrift fur Gastroenterologie*. 35(5):327-30, 1997 May.

Shirakawa S. Ishikawa S. Miyata M. Rea WJ. Johnson AR. [A pupillographical study on the presence of organochlorine pesticides in autonomic nerve disturbance]. [Japanese] *Nippon Ganka Gakkai Zasshi - Acta Societatis Ophthalmologicae Japonicae*. 94(4):418-23, 1990 Apr.

Wilhelm: *Pupillographic Devices*. *J Neuroophthalmol*, Volume 23(1).March 2003.42-49

## Objectives & Notes

**William J. Rea, M.D.**

Date of talk: Saturday, June 9, 2007, 9:05am

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### Training:

Current Job Description:	Founder and President of the Environmental Health Center - Dallas
Medical School/ University Attended	Ohio State University College of Medicine
Internship:	Parkland Memorial Hospital
Residency:	University of Texas Southwestern Medical School
Board Certifications:	American Board of Surgery, American Board of Thoracic Surgery, American Board of Environmental Medicine
Other Information: (including titles of books or articles you have recently written):	Optimum Environments for Optimum Health and Creativity (book); Averse Health Effects of Indoor Molds.

### SPEECH TITLE: **"Treatment of Autonomic Dysfunction"**

At the end of this Presentation, the participant should be able to:

1. Recognize that there is no drug therapy for autonomic syndrome.
2. Recognize how to perform the treatment.
3. Apply the treatment in a clinical setting.

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## Objectives & Notes

**Paul R. Cheney, M.D., Ph.D.**

Date of talk: Saturday, June 9, 2007, 9:35am

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### Training:

Current Job Description:	Private Practice: The Cheney Clinic
Current Faculty Appointments:	None
Medical School/ University Attended	Emory University, M.D.; Duke University, Ph.D.
Internship:	Emory University affiliated hospitals
Residency:	Emory University affiliated hospitals
Board Certifications:	Internal Medicine
Other Information: (including titles of books or articles you have recently written):	Written and presented over 25 publications

### SPEECH TITLE: “**High Patent Foramen Ovale (PFO) in Chronic Fatigue Syndrome**”

At the end of this Presentation, the participant should be able to:

1. Understand the physiology and function of PFO’s
2. The pathophysiology at right to left shunt through PFO’s
3. The possible meaning of a high frequency of PFO’s in CFS.

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## **ABSTRACT**

### **Evidence of Increased Frequency of Patent Foramen Ovale (PFO) in the Chronic Fatigue Syndrome with Enriched Oxygen Modulation of the PFO**

Cheney, PR and Lucki, NC

**Background Background** There is clinical evidence of a functional left shift on the oxygen-hemoglobin dissociation curve using pulse oximetry with end-expiratory breathhold in the chronic fatigue syndrome (CFS). There is also evidence of increased as well as decreased citrate excretion on urine organic acid analysis in many CFS patients. Citrate is a potent inhibitor of 2-3 DPG and therefore a significant modulation of oxygen-hemoglobin dissociation would be expected. Recent published reports of increased RBC 2-3 DPG levels in CFS was attributed to a physiologic compensation for increased RBC oxidative stress with an associated functional left shift. Finally, there is evidence of reduced oxygen utilization on exercise ergometry with gas analysis reported by many investigators which is consistent with a functional left shift. We hypothesized that a left shift (real or functional) on the oxygen-hemoglobin dissociation curve would raise pulmonary artery pressures and combined with low systemic pressures typical for CFS patients and evidence of cardiac diastolic dysfunction (see other abstract) would result in an increased frequency of PFO. Diastolic dysfunction due to a primary relaxation defect linked to an underlying cellular energy defect would be expected to enlarge the foramen ovale by left atrial dilation and combined with LA systolic cavitation also associated with diastolic dysfunction and with a resulting favorable right to left pressure differential would increase the chance of right to left shunting through a PFO.

**Methods** We interrogated 60 consecutive patients presenting to a national CFS referral clinic all of whom met the criteria for CFS. The average age was 49.1 yrs (range 16-68) and included 40 females and 20 males. The patients were drawn from a wide geographic area (19 states) and the great majority (94%) were disabled with an average length of illness of 15 years. Using a high frame rate, all digital, high resolution echocardiograph machine (GE Vivid-7), and standard saline bubble testing with and without valsalva and infusing through the antecubital vein, we looked for evidence of a positive right to left shunt and quantified the shunt using a standard grading system (Grade I-V). Virtually all positive shunts occurred early (within 3 beats) following valsalva. In addition, we interrogated 42 patients for PFO sensitivity to oxygen therapy. In 16 patients (all positive for PFO), enriched oxygen (FIO<sub>2</sub>=42%) was administered via demand regulator in a closed system for 15 minutes with the PFO re-tested followed by a third test for PFO at 5 minutes post-oxygen therapy. In another 19 patients (all positive for PFO), a lower level of enriched oxygen was administered by nasal prongs at 4.0 lpm for 15 minutes, also with re-testing for PFO and again at 5 minutes post-oxygen therapy. Using the diastolic parameter IVRT response to oxygen, all interrogated patients were assessed either as oxygen responsive (IVRT went down) or as oxygen toxic (IVRT went up). IVRT is the most energy specific echocardiographic parameter. Lower IVRT means increased energy whereas higher IVRT means decreased energy.

**Results** Out of 60 patients tested, 52 of 60 or 86.7% were positive for a right to left shunt. The grading of PFO's showed a wide range of severity (Grade I-42%, Grade II-23%, Grade III-20%, Grade IV-6%, Grade V-9%). Approximately one third of the patients showed right to left shunting without valsalva. Enriched oxygen therapy (FIO<sub>2</sub> = 42%) resulted in closure of 5 of 16 PFO's tested (31.3%) using a demand regulator in a closed system for 15 minutes. Three of five who closed off their PFO re-opened their PFO at 5 minutes post oxygen. Lower dose oxygen (4.0 lpm by NC) resulted in closure of PFO in 5 of 26 tested (19.2%). All re-opened their PFO given low dose oxygen at 5 minutes post oxygen. Two patients experienced migraine during the oxygen therapy and both significantly increased their PFO severity grade with oxygen. All PFO patients demonstrating oxygen responsiveness by IVRT criteria (falling on oxygen) had at least a one grade drop in PFO grade, many closing. Conversely, 35% of oxygen toxic patients worsened their PFO by at least one grade at 15 minutes and all were worse than baseline PFO grade after 5 minutes post oxygen. Conversely, all oxygen responsive patients were no worse than baseline after 5 minutes post oxygen and many improved their PFO grade.

**Discussion** The finding of an 87% incidence of PFO in CFS is higher than any other reported association including cryptogenic stroke and migraine which are associated with a 50% incidence of PFO. The expected frequency of PFO detected by bubble testing in the general population is 10-15%. The finding that CFS PFO's as well as intracellular myocardial energetics (IVRT) can be highly modulated by oxygen, strongly supports a defect in the handling of oxygen by-products (ROS) in CFS similar to that seen in fetuses. Indeed, fetal hemoglobin with its left shift on the

oxy-hgb dissociation curve is in part responsible for the presence of PFO in fetal life and fetal intra-cardiac physiology mimicks that seen in CFS with respect to oxygen modulation. Furthermore, the left shift and reduced oxygen transfer protects the fetus from the deleterious effects of oxygen metabolism (ROS). In this sense, CFS patient energetic decline may be viewed as a protective strategy against the effects of oxygen metabolism which they are unable to handle. Furthermore, the finding of a 78% incidence of stroke-like UBO's on MRI brain scans in the Lake Tahoe CFS epidemic (1992) may be explained in part by the 87% incidence of PFO in CFS combined with the possibility of enhanced altitude effects on PFO shunting expected at the Lake Tahoe elevation of 6,200 ft. Altitudes above 6,000 ft evoke an increase in pulmonary artery pressure and increases the right to left pressure gradient across a PFO and favors a R-L shunt.



## Objectives & Notes

**Monica Sandström, Ph.D.**

Umeå University  
90187 Umeå  
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Date of talk: Saturday, June 9, 2007, 10:50am

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Email: monica.sandstrom@envmed.umu.se

### Training:

Current Job Description:	Researcher, Non Ionizing Radiation Group, National Institut for Working Life, Umeå, Sweden
Current Faculty Appointments:	Researcher
Other Information: (including titles of books or articles you have recently written):	M. Sandström, E. Lyskov, R. Hörnsten, K. Hansson Mild, U. Wiklund, P. Rask, V. Klucharev, B. Stenberg and P. Bjerle. Holter ECG monitoring in patients with perceived electrical hypersensitivity. Internat J Psychophysiol. 49; 227-235, 2003. J. Wilen, A. Hohansson, N.a Kalezic, E. Lyskov and M. Sandström. Psychophysiological tests and provocation of subjects with mobile phone related symptoms. Published Online: 22 Nov 2005. DOI: 10.1002/bem.20195. Bioelectromagnetics27:pp 204-214, 2006.

**SPEECH TITLE: “Provocation with Mobil Phone Signals”** (Monica Sandström, Ph.D., Amanda Johansson, Ph.D. student, National Institute for Working Life, Umeå, Sweden)

At the end of this Presentation, the participant should be able to:

1. The most frequent reported symptoms
2. Method of provocation
3. result of the study

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# Provocation with mobile phone-like signals

Monica Sandström, PhD, Amanda Johansson, PhD student

National Institute for Working Life and the Department of Occupational and Environmental Medicine, Umeå University, Umeå, Sweden

In a previous epidemiological study in Sweden and Norway we found that heavy mobile phone (MP) users reported symptoms more often than people that seldom used their MPs. The most commonly reported symptoms were sensations of warmth on and around the ear, burning sensations of the skin, and headaches. This group of people (MP group) reported symptoms similar to those of people claiming to be sensitive to electromagnetic fields in general (electrical hypersensitivity, EHS). However, the MP group generally did not connect the perceived symptoms with other electrical appliances than MPs, and there might be reason to believe that these two groups are not similar with respect to the origin of the symptoms. In earlier provocation studies we found that people with EHS showed deviations in the autonomic regulation and in the response to sensory stimuli when compared to a control group. However, these deviations were not correlated to electromagnetic field exposure.

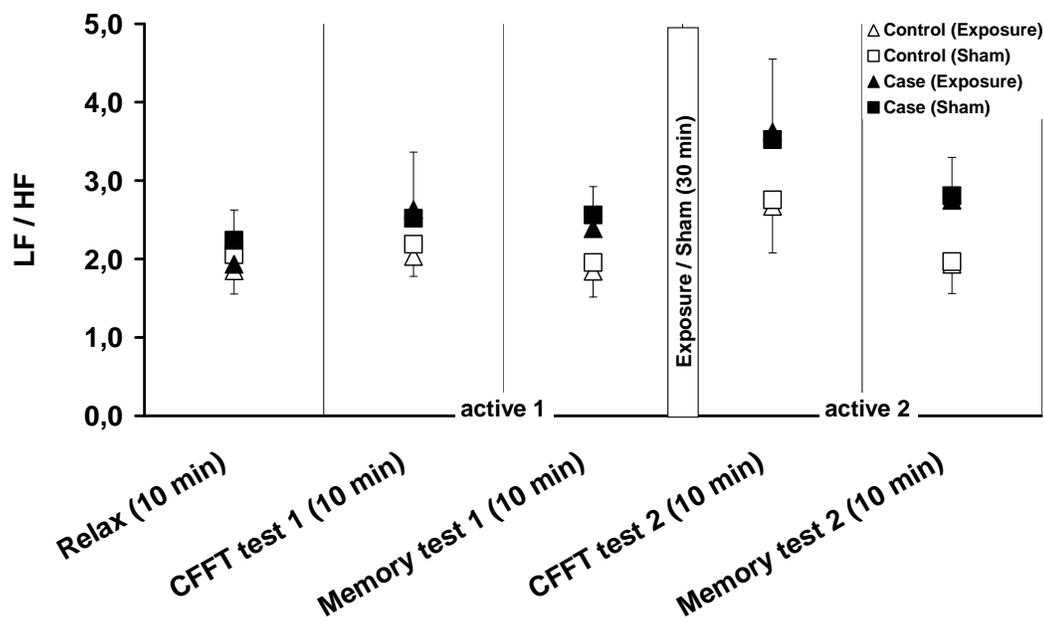
A provocation study was designed in order to find out if exposure to mobile phone-like radiofrequency (RF) signals would provoke symptoms in the MP group and furthermore if RF exposure would affect physiological and cognitive parameters.

Twenty subjects with MP-related symptoms were recruited together with 20 matched controls without MP-related symptoms. Each subject participated in two experimental sessions, one with true exposure and one with sham exposure, in random order. In the true exposure condition, the test subjects were exposed to an RF field generated by an indoor base station antenna attached to a 900 MHz GSM MP which generated a SAR<sub>g</sub> distribution 1 W/kg. Heart rate and heart rate variability (HRV), respiration, local blood flow, electrodermal activity, critical flicker fusion threshold (CFFT), short-term memory, and reaction time were measured before and after the exposure sessions.

Adaptation	CFFT test	Memory test	Exposure/Sham	CFFT test	Memory test
<i>10 min</i>	<i>10 min</i>	<i>10 min</i>	<i>30 min</i>	<i>10 min</i>	<i>10 min</i>

*Schematic drawing of the experimental procedure during Day 1 and Day 2.*

No significant differences related to RF exposure conditions were detected. Also no differences in baseline data between subject groups were encountered, except for a significantly longer reaction time among the cases than among the controls the first time the test was performed. This difference disappeared when the test was repeated. However, the cases differed significantly from the controls with respect to HRV as measured in the frequency domain. There were no differences in baseline HRV, but during the CFFT and memory tests the cases displayed a shift in low/high frequency ratio, suggesting a sympathetic dominance in the autonomous nervous system. This deviation was present regardless of exposure condition, and might be interpreted as a sign of differences in the autonomous nervous system regulation between persons with MP related subjective symptoms and persons with no such symptoms.



Mean values and 95% CI of LF/HF ratio during the experiment.

## References

- Haarala C, Ek M, Björnberg L, Laine M, Revonsuo A, Koivisto M, Hämäläinen H. 2004. 902 MHz mobile phone does not affect short term memory in humans. *Bioelectromagnetics* 25(6):452-6.
- Huber R, Schuderer J, Graf T, Jutz K, Borbely AA, Kuster N, Achermann P. 2003. Radio frequency electromagnetic field exposure in humans: Estimation of SAR distribution in the brain, effects on sleep and heart rate. *Bioelectromagnetics* 24(4):262-76.
- Koivisto M, Haarala C, Krause CM, Revonsuo A, Laine M, Hämäläinen H. 2001. GSM phone signal does not produce subjective symptoms. *Bioelectromagnetics* 22(3):212-215.
- Oftedal G, Wilén J, Sandström M, Hansson Mild K. 2000. Symptoms experienced in connection with mobile phone use. *Occupational Medicine-Oxford* 50(4):237-245.
- Sandström M, Lyskov E, Hörnsten R, Hansson Mild K, Wiklund U, Rask P, Klucharev V, Stenberg B, Bjerle P. 2003. Holter ECG monitoring in patients with perceived electrical hypersensitivity. *Int J Psychophysiol* 49(3):227-35.
- Sandström M, Wilén J, Oftedal G, Hansson Mild K. 2001. Mobile phone use and subjective symptoms. Comparison of symptoms experienced by users of analogue and digital mobile phones. *Occupational Medicine-Oxford* 51(1):25-35.



## Objectives & Notes

**Dale W. Griffin, Ph.D.**

Date of talk: Saturday, June 9, 2007, 11:20am

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### Training:

Current Job Description:	Environmental and Public Health Microbiologist
University Attended	University of South Florida
Other Information: (including titles of books or articles you have recently written):	MS PH 1994 University of South Florida College of Public Health, Ph.D. 1999 Marine Science – Specializing in environmental and public health microbiology -33 published peer reviewed papers, book chapter and 26 other pubs (convention papers, reports, etc.)

### SPEECH TITLE: “Desert Dust Microbiology and Human Health”

At the end of this Presentation, the participant should be able to:

1. Understand the variety and concentrations of microorganisms associated with dust storm clouds and their relationship to human health.
2. Understand the ability and frequency that large dust storms move through our atmosphere on a global scale.

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## Desert dust microbiology and human health

Dale W. Griffin, Ph.D., MSPH, Environmental and Public Health Microbiologist

U.S. Geological Survey, Florida Integrated Science Center, Tallahassee, Florida 32310

Dust storms move an estimated  $3 \times 10^{15}$  grams of soil some distance in Earth's atmosphere each year. Large dust events are capable of global dispersion. Microbial ecology studies in soil has shown that there are approximately 1 million to 1 billion bacteria cells (pathogens and non-pathogens) per gram, and that this community is composed of approximately 4,000 species. Other microorganisms present in a typical gram of soil include ~ 10 thousand to 10 million viruses, ~ 1 million fungi, and ~ 10 thousand protozoa. This presentation will cover our current state of knowledge in this emerging research field to include historical and current observations, and how specific organisms within these clouds may impact human health.

## Objectives & Notes

### Theodore R. Simon, M.D.

North Texas Imaging Center  
8345 Walnut Hill Lane, Ste. 210  
Dallas, TX 75231

Date of talk: Saturday, June 9, 2007, 1:00pm

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Email: ted@aya.yale.edu

### Training:

Current Job Description:	Physician
Medical School/ University Attended	Yale
Internship:	University of Rochester
Residency:	University of California at San Francisco and Yale
Board Certifications:	American Board of Nuclear Medicine
Other Information: (including titles of books or articles you have recently written):	See CV at <a href="http://www.theodorersimon.com">www.theodorersimon.com</a>

### SPEECH TITLE: “**Functional Imaging in 2007**”

At the end of this Presentation, the participant should be able to:

1. Describe the relationship between biochemical brain abnormalities and neuropsychological deficits
2. Determine whether neuropsychological testing should be supplemented by SPECT imaging
3. Address the need for objective data in the neurologically impaired patient

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# Neuropsychological Correlates to Brain SPECT Imaging

by Theodore R. Simon, M.D. and Nancy Didriksen, Ph.D.

## Hypothesis

Since the abnormalities associated with neurotoxicity on SPECT images diffusely encompass most of the cortex, higher level functioning should be compromised across various measures of neuropsychological testing that involve higher cortical function.

## Subjects

The most recent subjects who had Brain SPECT imaging in our laboratory were retrospectively considered for this study. Inclusion was limited to the last 55 who had a Halstead-Reitan (H-R) battery performed by one of us (ND). Twenty-one males and thirty-four females were included, all of whom had clinical evidence of neurotoxicity by balance testing.

## Method

H-R scores for “Tactual Performance”, “Category Test”, and “Overall” were compared to the SPECT criteria used in assessing neurotoxicity by grading individual scores as normal, mildly abnormal, moderately abnormal, and severely abnormal. SPECT scores were similarly graded with respect to the “salt and pepper pattern”, mismatch between the early and late phases, temporal asymmetry, and redistribution to the soft tissues. Exact matches and one grade differences were separately compiled.

## Results

Category Test grades showed more extensive abnormalities than the SPECT and other H-R scores. The highest agreement among the SPECT and various H-R scores was in the moderately impaired subjects that allowed for a one grade difference. In this group, correspondence was unity except for two subjects in the Tactual v Mismatch and Tactual v Redistribution groups and one subject in the Tactual v Temporal Asymmetry and H-R overall v Temporal Asymmetry groups.

## Conclusion

SPECT and neuropsychology (as measured by H-R Tactual Performance, Category Test and Overall) have good agreement when they identify patients with clinical evidence of neurotoxicity. This agreement was robust for higher functioning tests as well as the overall scores.

## Objectives & Notes

**Amer Suleman, M.D.**

Date of talk: Saturday, June 9, 2007, 1:30pm

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McKinney, TX 75069

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### Training:

Current Job Description:	E.P. Cardiologist (Consultant)
Medical School/ University Attended	King Edward Medical College Lahore, Pakistan
Internship:	Mayo Hospital Lahore, Pakistan
Residency:	State University of New York, School of Medicine at Buffalo
Board Certifications:	American Board of Internal Medicine, Cardiology and Electrophysiology
Other Information: (including titles of books or articles you have recently written):	Journal Reviewer, Journal of Clinical Electrophysiology. CME Editor, Section O Cardiology, Online Text Book of Medicine.

### SPEECH TITLE: “**Postural Orthostatic Tachycardia Syndrome**”

At the end of this Presentation, the participant should be able to:

1. Understand pathophysiology of POTS
2. Understand basic types
3. Construct a treatment algorithm for management of POTS

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## Objectives & Notes

**Divakar Dalela, M.D.**

Date of talk: Saturday, June 9, 2007, 2:00pm

Professor & Head, Department of Urology, King George  
Medical University  
Lucknow, U.P. 226 003  
India

Phone: 0522-2256543, 6570321  
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### Training:

Current Job Description:	Full time Consultant Urologist & Training Postgraduates for M.Ch. Urology
Current Faculty Appointments:	Professor & Head, Dept. of Urology, K.G. Medical University, Lucknow.
Medical School/ University Attended	K.G. Medical College, Lucknow -1979-1987, SGPGIMS, Lucknow – 1988-1992
Internship:	K.D. Medical College, Lucknow – 1985-86
Residency:	S.R. Surgery, K.G. Medical College, Lucknow – 1986-1988, S.R. Urology, SGPGIMS -1989-1992
Board Certifications:	M.B.B.S.; M.S. (Surgery); M.Ch. (Urology)
Other Information: (including titles of books or articles you have recently written):	Publication details available on website: kgmcindia.edu (Urology Department); drdivakardalela.com

### SPEECH TITLE: “Diet & Cancer Prostate: Interrelationships & Implications”

At the end of this Presentation, the participant should be able to:

1. Learn the role of dietary factors in causation of prostate cancer
2. Reason out a preventive strategy
3. Explore possibilities of newer researches to reaffirm the linkage.

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## **Diet and Cancer Prostate: Interrelationships and Implications**

Divakar Dalela<sup>1</sup>, Manish Ahuja<sup>2</sup>

<sup>1</sup>Professor & Head

<sup>2</sup>Senior Resident

Department of Urology, King George's Medical University, Lucknow (India)

### **Abstract**

Prostate cancer is the fourth most common malignancy in men worldwide. The prostate cell growth is under the influence of androgens. Oxidative damage by free radicals to DNA results in uninhibited growth of prostate cells resulting in cancer. Genetics, dietary and environmental factors play an important role in its etiology. There is ample epidemiological evidence to suggest that diet has a bearing on occurrence of this disease. There is an increased risk with the nonvegetarian diet. High intake of fat especially cooked red meat, leads to production of carcinogens. These have been found to initiate as well as cause progression of prostate cancer. Use of phytoestrogens, vitamin E, selenium and lycopene has been associated with decreased risk of occurrence of cancer prostate. Phytoestrogens act by lowering the testosterone level. Lycopene, vitamin E and selenium probably act by cleaning up the free radicals. Omega-3 fatty acids have also proved beneficial. Vitamin A, green tea, cruciferous vegetables, zinc, vitamin C also probably play a role which is under investigation.

## Objectives & Notes

### Riki Ott, Ph.D.

Alaska Forum for Environmental Responsibility  
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Date of talk: Saturday, June 9, 2007, 2:30pm

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### Training:

Current Job Description:	Independent researcher, lecturer, and author
Medical School/ University Attended	University of Washington (Ph.D.), University of South Carolina (Ms)
Internship:	Commercial fisherm'am in Cordova, Alaska, before, during and after the oil spill
Residency:	21 years in Cordova, Alaska, case study site for the sociology studies
Other Information: (including titles of books or articles you have recently written):	Sound Truth and Corporate Myth\$: The Legacy of the Exxon Valdez Oil Spill (Dragonfly sisters Press, 2005); Not One Drop: The True Story of Promises, Betrayal, and Courage in the Wake of the Exxon Valdez (Chelsea Green Publishing, 2007 fall); Resume posted on <a href="http://www.soundtruth.info">www.soundtruth.info</a>

### SPEECH TITLE: "Sociological Pardigm Shifts after the Exxon Valdez Oil Spill"

At the end of this Presentation, the participant should be able to:

1. Understand relationship of man-made disaster traumas to emotional well being and individual health
2. Understand mitigating measures for man-mad disaster trauma and application to treatments
3. Integrate concepts of man-made disaster traumas in diagnostic work and treatments

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# Sociological Paradigm Shifts after the Exxon Valdez Oil Spill

Riki Ott, PhD

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## Abstract

On March 24, 1989, a technological disaster devastated the natural and social environments of Prince William Sound, Alaska, when the super tanker *Exxon Valdez* grounded and spilled over 30 million gallons of oil. The government-approved industry contingency plans proved inadequate to respond to a spill of this magnitude. Over eighteen years later, this human-caused disaster continues to impact the environment and disrupt the social and economic fabric of communities dependent upon the Sound's living resources. Further, secondary disasters from the cleanup and prolonged litigation created secondary socio-economic impacts that continue to cause ongoing harm to communities, families, and individuals.

The fishing community of Cordova, Alaska, became a case study for the longest running (and still ongoing) study of the social effects of technological disasters. This became the defining work to distinguish technological disasters from natural disasters in terms of characteristics, trauma, and treatment. The *Exxon Valdez* spill occurred during a time when sociologists had begun to realize technical disasters were a “new species of trouble.” In natural disasters (e.g., hurricanes, fires, earthquakes), trauma is short-lived, people work together to rebuild what was lost, and a collaborative community emerges. In contrast, technological or human-caused disasters (e.g., oil spills, chemical releases, and radiation exposures) cause intense, long-term emotional and mental trauma for survivors and tremendous social dysfunction, which can lead to a corrosive community. Methods to treat community-level emotional trauma were found to be different for natural disasters and man-made disasters.

The Cordova study advanced the concept of social capital in maintaining quality of life and functional civilized societies. A working definition of social capital is “connections among individuals—social networks and the norms of reciprocity and trustworthiness that arise from them.” Social capital is largely about relationships built on trust whether among individuals, organizations, governments, states, or nations. When social capital crumbles, such as occurs after man-made disasters, people suffer, communities are paralyzed, and governments stumble. Disaster relief provides ways to mitigate community chaos by rebuilding social capital through treating emotional trauma.

This paper examines the key findings of trauma and treatment of survivors of technological disasters, based on the Cordova study and experience. Five measures of social capital are defined and discussed in terms of individual and community loss, including stress/ trauma, lifestyle/ lifescape changes, recreancy, secondary disasters, and corrosive communities. Application of Peer Listening Circles and collective problem-solving to mitigate trauma by rebuilding social capital is discussed. Finally, in light of linkages between mental and physical health, a case is made to include exposure to technological disasters and litigation as stressors in evaluating patients for post-traumatic stress disorders.

The story of what happened to Cordova in the wake of the *Exxon Valdez* oil spill has been used successfully to “hindcast,” or explain in retrospect, what happened to survivors of the toxic chemical exposure in Love Canal, NY; the Dow Chemical release in Bhopal, India; and the nuclear reactor meltdown in Chernobyl, Ukraine. The Cordova story also carries important insights and lessons for community preparation for, and response to, future technological disasters. Technological disasters have the potential to affect millions of people—often over generations—and they are expected to become increasingly prevalent during the 21<sup>st</sup> century.

## Objectives & Notes

**Klaus-Dietrich Runow, M.D.**

Date of talk: Saturday, June 9, 2007, 3:45pm

Institut for Functional and Enviromental Medicine (IFU)  
Im Kurpark 1  
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### Training:

Current Job Description:	Medical Director: Institute of Functional and Environmental Medicine in Bad Emstal
Medical School/ University Attended	University of Munich and Marburg/Germany
Internship:	Klinikum Kassel - Germany
Board Certifications:	Internat. Board of Environmental Medicine (USA)
Other Information: (including titles of books or articles you have recently written):	Books: <i>Klinische Oekologie (Clinical Ecology)</i> and <i>Nervenschutz durch Entgiftung (Nerve Protection by Detoxification)</i> . First honorary member of the German Society for Environmental and Human Toxicology; The society for Threatened Peoples of Goettingen, Germany, brought me to Kosovo to test for toxic heavy metals in refugee camps near Mitrovica. Received in May 2006 the renowned <i>Environmental Award 2006 (B.A.U.M. Umweltpreis 2006)</i> . Signed by the Federal Minister for Environment (Sigmar Gabriel), Prof.Gege (President of B.A.U.M.) and Uwe Moeller, General Secretary, Club of Rome. B.A.U.M. means: German Society For Ecological Management.

### SPEECH TITLE: “Poisoned Children – Toxic Metal Intoxication in Kosovo”

At the end of this Presentation, the participant should be able to:

1. Estimate the toxic load from a group of more than 500 Roma people living at the edge of the derelict Treпча mines near Mitrovica/North Kosovo with the slag heaps that waft clouds of heavy metal containing dust into the air, water and soil.
2. See which kind of symptoms and diseases developed the IDP (internally displaced people) during the last 6 years.

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## **Poisoned Children – Toxic Metal Intoxication in Kosovo**

Including a video presentation. The Film *Gypsy Blood* from Paul Polansky shows the situation in the polluted refugee camps next to the Trepca Mines in North Kosovo.

Klaus-Dietrich Runow

Speech #1

More than 14,000 Romani homes were looted and destroyed after KFOR troops arrived in 1999. Only a few hundred of those homes have been rebuilt. No one has been allowed to go back to the job they had before the war. Since they were burned out of their homes during the Kosovo war in 1999 a group of more than 500 Roma people living at the edge of the derelict Trepca mines near Mitrovica / North Kosovo. The toxic load comes from the slag heaps that waft clouds of heavy metal containing dust into the air, water and soil.

On 19th October 2005, the Society for Threatened Peoples of Goettingen, Germany, brought me to Kosovo to test for toxic heavy metals in the IDP (internally displaced people) camps near Mitrovica: Zitkovic and Chesmi Lug. Hair samples were collected from 64 persons ( 49 children between the ages of 1-15) and 15 adults. Analytical method: High Resolution ICP-MS (Inductively Coupled Plasma Mass Spectrometry). In addition to Lead we determined other heavy metals, including Aluminum, Antimony, Arsenic, Cadmium, Manganese, Tin etc. and we measured minerals and trace elements – together 39 elements.

The analysis was completed between 28th and 31st October 2005. Results: All analysis showed extreme high levels of Antimony, Arsenic, Lead, Cadmium and Manganese. In only one sample the Arsenic level was in the reference range. 62 hair samples (97%) showed high Aluminum levels, 44 hair samples (69%) high Vanadium levels und 37 hair samples (58%) high Mercury levels.

Regarding Lead the readings range from 20 to 1200 µg/g (Reference Range < 1,0 µg/g). The analysis showed disturbingly low levels of Selenium, which is essential for thyroid and heart function, immune system and for binding and inactivating toxic heavy metals.

Selenium deficiency is associated with cardiovascular disorders. Symptoms from low levels of selenium include arthritis, heart diseases (dilatative cardiomyopathy), hurting muscles, muscle weakness, losing weight, losing hair, changing of hair structure, suppression of the immune system, reduction of fertility and eye diseases. Pediatricians see a connection between low Selenium levels and SIDS (Sudden Infant Death Syndrome).

More specifically the lead levels from the Roma children were:

- 8 Children had readings between 20 - 100 µg/g
- 9 Children had readings between 101-200 µg/g
- 13 Children had readings between 201-300 µg/g
- 4 Children had readings between 301-400 µg/g
- 2 Children had readings between 401-500 µg/g
- 4 Children had readings between 501-600 µg/g
- 3 Children had readings between 601-700 µg/g
- 6 Children had readings between 701-1200 µg/g

***Reference Range of Lead in hair < 1,0 µg/g***

Such high lead level readings are unprecedented in the world and pose an extreme health risk to the children in the camps. Adults are at risk as well especially pregnant women. In 2004 WHO conducted blood tests on several children in the camps after a four-year-old Romani girl died of lead poisoning. All children tested had dangerously high lead levels. WHO recommended immediate evacuation but the people still live in the contaminated area.

In 2005 Kosovo Roma Refugee Foundation followed the pregnancies of 50 Romani women in the three camps. Only six children were born, all suffering from mental retardation. Four other children were still-born. The rest of the pregnancies ended in miscarriage. Medical doctors familiar with the camps and the conditions attributed the miscarriages, still-births and mental retardation to the lead poisoning.

Medical treatment for the first patients out of the refugee camp started in April 2006 in our clinic in Bad Emstal/Germany.

Observations and results regarding the effectiveness of the detoxification therapy will be presented in Speech # 2.

Klaus Dietrich-Runow, Institute for Functional and Environmental Medicine (IFU)  
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**Special thanks** to the Society for Threatened People (GfbV), Goettingen, Germany, [www.gfbv.de](http://www.gfbv.de) : Tilman Zülch, Jasna Causevic, Frank Witte and Paul Polansky who is living in Kosovo and has been struggling since over 6 years for the evacuation of the polluted camps.



## Objectives & Notes

**Cyril W. Smith, Ph.D.**

36 Westminster Road  
Ellesmere Park  
Eccles, Manchester M30-9EA  
U.K.

Date of talk: Saturday, June 9, 2007, 4:15pm

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Email: cyril.smith@which.net

### Training:

Current Job Description:	Retired Physicist and author
Medical School/ University Attended	University of Exeter and University of London
Other Information: (including titles of books or articles you have recently written):	“Frölich’s Interpretation of Biology Through Theoretical Physics,” “ANS Involvement in Chemical and Electromagnetic Sensitivities,” “Quanta and Coherence Effects in Water and Living Systems” and “Watergates – Logic Operations in Water”

### SPEECH TITLE: “The ANS - a Unified Approach”

At the end of this Presentation, the participant should be able to:

1. Appreciate the relation between allergen dilutions and frequency patterns
2. Appreciate the effects of chemicals, frequencies and CAM therapies on the ANS
3. Appreciate the fractal nature of frequency in a living coherent system

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# ANS Involvement in Chemical & Electromagnetic Sensitivities

Cyril W. Smith, Ph.D.

This presentation considers chemicals, frequencies, interactions and stability in the ANS. Tables of measurements demonstrate stimulation and depression of the ANS through connected acupuncture points. These include various therapeutic and nutritional substances and homoeopathic potencies. They are not a summary of clinical trials. The potencies and materials happened to be those readily available.

Meridians and the Voll related parts of the ANS are stimulated by those magnetic field strengths which satisfy proton-NMR conditions at the meridian frequency.

Two tables list some readily available homoeopathic potencies which were found to stimulate the sympathetic and parasympathetic branches of the ANS; potencies stimulating the greatest number of the Voll points were selected to give complete coverage.

The normal condition is for the acupuncture meridian frequencies to fluctuate in a quasi-periodic manner over about a one hour period. If the meridian is synchronized to a stimulatory phase of frequency, its fluctuation rate increases ten-fold. If it is synchronized to a depressive phase frequency, all fluctuations cease, this is analogous to the effect of a toxic chemical.

## Theoretical – Coherence

The presentation concludes with a brief discussion of the relation between coherent frequencies and the ANS. Coherence relates to the constancy of frequency and phase between two or more oscillators which may be represented by molecules, cells, tissues, meridians or an entire living system and is a fundamental property of a quantum field.

The phases of their individual quantum fields and particle numbers are related by Heisenberg Uncertainty Principle. Within a coherent system, the range of the coherence (*coherence length*) becomes the constant quantity instead of the velocity. This makes frequency proportional to velocity apparently without restriction so long as one remains within the coherence length. There can be many velocities each with frequencies in proportion. Because these frequencies no longer have absolute values, the system has become *fractal in frequency*.

Consequently, identical effects can be induced from frequencies in many different parts of the electromagnetic spectrum. It is this which links effects of frequencies characteristic of chemical, technical and biological systems and why environmental frequencies can mimic chemical exposure for hypersensitive patients. For a wave - its constant velocity of propagation equals its frequency multiplied by its wavelength.

A duality exists between chemical structure and frequency patterns - otherwise chemical analysis by spectroscopy would be impossible. Within a coherent system coherence length becomes the constant parameter and frequency becomes proportional to velocity of coherence propagation with no characteristic frequency scale. This implies a *fractal system* with self-similarity, scale invariance and power law. This gives rise to the observed RF and ELF frequency bands coupled to chemical and technological frequencies with implications for the ANS.

In respect of a mechanism for frequencies in water, it is to be noted that there is an increase in pH on imprinting a frequency into water. This corresponds to the removal of H<sup>+</sup> ions and the generation of an equal number of OH<sup>-</sup> ions. The total number of protons involved per imprint is the number required to become coherent to satisfy the local proton NMR conditions and that is completely independent of the imprinted frequency. Thus, imprinting a frequency must create a coherence domain in the protons of the water which stores that frequency. The critical magnetic field for the memory erasure condition supports this.

## Conclusions

The first body system to become compromised in chemical and electrical hypersensitivities is the ANS. Just a few of the many factors which can affect the ANS have been listed. In health, the body will be aware but not incapacitated by them. Voll's connections between the ANS and the acupuncture system have been used to investigate the frequencies involved in these connections. All cells can emit a chemical in response to an electrical signal and an electrical signal in response to a chemical stimulus. Regulatory systems use both frequency and chemical signals to avoid feedback instability. If a reference frequency becomes locked to a frequency or a chemical frequency signature, it cannot respond to metabolic demands and the ANS feedback path will go open-circuit.

The sympathetic ANS frequency corresponds to 300 sec. and the parasympathetic ANS to 3 sec. This implies parasympathetic inhibition is reduced for a 'fight-flee' response.

Frequencies are the fundamental 'bits' in bio-computation. Arithmetic and logic operations on frequencies in water are possible and these are sensitive enough to be 'clocked' by a train of nerve impulses. The phase of coherence allows the possibility of a 'quantum-holographic' memory.

**25th ANNUAL INTERNATIONAL SYMPOSIUM**  
**On Man and His Environment in Health and Disease**  
**June 7-10, 2007 Dallas, Texas**

**Cyril W. Smith, Ph.D.**

**#2. ANS Involvement in Chemical & Electromagnetic Sensitivities**

The following tables of measurements were made by the presenter on himself to demonstrate stimulation and depression of the ANS through connected acupuncture points.

These measurements included various therapeutic and nutritional substances and homoeopathic potencies.

They are not a summary of clinical trials.

The potencies and materials happened to be those readily available.

**Homoeopathic Potencies to Stimulate the ANS**

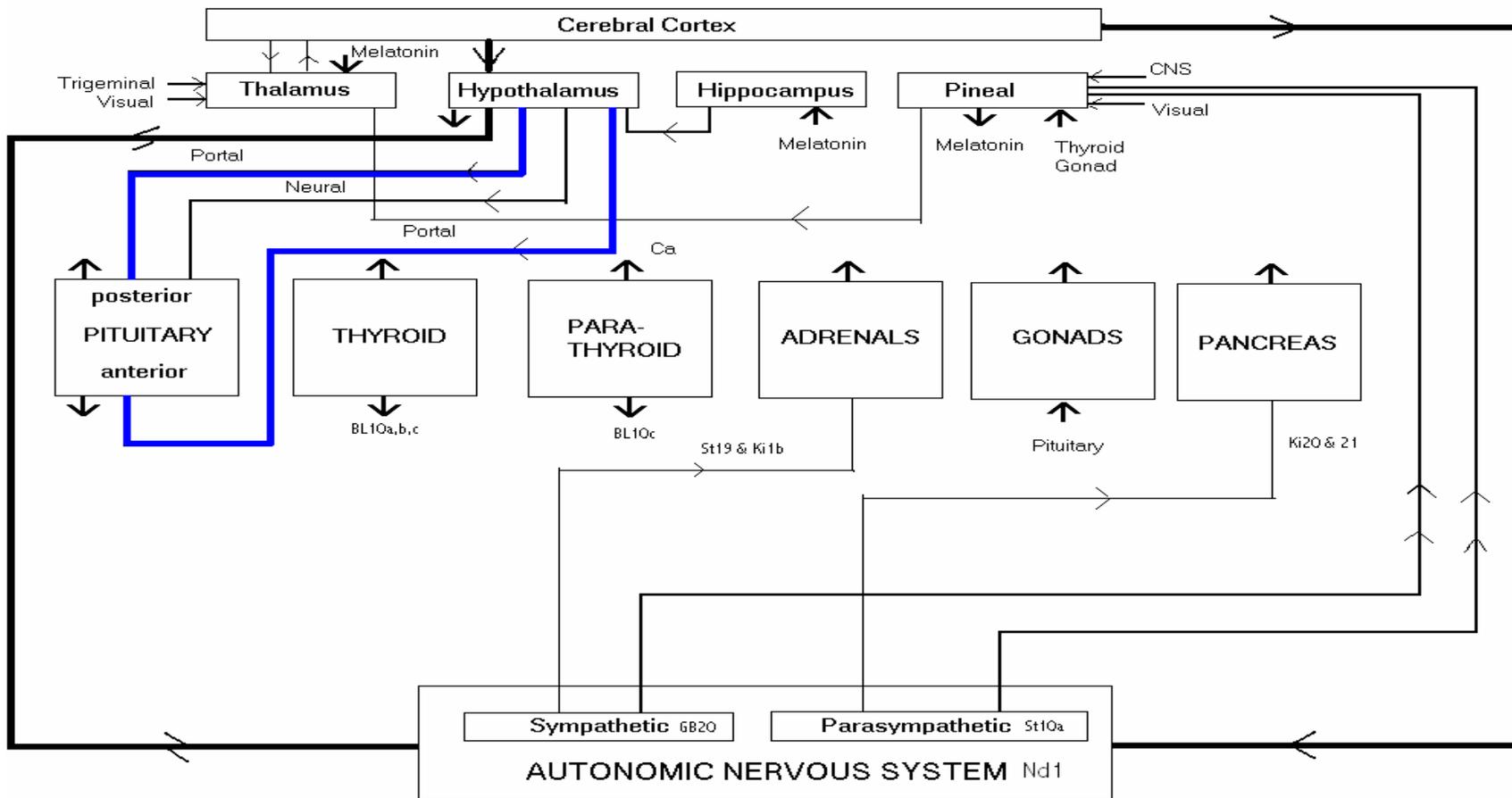
The following two tables list some of the available potencies which stimulated the sympathetic and parasympathetic branches of the ANS. The potencies stimulating the greatest number of the Voll summation points were selected from those readily available.

**In addition to the frequencies of Voll's linked meridian points:**

**Sympathetic ANS linked points carry  $3 \times 10^{-3}$  Hz**

**Parasympathetic ANS linked points carry  $3 \times 10^{-1}$  Hz**

**ELECTROMAGNETIC INTERACTION SITES AND FEEDBACK PATHWAYS**



## Homoeopathic Potencies to Stimulate the Parasympathetic ANS

	Para-sympathetic St10a	Pregang. mid-brain GB 10a	Vagus nucl. in medulla GB 11b	Vagus-cervic./Pharang. plexus St8 c/d	St 16 Vagus-thoracic	Oesoph. plexus St 15	Pulmon. plexus St 18	Vagus – abdom. St21
<b>Arsenicum alb. 1M</b>				x		x		
<b>Graphites 10M</b>	x		x	x				
<b>Cu. met. 6X</b>		x	x		x			x
<b>Carcinosin 200C</b>	x				x		x	x
<b>Phosphorous 6C</b>		x						x
<b>Electricitas 200C</b>	x		x					
<b>Crotolus 6C/12C</b>	x							x
<b>Rad. iod. 200C</b>				x				

## Homoeopathic Potencies to Stimulate the Sympathetic ANS

	Sympathetic GB20	Sympathetic - cranial GB19a	Sympathetic - cervical GV16	Cervical ganglion TW1	Sympathetic - thoracic BL16	Sympathetic - abdominal BL24	Coeliac plexus St44c	Sympathetic - pelvic BL33	Inf. Hypo- gastric plexus BL63
Arsenicum alb. 1M	X	X	X	X		X			
Lycopodium 6C	X	X				X	X	X	
Chamomilla 30C	X	X	X					X	
Ac. fluor. 6C	X	X	X	X					
Crotalus 6C/12C			X		X		X		
Electricitas 200C			X	X		X			
X-ray 200C	X	X					X		
Carcinosin 200C	X	X			X				
CA colon 200C	X	X			X		X		X
Petroleum 30C							X	X	X
Rad. Brom. 1M	X					X			X

## Stimulation of Meridians and ANS by Magnetic Fields

Stimulation when magnetic field satisfies proton-NMR conditions

‘Classical’ Acupuncture Meridians	Point Measured	Low Band Frequency	Resonance Magnetic Field	High Band Frequency	Resonance Magnetic Field
		Hz	Gauss	MHz	Gauss
Lung	Lu1	0.48		24	8.56
Large Intestine	LI1	0.055		2.7	0.96
<b>Stomach</b>	<b>St45 / right</b>	<b>0.044</b>		<b>22</b>	<b>7.85</b>
<b>Stomach</b>	<b>St45 / left</b>	<b>0.44</b>		<b>2.2</b>	<b>0.79</b>
Spleen	Pn1	0.055		2.7	0.96
Heart	He9	7.8		380	137
Small Intestine	SI1	0.025		1.2	0.43
<b>Urinary Bladder</b>	<b>BL67</b>	<b>5.5</b>		<b>270</b>	<b>96</b>
Kidney	Ki1	0.00095		0.047	<b>11.2</b>
Pericardium	Pe9	0.25		13	4.64
Sanjiao (TW)	TW1	6000	<b>1.42</b>	300,000	
<b>Gall Bladder</b>	<b>GB44</b>	<b>0.05</b>		<b>2.46</b>	<b>0.89</b>
Liver	Liv1	4.8		240	8.7
Du Mai (GV)	GV14	4.3		149	53
Ren Mai (CV)	Ren24	14		730	261
<b>‘Extra’ Points</b>					
Anmian I & II	Ex 8 & 9	3,000	<b>0.74</b>		
<b>Extra ‘Ting’ Points</b>					
Lymphatics	Ly1	0.06		2.95	1.05
<b>Nerve Degen.</b>	<b>ND1</b>	<b>0.00055</b>		<b>0.027</b>	<b>6.43</b>
Allergy	AD1	2		98.4	3.51
Organ Degen.	Or1	0.078		3.85	1.37
Fatty Degen.	FatD1	0.74		36	12.90
Skin Degen.	Sk1	0.0035		0.172	<b>40.9</b>
Joint Degen.	JD1	0.3		148	53
Fibroid Degen.	FibD 1	800	<b>0.19</b>	39,400	140
Circulation	Ci9	0.05		2.46	0.89

## Optical Spectra for Mercury Imprinted in Water - Showing Multiple Frequencies Fractal Effect

Hg lines	Optical		Microwave		ELF
nm	Hz		Hz		Hz
	$\times 10^{15}$	$\times 10^6$	$\times 10^6$	$\times 10^6$	$\times 1$
185	1.62		935		19.31
254	1.18		680		14.38
365/6	0.820		472		9.843
405	0.740		425		8.925
436	0.688		396		8.358
492/6	0.607		347		7.235
546	0.549		315		6.633
577/9	0.519		298		6.262
615	0.488		280		5.832
623	0.482		276		5.832
<b>Ratio</b>		1.7340		47.70	
<b>Std. Dev.</b>		$\pm 0.34\%$		$\pm 0.75\%$	

### Theoretical – Coherence

Coherence relates to the constancy of frequency and phase between two or more oscillators and is a fundamental property of a quantum field.

The phases of their individual quantum fields and particle numbers are related by Heisenberg Uncertainty Principle.

Within a coherent system, the range of the coherence (*coherence length*) becomes the constant quantity instead of the velocity.

This makes frequency proportional to velocity apparently without restriction so long as one remains within the coherence length. There can be many velocities each with frequencies in proportion. Because these frequencies no longer have absolute values, the system has become *fractal in frequency*.

Consequently, the same effects can occur in many different parts of the electromagnetic spectrum. It is this which links effects of frequencies characteristic of chemical, technical and biological systems and why environmental frequencies can mimic chemical exposure for hypersensitive patients. For a wave - its constant velocity of propagation equals its frequency multiplied by its wavelength.

Duality exists between chemical structure and frequency patterns - otherwise chemical analysis by spectroscopy would be impossible!

Within a coherent system coherence length becomes the constant parameter and frequency becomes proportional to velocity of coherence propagation with no characteristic frequency scale. This implies a *fractal system* with self-similarity, scale invariance and power law

This gives rise to the observed RF and ELF frequency bands coupled to chemical and technological frequencies.

## **Conclusions**

The first body system to become compromised in chemical and electrical hypersensitivities is the ANS.

Just a few of the many factors which can affect the ANS have been listed. In health, the body will be aware but not incapacitated by them.

Voll's connections between the ANS and the acupuncture system are used to investigate the frequencies involved in these connections.

All cells can emit a chemical in response to an electrical signal and an electrical signal in response to a chemical stimulus. Regulatory systems use both frequency and chemical signals to avoid feedback instability.

If a reference frequency becomes locked to a frequency or a chemical frequency signature, it cannot respond to metabolic demands and the ANS feedback path will go open-circuit. Frequency can effect L/D isomers and control enzyme reactions.

The sympathetic ANS frequency corresponds to 300 sec. and the parasympathetic ANS to 3 sec. This implies parasympathetic inhibition is reduced for a 'fight-flee' response.

The allergy meridian frequency corresponds to a 1/2 sec. response as observed in hypersensitivity.

Frequencies are the fundamental 'bits' in bio-computation and the phase allows for 'quantum-holographic' memory.

## **Bibliography on Electrical Hypersensitivity and Water Phenomena**

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### **Presentations at:**

#### **International Annual Symposia on “Man and His Environment in Health and Disease” held in Dallas, Texas,**

Smith CW, Al-Hashmi SAR, Choy RYS, and Monro JA. *Preliminary Investigations into the Use of Ion-Bombardment Treatments to Improve the Acceptability of Fabrics for Allergy Patients.* 4th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 27- March 2, 1986\*.

Smith CW, Choy RYS and Monro JA. *Electromagnetic Man and His Electromagnetic Environment in Health and disease.* 5th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 26 - March 1, 1987\*.

Smith CW. *The Measurement of Environmental Electromagnetic fields and the Values Effective in Triggering Responses in Hypersensitive Patients.* 6th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 25-28, 1988\*.

Smith CW. *Electricity and Water (Parts 1 & 2).* 7th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 23-26, 1989\*.

Smith CW. *Health and Hazard in the Electrical Environment.* 8th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 22-25, 1990\*.

Smith CW. *1. Electromagnetic Fields and Health. 2. Electromagnetic Fields and Disease.* 9th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 28 - March 3, 1991\*.

Smith CW. *Electromagnetic Fields and the Endocrine System.* 10th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 27- March 1, 1992\*.

Smith CW. *Electrical Environmental influences on the Autonomic Nervous System.* 11th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 25-28, 1993\*.

Smith CW. *The Electrical Aspects of Biological Cycles.* 12th. Intl. Symp. on “Man and His Environment in Health and Disease”, Dallas Texas, February 24-27, 1994\*.

Smith C.W. *1. Basic Bioelectricity; 2. Bioelectricity and Environmental Medicine.* 15th. Intl. Symp. on “Man and His Environment in Health and Disease: focus on the environmental aspects of EMF and bioelectricity”, Dallas Texas, February 20-23, 1997\*.

[\***Audio Tapes** from: Professional Audio Recording, 2300 Foothill Blvd. #409, La Verne, CA 91750, U.S.A.]

Smith C.W. *1. “The Diagnosis and Therapy of EM Hypersensitivity”; 2. “EM Fields in Health, in Therapies and Disease”.* 18<sup>th</sup>. Annual Symposium on Man and His Environment, June 8-11, 2000, Dallas, Texas. Symposium Notes for Participants.

Smith C.W. *1. “Electromagnetic Sensitivity and the ANS”. 2“ANS Involvement in Chemical and Electromagnetic Sensitivities”.* 23<sup>rd</sup>. Annual Symposium on Man and His Environment, June 9-12, 2005. Syllabus pp 162-194.

### Publications 1975- 1998

- Ahmed NAG, Calderwood JH, Fröhlich H, Smith CW (1975) Evidence for collective magnetic effects in an enzyme: likelihood of room temperature superconductive regions. *Phys. Lett.* **53A**:129-130.
- Ahmed NAG, Smith CW, Calderwood JH, Fröhlich H (1976) Electric and magnetic properties of lysozyme and other biomolecules. *Collect. Phenom.* **2**:155-166.
- Shaya SY, Smith CW (1977) The effects of magnetic and radiofrequency fields on the activity of lysozyme. *Collect. Phenom.* **2**:215-218.
- Ahmed NAG, Smith CW (1978) Further investigations of anomalous effects in lysozyme. *Collect. Phenom.* **3**:25-33.
- Aarholt E, Flinn EA, Smith CW (1981) Effects of low frequency magnetic fields on bacterial growth rate. *Phys. Med. Biol.* **26**:613-621.
- Aarholt E, Flinn EA, Smith CW (1982) Magnetic fields affect the lac operon system. *Phys. Med. Biol.* **27**:603-610.
- Smith CW, Aarholt E (1982) Possible effects of environmentally stimulated endogenous opiates. *Health Phys.* **43**:929-930.
- Smith CW, Baker RD (1982) Comments on the paper "Environmental Power-Frequency Magnetic Fields and Suicide". *Health Phys.* **43**:439-441.
- Jafary-Asl AH, Solanki SN, Aarholt E, Smith CW (1983) Dielectric measurements on live biological materials under magnetic resonance conditions. *J. Biol. Phys.* **11**:15-22.
- Smith CW, Al-Hashmi SAR, Kushelevsky A, Slifkin MA, Choy RYS, Monro JA, Clulow EE, Hewson MJC. Preliminary Investigations into Acceptability of Fabrics by Allergy Patients. *Clinical Ecology* **4(1)**: 7-10, 1987.
- Choy RYS, Monro JA , Smith CW. Electrical Sensitivities in Allergy Patients. *Clinical Ecology* **4(3)**: 93-102, 1987.
- Smith CW, Jafary-Asl AH, Choy RYS, Monro JA. *The Emission of Low Intensity Electromagnetic Radiation from Multiple Allergy Patients and other Biological Systems*. In: Jezowska-Trzebiatowska B, Kochel B, Slawinski J, Strek W (Eds.). *Photon Emission from Biological Systems*. Singapore: World Scientific, 110-126, 1987.
- Smith CW, *Electromagnetic Effects in Humans*. In: Fröhlich H (Ed.). *Biological Coherence and Response to External Stimuli*. Berlin: Springer-Verlag, 205-232, 1988.
- Smith CW, Best S. *Electromagnetic Man: Health and Hazard in the Electrical Environment*. London: Dent, 1989,1990; New York: St. Martin's Press, 1989; Paris: Arys/Encre, 1995 (French edition), Bologna: Andromeda, 1997, 1998 (Italian editions).
- Smith CW. *Coherent Electromagnetic Fields and Bio-Communication*. In: Popp F-A, Warnke U, König HL, Peschka W (Eds.). *Electromagnetic Bio-Communication*. Munich, Baltimore: Urban & Schwarzenberg, 1-17, 1989.
- Del Giudice E, Doglia S, Milani M, Smith CW, Vitiello G. Magnetic flux quantization and Josephson behaviour in living systems. *Physica Scripta* 1989;**40**: 786-791, 1989.
- Aarholt E, Jaberansari J, Jafary-Asl AH, Marsh PN and Smith CW. *NMR conditions and biological systems*. In: Marino AA (Ed.) *Modern Bioelectricity*. New York: Marcel Dekker, 75-104, 1990.
- Smith CW, Choy RYS, Monro JA. The Diagnosis and Therapy of Electrical Hypersensitivities. *Clinical Ecology* **6(4)**: 119-128, 1990.
- Smith CW. *Bioluminescence, Coherence and Biocommunication*. In: Jezowska-Trzebiatowska B, Kochel B, Slawinski J, Strek W (Eds.). *Biological Luminescence*. Singapore: World Scientific, 3-18, 1990.

- Smith CW. *Homoeopathy, Structure and Coherence*. In: Schlebusch K-P (Ed.) *Homoeopathy in Focus*. Essen: VGM, 96-104, 1990.
- Smith CW. A Voyager of Discoveries: a tribute to Herbert Fröhlich. *Electromagnetics News* 2(1): 6-7, 1991 [P.O. Box 25, Liphook, Hants GU30 7SE, England].
- Smith CW. *Foreword*. In: Bistolfi F. *Biostructures and Radiation Order Disorder*, Torino: Minerva Medica, vii-xi, 1991.
- Milani M, Del Giudice E, Doglia S, Vitiello G and Smith CW. Superconductive and Josephson-like behaviour of cells. *La Radiologica Medica - Radiol. Med.* **81** (Suppl. 1 al N.4): 51-55, 1991.
- Endler PC, Pongratz W, Smith CW. Effects of highly diluted succussed thyroxin on amphibia development. *Frontier Perspectives* 3(2): 26-28, 1993.
- Smith CW. Biological effects of weak electromagnetic fields. In: Ho M-W, Popp F-A, Warnke U (Eds.). *Bioelectrodynamics and Biocommunication*. Singapore: World Scientific, 81-107, 1994.
- Smith CW. *Electromagnetic and Magnetic Vector Potential Bio-Information and Water*. In: Endler PC, Schulte J (Eds.). *Ultra High Dilution: Physiology and Physics*. Dordrecht: Kluwer Academic, 187-202, 1994.
- Smith CW, Endler PC. *Resonance Phenomena of an UHD*. In: Endler PC, Schulte J (Eds.). *Ultra High Dilution: Physiology and Physics*. Dordrecht: Kluwer Academic, 203-208, 1994.
- Citro M, Smith CW, Scott-Morely A, Pongratz W, Endler PC. Transfer of information from molecules by means of electronic amplification - preliminary results. In: Endler PC, Schulte J (Eds.). *Ultra High Dilution: Physiology and Physics*. Dordrecht: Kluwer Academic, 209-214, 1994.
- Smith CW. Coherence in Living Biological Systems. *Neural Network World* **3**: 379-388, 1994.
- Smith CW. *Sensibilité chez les sujets allergiques*. In: Lannoye P. (Ed.) *La Pollution électromagnétique et la santé*. Paris: Frison-Roche, 79-89, 1994.
- Smith CW. Electromagnetic aspects of biological cycles. *Environmental Medicine* 9(3): 113-118, 1995.
- Smith CW. Measurements of the Electromagnetic Fields Generated by Biological Systems. *Neural Network World* **5**: 819-829, 1995.
- Endler PC, Heckmann C, Laupert E, Pongratz W, Smith C, Senekowitsch F, Citro M. *Amphibienmetamorphose und Information von Thyroxin. Speicherung durch bipolare Flüssigkeit Wasser und auf technischen Datenträger; Übertragung von Information durch elektronischen Verstärker*. In: Endler, PC, Schulte J (Eds.). *Homöopathie-Bioresonanztherapie*. Wein: Wilhelm Maudrich, 127-162, 1996.
- Smith C.W. Nursing the Electrically Sensitive Patient, *Complementary therapies in nursing & midwifery* **3**, 111-116, 1997.
- Smith C.W. Is a living system a macroscopic quantum system? *Frontier Perspectives*, 7(1), 9-15 (1998), (Temple University, Philadelphia, audio tape of 1997 lecture from Frontier Sciences Department). ISSN:1062-4767.
- Senekowitsch F, Citro M, Vinattieri C, Pongratz W, Smith CW and Endler PC. *Amphibienmetamorphose und die elektronische Übertragung von Bioinformation*. In: Endler PC and Stacher A (Eds.) *Niederenergetische Bioinformation*, Wein: Facultas-Univ.-Verlag, 1997, 100-114.

Smith CW. *Coherence in biological systems and water*. In: Taddei-Ferretti C and Marotta P (Eds.) High Dilution Effects on Cells and Integrated Systems, Series on Biophysics and Biocybernetics Vol 3 - Biophysics. Singapore: World Scientific, 1998, pp.88-94. ISBN 981-02-3216-0.

Smith CW. *Water and the diagnosis and treatment of electromagnetic hypersensitivity*. In: Taddei-Ferretti C and Marotta P (Eds.) High Dilution Effects on Cells and Integrated Systems, Series on Biophysics and Biocybernetics Vol 3 - Biophysics. Singapore: World Scientific, 1998, pp.184-192. ISBN 981-02-3216-0.

Smith CW. *Water and bio-communication*. In: Taddei-Ferretti C and Marotta P (Eds.) High Dilution Effects on Cells and Integrated Systems, Series on Biophysics and Biocybernetics Vol 3 - Biophysics. Singapore: World Scientific, 1998, pp.295-304. ISBN 981-02-3216-0.

Endler PC, Heckmann C, Lauppert E, Pongratz W, Alex J, Dieterle D, Lukitsch C, Vinattieri C, Smith CW, Senekowitsch F, Moeller H and Schulte J. *The metamorphosis of amphibians and information of thyroxin storage via the bipolar fluid water and on a technical data carrier; transference via an electronic amplifier*. Schulte J and Endler PC (Eds.) Fundamental Research in Ultra High Dilution and Homoeopathy. Dordrecht: Kluwer Academic, 1998. pp.155-187.

Smith CW. *Coherent frequencies in living systems and homoeopathic medicine*. Proc. 53rd. Congress of the Intl. Homoeopathic Medical League. April 25-29, 1998. RAI, Amsterdam. Paper T001.

Smith C.W. Electromagnetic Therapy. *Positive Health*, Issue 27, pp.26-34, April 1998.

#### **Publications 1999**

Smith CW. Physics and Physicks. *The J. of Alternative and Complementary Medicine* 5(2): 191-193, April 1999.

Smith CW (Section 10 with Griffiths BB and Rea WJ), "The Fröhlich Approach to Cellular Communication Systems". Proc. First World Congress on "Effects of Electricity and Magnetism in the Natural World", Funchal, Madeira 1-6 October, 1998. Pontypool (Gwent): Coghill Research Laboratories (to be published).

Smith CW. (1999) *The Physics of Homoeopathy*. Proc. Intl. Conf. "Improving the Success of Homoeopathy 2: Developing and Demonstrating Effectiveness". London: 15-16 April, 1999. Abstract p. 89.

Smith CW. (1999) Re: Correspondence, *Frontier Perspectives*, 8 August, 1998 By Thomas Phipps. *Frontier Perspectives*, 8(1): 9, Spring 1999.

Smith CW. (1999) *The Physics of Biological and Cognitive Sciences*. Proc. Intl. Multi-Conf. of the Information Society IS'99, Conference on "Biology and Cognitive Sciences" pp.36-39, Ljubljana, Slovenia, 12-14 October, 1999. ISBN 961-6303-18-X

Smith C.W. (1999) *Frequency and Coherence in Water and Living Systems*. Paper presented at a Workshop in Naples, Italy, 11 December 1999 (to be published).

#### **Publications 2000**

Smith C.W. (2000) 18<sup>th</sup>. Annual Symposium on Man and His Environment, June 8-11, 2000, Dallas, Texas. Symposium Notes for Presentations: "The Diagnosis and Therapy of EM Hypersensitivity" and "EM Fields in Health, in Therapies and Disease".

#### **Publications 2001**

Smith C.W. (2001) *Coherent Frequencies and Homoeopathy*, Intl. Conf. Improving the Success of Homoeopathy-3: Reuniting Art with Science, Royal London Homoeopathic Hospital, 22-23 February 2001, p.103, London, UK.

Smith C.W. (2001) *Learning from Water, a Possible Quantum Computing Medium*, 5<sup>th</sup>. International Conference on "Computing Anticipatory Systems", HEC Liège, Belgium, 13-18 August 2001. CASYS'01 Abstracts - Symposium 10, p.19. *Intl. J. of Computing Anticipatory Systems* **13**:406-420 (2002).

Smith C.W. (2001) Distance –related effects near radio and TV transmitters, *Electromagnetic Hazard & Therapy* **11(2-4)**:10-11.

Smith C.W. (2001) Comments on "Quantitative Analysis of Reproducible Changes in High Voltage Electrophotography" by Russo et al. *The Journal of Alternative and Complementary Medicine* **7(6)**: 629-631.

Cardella C, de Magistris L, Florio E and Smith CW. (2001) Permanent Changes in the Physico-Chemical Properties of Water Following Exposure to Resonant Circuits. *Journal of Scientific Exploration* 15(4): 501-518 (2001). Correspondence: 16(2): 256-259 (2002).

### **Publications 2002**

Smith C.W. (2002) Homoeopathy, Acupuncture and Electromagnetism: unlikely (sick) bedfellows, *Homoeopathy in Practice* March 2002, pp.10-14.

Smith C.W. (2002) Toroidal fields may explain mobile phone radiation effects. *Electromagnetic Hazard & Therapy* **13(1)**:6-7.

Smith C.W. (2002) *Effects of Electromagnetic Fields in the Living Environment*. Proc. Intl. Conf. Electromagnetic Environments and Health in Buildings, Royal College of Physicians, London, 16-17 May, 2002. In: Clements-Croome D (Ed.). *Electromagnetic Environments and Health in Buildings*. London: Taylor & Francis, October 2003. Chap. 3, pp. 53-118.  
ISBN 0-415-316-561

Smith C.W. and Best S. *L'Homme Electromagnetique*. (Updated version translated by J-M Danze). December 2002. Embourg, Belgium: Collection Resurgence Editions Marco Pietteur.  
ISBN 2-87211-064-X.

### **Publications 2003**

Smith CW. (2003) *An Alternative Medicine Approach to RF Interactions with Humans*. Conference on: "RF Interactions with Humans: Mechanisms, Exposure and Medical Applications". Institute of Physics, London, 27-28 February, 2003. Abstract ENV 7.4

Smith CW. (2003) Guest Editorial - Straws in the Wind. *Journal Alternative and Complementary Medicine* **9(1)**: 1-6.

Smith CW. (2003) Guest Editorial - Energy Medicine United. *Complementary Therapies in Nursing and Midwifery* **9(4)**:169-175 (Nov 2003).

### **Publications 2004**

Smith, C. W. Book Review of: "Energy Medicine in Therapeutics and Human Performance", James L. Oschman, Butterworth /Heinemann, London, 2003. ISBN 0-7506-5400-7.  
Institute for Complementary Medicine Newsletter, February 2004: journal@icmedicine.co.uk  
and in: *Electromagnetic Hazard & Therapy*, **14(3-4)**: 6 (2004).

Smith CW. (2004) Quanta and Coherence Effects in Water and Living Systems. *J Altern Complement Med*. 10(1); 69-78.

Smith CW. (2004) Correspondence: Dowsing as a Quantum Phenomenon. *Frontier Perspectives*, 13(1): 4-6, Spring/Summer 2004.

### **Publications 2005**

Smith C.W. 1. "Electromagnetic Sensitivity and the ANS. 2. ANS Involvement in Chemical and Electromagnetic Sensitivities. 23<sup>rd</sup>. Annual Symposium on Man and His Environment, June 9-12, 2005. Syllabus pp 162-194.

Smith C.W. (2005) *Watergates – Logic Operations in Water*, 7<sup>th</sup>. International Conference on "Computing Anticipatory Systems", HEC Liège, Belgium, 8 - 13 August 2005. CASYS'05 Abstracts - Symposium 10, p. 9.

Smith CW. *Herbert Fröhlich: A Coherent, Collective Phenomenon*. Centenary Symposium, 20-21 August 2005. International Institute of Biphysics, Station Hombroich, Kapellener Straße, Neuss, Germany, D-41472.

### **Publications 2006**

Smith CW. *Frequencies in Homoeopathy and Acupuncture*. In: Improving the Success of Homoeopathy – 5. "A Global Perspective". Intl. Conf. Royal London Homoeopathic Hospital, 26-27 January 2006. Programme – Poster Abstracts - pp154-155.

Smith CW. (2006) *Electrical Pollution Around Us: does it make the effects of chemical pollution an even greater health hazard?* Anti-Aging Medicine World Congress, CNIT- Paris, March 23-25. Congress Book, Abstract p124.

Smith CW (2006) *Fröhlich's Interpretation of Biology through Theoretical Physics*. In: Hyland GJ and Rowlands P (Eds.) *Herbert Fröhlich FRS: A physicist ahead of his time*. Liverpool: University of Liverpool. pp 91-138.

### **Publications 2007**

Smith CW (2007) *Water - its clinical and scientific depths*. In: Emoto M. (Ed.) *The Miracle of Water*. (in press).

Smith CW. (2007) Editorial: CAM in Camera. *J. of Alternative and Complementary Medicine*. 13(1) 3-4.

## Objectives & Notes

### Professor Tang G. Lee

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### Training:

Current Job Description:	Professor of Building Science and Technology
Current Faculty Appointments:	The University of Calgary, and The University of Manitoba
Medical School/ University Attended	Case Western Reserve University, Cleveland; The Ohio State University, Columbus; Ryerson University, Toronto
Board Certifications:	Registered Architect
Other Information: (including titles of books or articles you have recently written):	Tang Lee has been teaching building science, sustainable design and indoor environmental quality for over 25 years at The University of Calgary as well as Universities in Manitoba, California, Europe and Asia. He conducts comprehensive indoor air quality investigations in an interdisciplinary team for those cases that cannot be solved by others. He is a member of several committees of Health Canada, National Research Council of Canada and the Canadian Standards Association. He has also served as an expert witness in civil and criminal cases in the areas of indoor air quality, building science, architecture, building regulations, construction, general health, and others.

### SPEECH TITLE: **“Beyond 60 Hz EMF to Radio-Frequencies”**

At the end of this Presentation, the participant should be able to:

1. Understand that the available electromagnetic spectrum for wireless communication is nearing saturation
2. Understand the potential health risk as a result of increasing EMF and radio-frequencies in our society.
3. Identify some potential health symptoms resulting from exposure to EMF and RF radiation.

*The American Environmental Health Foundation and the University of North Texas Health Science Center is not responsible for the contents of this presentation. AEHF has not altered or modified the contents of the information provided by this speaker.*

## **Beyond 60 Hz EMF to radio-frequencies up to 100 GHz.**

Robin Ashton, Tang Lee\*, Karen Taylor

\*The University of Calgary

Calgary, Alberta, Canada

### **Goals and objectives**

To demonstrate society is exposed to many more radio frequencies than the 60 Hz EMF from household currents.

To understand that there is potential health consequences of being exposed to the radiation from the ever increasing numbers of wireless communications such as cell phones, WiFi, internet, etc. exposes everyone to higher intensities.

This paper explains radio frequency and electromagnetic field and its potential health impacts. The next paper addresses current and proposed government exposure guidelines and the discussions on attenuation strategies. Awareness of such potential health hazards will help the physician advise their electrical hypersensitive patients on methods to reduce their exposures.

### **1.0 Radio frequency & Electromagnetic Waves**

All of life is made of elements that produce frequency. Frequency represents sound, motion, light, and the physical structure of life. A frequency represents life at rest, life in motion and the potential of energy stored. Frequency is a measurement to determine motion, the potential of motion and the transfer of energy. Waves and their motion are modes of transporting energy and momentum. The measurement of frequency is in Hertz which is the number of waves that pass by a specified point per second.

There is a very broad frequency spectrum that measures radio waves, includes light waves, x-rays, gamma rays, and cosmic rays. The radio frequency spectrum ranges from an Extremely Low Frequency (ELF 3 – 30 Hz) to Extremely High Frequency (EHF 30 -300 GHz) used in radio astronomy and microwave radio relay. Microwave radio relay is the technology that transmits worldwide communication signals for radio, television, telephone, and other digital signalling such as those used on the Internet (data transfer, email and document exchange).

Frequencies produce electromagnetic (EM) waves. These waves are referred to as radio waves, heat rays, and light rays. Common radio waves are emitted from the operation of cellular phones, televisions, radio stations, walkie-talkies and other telecommunication devices. In addition, EM waves are produced from electric motors, ignition systems, gasoline engines, medical equipment, lighting, computers, common household appliances and even various chemicals and their outgassing.

### ***1.2 The Electromagnetic Field***

An electromagnetic field (EMF) is produced from the transfer of energy. An EM field happens when charged particles are accelerated and moved from one point to another in space, it is a “force field” that is capable of producing an action from a distance. An EM field is made up of two components: electricity (E) and magnetism (H). The moving charged particle creates the electrical field and when it stops, the charged particles are not accelerated and create the magnetic field. When particles are accelerated they make light, when they are static they store heat, resulting in magnetism.

Naturally occurring EMF include the earth’s magnetic field, electrical charges in clouds, lightning, volcanoes, earthquakes, rain, and wind. Man-made EMF is the by-product of mining, production, and storage of electricity.

The occurrence of an EMF happens when there is an alternating current in an enclosed area. This occurrence of EM fielding happens in electrical conductors, similar to that of a common household lamp or television set, or in those found in large electrical transformers, or satellite/radio communication towers. It also occurs during the operation of gas and electric motors, engines, and from chemical vapours. The common term to describe the various ranges of radio frequency from all sources is “Electromagnetic Field”.

Radio frequency and its resulting field produce radiation. Radiation is the physical process of transferring heat. Some electromagnetic frequencies are called ionizing frequencies such as those used in x-ray imaging, and are self-propagating. Low electromagnetic frequency produced by small intelligent devices such as cellular telephones produce non-ionizing frequency with measurable heat emission during operation.

## **2.0 Increasing Radio frequency Emissions**

The products, material and devices that emit radio frequency and produce EMF are growing in our technological society. Our society's need for energy, agriculture, and communication is the driving agent of them. All current communication devices such as cell phones, PDAs, computers, hand-held telephones, microwaves and televisions produce EMF and radio frequency. Many household and workplace electronic items have measurable levels of radio frequency. In addition, the energy sources from which we derive electricity such as oil, gas, coal, uranium, and chemicals produce radio frequency.

Radio frequency emissions are caused by three sources: Energy Production, Storage, and Usage. Mining and coal burning plant production. The largest toxic polluter in the country is energy production, with coal being the dirtiest of all fuels. (Clean Air Task Force, 2001) Global satellite communications deployment is growing every year. As energy needs continue to climb, so will the requirement for production and manufacturing infrastructures to support its distribution. The product from operation emissions from all sources is called "electropollution." It contributes to the total electromagnetic radiation emissions from all sources around the world. It is an additional factor creating heat in the atmospheric environment affecting all ecosystems and contributing to climate change. This increase in heat also affects human physiology.

## **3.0 Technology Emission - Intensity**

It has become the sheer number of products, processes, and infrastructure in industrialized society that contributes to its propagation thus contributing to intensity. Undoubtedly, the larger the electrical equipment such as generators, electrical storage facilities, communication towers, wireless networks, and frame relay systems the greater the output of emissions is going to be.

Intensity of radio frequency emission is not only a function of the equipment and technology we use, it is the amount of time we spend in an exposure period. Exposure to radio frequency occurs in the home, workplace, grocery store, and during the operation of a motor vehicle. Exposure occurs at the wireless network "Hotspots" that are encroaching the air space of coffee shops around the world.

Governmental Health & Safety commission and bodies recognize that everyone in society is exposed to some level of "natural background ionizing radiation". This radiation is sourced from: X-rays, gamma rays, alpha particles and beta particles; chemical products such as radon gas, medical diagnostic imaging, industry and the production of electricity. (Great Britain: Health & Safety Commission Executive: Web Statement: Radiation)

In addition to these, there are work place and home exposures to ionizing radiation sourced from petroleum products, mercury gas lighting systems, herbicides, pesticides, biocides, cleaning products and dental products.

### ***3.1 Intensity as a Function of Proximity***

As communities grow they move closer and closer to the sources of electromagnetic fields such as refineries, mining, chemical production such as fertilizer, communication and industrial facilities. Not only does the intensity of radio frequency increase, so does the length of time in which people, families, and communities are exposed. Constant, persistent exposure to radio frequency at any intensity, without cessation, is damaging to all forms of life on this planet, not only human.

As electromagnetic waves travel through space they transfer energy and heat to objects (organic and inorganic mass) within the field. EMF and radio frequency are problematic to the environment as they increase the rate of oxidation and corrosion. Environmental oxidation and corrosion stress cause the weakening of structures and increases the risk of failure.

#### **4.0 Investigating Health Effects from EM Field & Frequency Exposure**

The primary issue about radio frequency exposure is the generation of heat and how that heat affects the human biosphere. The Occupational Safety and Health Department of the Communication Workers of America (CWA) states “As high radio frequency radiation...penetrates the body, the exposed molecules move about and collide with one another causing friction and, thus, heat...If the radiation is powerful enough, the tissue or skin will be heated or burned.” (Earth Talk, 2006)

The World Health Organization, through the International EMF Project is the only international organization with the mandate to determine health impacts from exposure. The focus of the investigation is to determine the risk of cancer and disease by exposure to static electric and magnetic fields (ranging from 0 to 300 GHz) and establish guidelines for exposure.

Private and corporate institutes and bodies produced multiple research reports stating that the impacts of radio frequency exposure are causing serious health complications, disease, morbidity, and death. A 2-hour exposure to a microwave caused the death of brain cells in rats. (Kendal Morgan, 2003) In addition, rat brains exposed to cell phone radiation caused the “...protective barrier in rats' brains to leak, permitting blood proteins that are normally kept away from brain tissue to contact neurons, breach of the so-called blood-brain barrier. (Leif G. Salford, in press)

An Israeli research team found that there *might* be a link between visual damage and microwave radiation. “At least one kind of damage seems to accumulate over time and not heal, challenging the common view and leading the researchers to the assertion that the duration of exposure is not less important than the intensity of the irradiation. The researchers also emphasized that existing exposure guidelines for microwave radiation might have to change. (IsraCast, 2005)”

#### ***4.1 Investigative Findings***

The principal dynamic of how EMF and radio frequencies affect biological systems is understood through thermodynamics. Radiation, or thermodynamics, is the physical transfer of heat. Radiation can be ionizing, self-propagating, and heat generating; or it can be non-ionizing, meaning it will dissipate and disperse. It has no charged particle that can maintain heat.

When heat enters a human body it causes motion in the cells. Moving cells make friction and this friction makes **exothermic** heat. In small, undetected increments the cells begin to heat up.

The biological processes induced by heat transfer are:

- Emulsification
- Dehydration
- Desalination

Combined, these processes have major health impact on human biology and health. Artificially increased body heat at the cellular level changes the way the body produces and manufactures enzymes, constructs proteins and burns complex sugars.

Heat destroys essential fatty acids necessary for the construction of biological enzymes and proteins. As the heat increases, it continues to destroy the fats, there are less essential amino acids, and without amino acids there is no protein construction. Complex protein metabolism interference then occurs with human growth hormone, insulin, myelin, thyroid, secondary sexual hormones like testosterone, estrogens, adrenal and pituitary.

Research about the health impacts of radio frequency using standard epidemiological and toxicological standards have been used for decades. These studies characterize the health response to high radiation exposure and helped set standards for exposure in the workplace and home.

More recent investigative findings are discovering the effects of radio frequency radiation exposure implicated in Alzheimer's Disease. The brain of an 86 year old patient with Alzheimer's Disease found evidence of radon daughters

in different areas of the brain. This indicating exposure to the noble gas radon produced cell damaging high-energy alpha particles. (Molecular Neurodegeneration Journal)

“AD is a complex and progressive brain disease characterized by the failing ability to cope with environmental xenobiotic hazards (Momčilović B, et al, 1999) (Momčilović B, et al, 2001), excessive free radical injury, inflammation and immunity deficiency (Huang J, 1999), cell repair impairment (Kruk PA, Rampino NJ, Bohr VA., 1995), and the protein synthesis (Lee C-K, Weindruch R, Prolla TA, 2000). The ubiquitous environmental RAD exposure, and high RAD accumulation in the sensitive brain structures may either induce or hasten or both the irreversible "shut down" process of the ailing human brain in AD.”

### **5.0 The Emerging Evidence of Electromagnetic Field and Radio frequency Impact on Human Health**

Standards for exposure are being established by a number of governments world-wide based on the studies of biological effects as the epidemiological studies are inconclusive (ELF Working Group, 2005).

Studies are surfacing that show exposure to electromagnetic field, caused by radio frequency emission and by-product and volatile organic compounds (VOC) are having a significant impact on human health (Kavet R, Zaffanella LE, Daigle JP and Ebi KL., 2000).

The difficulty with making definitive correlations showing impact to health from exposure to radio frequency to date is that the transfer agent between ELF and EM fields and disease has not been established. However, many disciplines suspected the transfer agent causing disease and biological affect is that of compound fluoride (Guan ZZ, et al., 2000) ( Reddy GB, et al., 2003) (Rzeuski R, Chlubek D, Machoy Z., 1998) (Susheela AK, Bhatnagar M., 2002), a by-product emission of petroleum energy production, a gas among the greenhouse gases emitted from industry, and a radio frequency found in volatile organic compounds (VOCs).

Studies showing toxicity, oxidation, and thermological damage from exposure to compound fluoride are prodigious. Compound fluoride includes any fluoride in a compound state such as sodium fluoride, thionyl fluoride, uranium hexafluoride, aluminum fluoride, hydrogen fluoride and thousands of others. Industrialized society is being exposed to fluoride compounds and their electromagnetic fields and radio frequency on a daily basis from multiple sources.

### **6.0 Observed Effects of Radio frequency Heat on Human Health**

To understand the effects of ELF on human health, development, and disease it is helpful to think of human anatomy and systems as an interconnection of magnetic and electric fields contained inside a permeable air envelope.

The dense muscular areas of the body being magnetic fields represented by bone, muscle, ligaments, tendons, and the structural formation of the cells. The electrical fields of the body are comprised of flowing fluid. This includes the blood, lymphatic, pericardium, brain, and interstitial fluid both within and surrounding the cells. Electrical exchange within the human anatomy is conducted through ionic channels with acetylcholine and maintained within an electrolyte medium.

Virtually all ionizing radio frequency produces charged particle matter that can be inhaled, ingested, and absorbed. Exposure causes acceleration of electrolyte mediums found in the blood, brain, heart and bone, with principle effect antagonistic to acetylcholine based bioprocesses in the central nervous system, and potassium concentrations in bodily fluid causing depletion and systemic stenosis.

Exposure to radio frequency produces cellular kinetic reactions. The primary physiology effected by radio frequency ionization are the electrolytes calcium ( $\text{Ca}^{2+}$ ), phosphorus ( $\text{PO}_4^{3-}$ ), magnesium ( $\text{Mg}^{2+}$ ), sodium ( $\text{Na}^+$ ), potassium ( $\text{K}^+$ ), chloride ( $\text{Cl}^-$ ), and hydrogen carbonate ( $\text{HCO}_3$ ).

In the human organism, there is a complex balance between the intracellular and extracellular milieu. The thermodynamic balance and regulation of the natural human environment determines the efficiency of hydration, mineralization, ion channel reception, blood pH, and protein synthesis. In addition, the electrolyte balance is critical to the central nervous system for both nerve and muscle function throughout the body.

Ionic channels regulate fluid exchange and transfer of electrochemical information between the structural components of the body, enabling the body's autonomic and peripheral nervous system to respond and stabilize to changes in its environment. The principal dynamics of this exchange are explained by the principle developed by Henry Louis Le Chatelier.<sup>2</sup>

The dynamic of the Le Chatelier Principle defines how a homeostatic system is affected by concentration, temperature, and pressure changes. Le Chatelier's Principle states that when a chemical system at equilibrium experiences a change in concentration, temperature, or total pressure the equilibrium will shift to maintain homeostasis and minimize the change.

When the human organism is exposed to EMF and its radio frequency it causes a change in cellular equilibrium by increasing cellular pressure through increased kinetic heat exchange. The change in pressure and heat changes the concentration of the electrolyte medium, producing gases and water, and further increases in temperature.

Human exposure to ionizing radio frequency causes exothermic stress in human tissue, affecting the tissues concentration, mass and temperature. Exothermic stress affects amino acid synthesis, metabolism of proteins, and neurochemical acetylcholine transfer and synthesis throughout the central nervous system.

The increased exothermic reactions within human tissue after acute and chronic exposure to ionizing radio frequency causes changes in cellular equilibrium, concentration, and total pressure resulting in temperature change increase. Increased thermo-kinetic cellular reaction causes electrolyte medium emulsification, dehydration, and demineralization. The thermal changes in electrolyte homeostasis causes decreases in total body core temperature that affects all metabolic process in human physiology.

The disruption of body thermodynamics causes a loss of water and salt and structural integrity at the cellular level.

### **6.1 Exothermic Effects of Accumulating Radio frequency Exposure on Physiology**

Exposure, acute or chronic, to EM fields and its radio frequency accelerates the electrolyte medium of the human body. Acceleration of the electrolyte chemicals calcium, potassium, phosphate, magnesium sodium, chloride, and hydrogen carbonate causes an increase in kinetic biochemical processes.

The heat that is produced accelerates biological process and diminishes the biological availability of the electrolyte minerals. Potassium regulates fluid balance within cells. It is necessary for cellular enzymatic and electrochemical reactions and homeostasis (equilibrium) of interstitial fluids. When potassium is ionized or depleted through bio-oxidation processes caused by chronic exposure to EMF and radio frequency there is reduction in human nerve transmission, diminished body energy production caused by diminished conversion of stored glucose to glycogen (ATP and cAMP), impaired muscle function, and hormone secretion.

Exothermic reactions caused by radio frequency oxidize calcium stores within the human body and inhibits those processes associated with the electrolyte. When calcium is ionized, or oxidized, critical processes such as calcium blood exchange, blood pressure and pulse, cellular repair, and blood clotting do not occur normally.

Calcium that is accelerated, or ionized, cannot participate in biological processes such as neurotransmission, muscle contraction, heart function, bone development, and blood cell formation, lymphocyte production, haemoglobin production, or oxygen transport.

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<sup>2</sup> <http://www.woodrow.org/teachers/ci/1992/LeChatelier.html>

An example of the biological effects of radio frequency is symptomatic of reduced fertility. This reduction in fertility is caused by the acceleration of calcium signalling impeding calcium oscillations that interrupt the release of inositol (Vitamin B<sub>h</sub>) 1,4,5-trisphosphate (IP<sub>3</sub>). (Developmental Biology, 1999)

Heat, under pressure within the human body, causes kinetic changes to occur in four critical human thermodynamic processes:

- **Deamination** - making of amine groups and amino acids in the liver resulting in diminished enzyme and amino acid production, hormone metabolism, and interruption of glyconeogenesis (formation of glucose in liver).
- **Hydrolysis** - phosphorylation and rate of ATP production and endocrine electrochemical transmission affecting the endocrine organs, lungs, and kidneys. Metabolism of amino acids affecting urea production (ureagenesis) and kidney function.
- **Acetylation** - formation of acetylcholine and associated autonomic and central nervous system function with nerve transmission, muscle contraction, neurological function, and cardiovascular health impairment.
- **Methylation** – regulation of DNA methylation, methyltransferase, and production of cytosine to 5-methylcytosine and glutathione production, impacting gene activity/expression and transcription factors, fetal prenatal and post natal development, neurodevelopment and cognitive impairments, disruption of choline and hepatocyte formation disrupting albumin, fibrinogen, prothrombin (clotting factors).

### ***6.2 How Radio frequency Effects Human Health & Development: The Human Greenhouse Gassing Model***

When human tissue is exposed to EMF and radio frequency it heats up, dehydrates, loses sodium, and over time coagulates. The exothermic process caused by radio frequency exposure produces extreme damage from mitochondrial cellular changes to liver enzyme function, demyelization, and amyloid protein formation in various organs.

In effect, the persistent and consistent exposure to radio frequency causes a condition called **Human Greenhouse Gassing**.

Mass, such as the human body, that is compressed and subjected to increases in heat and pressure combust protein material (Le Chatelier's Principle). Human protein under pressure and heat composts, resulting in changes to the human blood and tissue gases and their concentrations. For example, exothermic events reduce through dissipation nitric oxide causing a loss of Endothelial Relaxing Factor causing coronary artery stenosis and restenosis.

The short term condition of the body with small levels of nitric oxide build-up cause symptoms of nitrogen narcosis and central nervous system response (Dalton's Law).

When human metabolic processes operate at higher temperatures, essential proteins breakdown to form nitrogen, sulphides, hydrogen, phosphates and carbon dioxide gases within the body. Essential fat soluble vitamins A, D, E, and K are emulsified with their corresponding catalytic participation in production of hormones such as myelin is arrested.

Characteristic of human exposure to chronic, long-term exposure to radio frequency is reduced body temperature measuring below 97.4 degrees Fahrenheit. The lower body temperature demonstrates physiological changes in reduction of sodium, potassium, water and other electrolyte minerals. It also demonstrates the loss of participation of the endocrine system in human development and maintenance.

### ***6.3 Symptom Expression of Human Exposure to EMF & Radio frequency***

Acute exposure to EMF and radio frequency is almost always fatal. The standard treatment practice for acute exposure is calcium gluconate, with standard medical practice treatment for burns.

Chronic exposure, defined as low-level exposure that is continuous and constant over duration of time, causes various health ailments ranging from mild to severe, and can cause death (See Table 1 in the Appendix). The symptom presentation of low-level chronic exposure is difficult to ascertain and assess.

## **Conclusion**

As it is still to be absolutely proven there are no health risks, it would behoove doctors and health care providers to caution their patients with complaints that to add some shielding to cellular and battery operated telephones. While the various science fields sort out what is what, it is better to err on the side of caution with the protection of the patients as the priority. The next paper addresses current and proposed government exposure guidelines and a discussion on the difficulty of attenuating radio-frequencies.

## **References**

Clean Air Task Force report on coal “Cradle to Grave: The Environmental Impacts from Coal” (Published: June 2001).

The Environmental Magazine, Earth Talk, October 22, 2006: Effects of Radio Frequencies on Our Health.

Kendal Morgan, Science News Online, reporting a Swedish Research team findings, Feb22, 2003;Vol.163, No 8, p. 115.

Forthcoming paper in *Environmental Health Perspectives* by Dr. Leif G. Salford (Lund University Hospital Sweden) and associates.

IsraCast: Israeli Technology News, Jul. 25, 2005, IsraCast.com.

Published in *Molecular Neurodegeneration Journal* V.1; 2006, PubMed Central, Journal List, September 11, 2006.

Momčilović B, Alkhatib HA, Duerre JA, Cooley MA, Long WM, Harris TR, Lykken GI. Environmental radon daughters reveal pathognomonic changes in the brain proteins and lipids in patients with Alzheimer's disease and Parkinson's disease and cigarette smokers. *Arh hig rada tokiskol.* 1999;**50**:347–369.

Momčilović B, Alkhatib HA, Duerre JA, Cooley MA, Long WM, Harris TR, Lykken GI. Environmental lead-210 and bismuth-210 accrue selectively in the brain proteins in Alzheimer disease and brain lipids in Parkinson disease. *Alzheimer Dis Assoc Disord.* 2001;**15**:106–115.

Huang J, Kim LJ, Mealey R, Marsh HC Jr, Zhang Y, Tenner AJ, Sander Connolly E Jr, Pinsky DJ. Neuronal protection in stroke by an sLe<sup>x</sup>-glycosylated complement inhibitory protein. *Science.* 1999;**285**:595–599.

Kruk PA, Rampino NJ, Bohr VA. DNA damage and repair in telomeres: Relation to aging. *Proc Natl Acad Sci USA.* 1995;**92**:258–262.

Lee C-K, Weindruch R, Prolla TA. Gene-expression profile of the ageing brain in mice. *Nature Genetics.* 2000;**25**:294–297.

Excerpt: Health Effects and Exposure Guidelines Related to Extremely Low Frequency Electric and Magnetic Fields – An Overview, prepared by The ELF Working Group of the Federal-Provincial-Territorial Radiation Protection Committee – Canada, January 2005.

Kavet R, Zaffanella LE, Daigle JP and Ebi KL. The possible role of contact current in cancer risk associated with residential magnetic fields. *Bioelectromagnetics* 2000;21(7):538-553.

Guan ZZ, et al. (2000). Changed cellular membrane lipid composition and lipid peroxidation of kidney in rats with chronic fluorosis. *Archives of Toxicology* 74: 602-8.

Reddy GB, et al. (2003). Antioxidant defense system and lipid peroxidation in patients with skeletal fluorosis and in fluoride-intoxicated rabbits. *Toxicological Sciences* 72: 363-8.

Rzeuski R, Chlubek D, Machoy Z. (1998). Interactions between fluoride and biological free radical reactions. *Fluoride* 31: 43-45.

Susheela AK, Bhatnagar M. (2002). Reversal of fluoride induced cell injury through elimination of fluoride and consumption of diet rich in essential nutrients and antioxidants. *Molecular and Cellular Biochemistry* 234-235(1-2):335-40.

Developmental Biology, 1999 Jul;152:11(2);157-76

## Objectives & Notes

**Donald Hillman, Ph.D.**

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### SPEECH TITLE: “EMF in Homes, Workplaces, and Schools, Part 2”

At the end of this Presentation, the participant should be able to:

1. Recognize that chronic exposure to EMF can produce Electropathic Stress Syndrome in humans.
2. Identify and locate sources of EMF exposure in homes, workplaces, and other environments.
3. Consider circadian blood ANS profiles and micronuclei analyses in diagnosis and treatment of stress syndrome.

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## Objectives & Notes

**Amy Dean, D.O.**

Date of talk: Saturday, June 9, 2007, 5:45pm

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Board Certifications:	American Osteopathic Board of Internal Medicine, American Board of Holistic Medicine

### CASE STUDY: “Vasculitis Responsive to Environmental and Osteopathic Medical Intervention”

At the end of this Presentation, the participant should be able to:

1. Recognize the efficacy of Environmental and Osteopathic Medicine in critical medical conditions.
2. Articulate physical examination methods to assess autonomic nervous system dysfunction and impaired detoxification.
3. Understand the value of osteopathic manipulation in balancing the autonomic nervous system and enhancing detoxification.

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## CASE REPORT

### Vasculitis Responsive to Environmental and Osteopathic Medical Intervention

Amy L. Dean, D.O.

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A 39 year old male presented to Ecological Internal Medicine with dyspnea, rash, fevers, fatigue, hypertension, GERD, and attention deficit disorder. Two years prior he was diagnosed with leukocytoclastic vasculitis unresponsive to immunosuppressive therapy. Over the past two years, he had two episodes of multiple pulmonary emboli. Two months before presentation, he developed proteinuria and hematuria. Prior to presentation, he was in the emergency department once to twice monthly with dyspnea and chest discomfort. Environmental history revealed this patient to be a nurse in the radiology department at the University of Michigan Hospital. Two years ago, a new master bedroom was added to his home using traditional building materials. Seven years ago his home was treated with bendiocarb and chlorpyrifos for spider and termite infestation.

Physical examination revealed oxygen saturation 95%, lower extremity rash, pitting edema, and a failed tandem gait test. Osteopathic examination revealed thoracic and lumbar paraspinal muscle spasm, an indication of sympathetic nervous system dysregulation. Celiac, superior and inferior plexuses were tender with palpation and surrounding tissue was hypertonic, indicating autonomic nervous system dysfunction. Liver motion was restricted. The left kidney was restricted and painful with mobilization.

The patient was started on an organic elimination/rotation diet. Air and water filtration were instituted in the patient's home. Weekly intravenous nutrients and daily oral supplements were prescribed to support immune regulation and detoxification. Osteopathic manipulation was used initially every other week to stabilize the autonomic nervous system and detoxification systems.

The patient reported improved energy with resolution of fevers, proteinuria and hematuria 6 weeks after initiating treatment. The skin rash caused by vasculitis resolved four months from starting treatment and the patient began tapering off prednisone. The patient was able to discontinue warfarin and prednisone fourteen months after initiating treatment at which time he noted significant improvement in fatigue. He continues to be in remission from vasculitis, exhibits no dyspnea, fevers, GERD, hypertension or attention deficit disorder. With the exception of one hospital admission three months after starting treatment, the patient has not been to the emergency department or been admitted to the hospital in the past 21 months.

#### References:

Barral JP, Mercier P. 1988 *Visceral Manipulation*. Seattle, WA. Eastland Press.

Kuchera WA, Kuchera ML. 1994 *Osteopathic Principles in Practice*. 71-80. Columbus, OH. Greyden Press.

Pischinger A 1991 english ed. *Matrix and Matrix Regulation: Basis for a Holistic Theory In Medicine*. Brussels. Haug International.

Rea WJ. 1997 *Chemical Sensitivity: Volume 4*. Boca Raton, Fl. Lewis Publishers.

**25<sup>th</sup> ANNUAL INTERNATIONAL SYMPOSIUM  
ON MAN & HIS ENVIRONMENT  
Schedule**

**Sunday, June 10, 2007**

8:15 **ANNOUNCEMENTS/MODERATOR:** Doug Seba, Ph.D.

8:30 Russel J. Reiter, Ph.D., "The Use of Melatonin to Protect Against Toxic Agents"

8:50 Q & A

9:00 Klaus-Dietrich Runow, M.D., "Detoxification of Heavy Metal Intoxication in Children"

9:20 Q & A

9:30 Kaye H. Kilburn, M.D., "Mold/Mycotoxins Impair Neurobehavioral Function"

9:50 Q & A

**10:00 BREAK**

10:30 Jon Pangborn, Ph.D., "Biochemistry - Gone - Wrong in Autism: Some Remedies"

10:50 Q & A

11:00 Mohamed B. Abou-Donia, Ph.D., "Autoantibodies against Nervous System Proteins as Biomarkers for Brain Injury"

11:20 Q & A

11:30 Lisa Nagy, M.D. "Why Horse Women are Crazy - Mycotoxicosis"

11:50 Q & A

12:00 Doris Rapp, M.D., "The Fetus to Adult, Good, Bad, and Ugly in Medicine"

12:20 Q & A

12:30 Jesus Suarez, M.D., "Young Man Dizziness Treated with Immunotherapy"

12:50 Q & A

**1:00 SUMMARY AND CLOSE:** Doug Seba, Ph.D.

**SUNDAY, JUNE 10, 2007**

**ABSTRACTS**

**AND**

**HANDOUTS**

## Objectives & Notes

### Russel J. Reiter, Ph.D.

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Current Faculty Appointments:	Professor University of Texas Health Science Center
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### SPEECH TITLE: "The Use of Melatonin to Protect Against Toxic Agents"

At the end of this Presentation, the participant should be able to:

1. Discuss the role of free radicals in the negative metabolic effects of toxins, including drugs
2. Understand the mechanisms of melatonin as a ubiquitously – acting antioxidant
3. Consider the use of melatonin as a treatment or co-treatment in conditions of toxin exposure.

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## The Use of Melatonin to Protect against Toxic Agents

Russel J. Reiter, Department of Cellular and Structural Biology, The University of Texas Health Science Center, 7703 Floyd Curl Drive, San Antonio, Texas, 78229-3900, E-mail: Reiter@uthscsa.edu

The goals of this presentation are a), to describe the mechanisms by which melatonin scavenges toxic free radicals and b), to illustrate how toxic substances contribute to degeneration of the central nervous system. Of particular interest in this report are destructive changes in the brain that lead to Alzheimer's disease and Parkinsonism.

Most toxins are destructive to the central nervous system (and other tissues) because they generate free radicals and related substances. Many of these toxic agents are derived from oxygen; about 1-4 % of the oxygen that is inhaled eventually ends up as partially reduced species that damage essential intracellular molecules.

The brain is highly vulnerable to free radical attack for several reasons. Firstly, although the brain is only 2% of the body weight, it utilizes 20% of the inhaled oxygen. Thus, proportionally the brain gives rise to more free radicals than any other tissue. Also, the brain contains large amounts of fatty acids which are easily oxidized. Despite the fact that the brain is highly susceptible to mutilation by derivatives of oxygen, it is not very well protected against them, i.e., the brain has a relatively deficient antioxidative defense system. For these reasons the brain exhibits many age-related changes, i.e., memory loss, that are a result of continual mangling of essential molecules in neurons and glia by free radicals and related reactants.

Classic examples of neurodegenerative diseases that are, in part, mediated by free radicals include Alzheimer's disease (AD) and Parkinsonism (PD). One of the major toxic agents that contribute to AD is amyloid  $\beta$ -peptide ( $A\beta$ ). Neural deposits of  $A\beta$  derive from amyloid precursor protein which circulates in the blood. When this molecule enters the extracellular space of the brain two enzymes, the secretases, "cut out"  $A\beta$  from the larger amyloid precursor protein. In the intracellular space,  $A\beta$  deposits near neurons are destroyed as  $A\beta$  generates free radicals. The resulting molecular damage to the neurons causes them to die. Once lost, brain cells do not generally regenerate so that the persistent loss of neurons over time leads to dementia.

Melatonin is highly effective in limiting the deposition of  $A\beta$  in the brain and when deposits do form the resulting radicals and radical products that are generated are immediately scavenged by melatonin if it is available.

A second major feature of the AD brain is the intracellular neurofibrillary tangles (NFT). NFT are produced in neurons when kinases phosphorylate the cytoskeletal protein, tau. Once formed, NFT are deadly to cells because they generate free radicals that are detrimental to cellular function. Melatonin inhibits the kinases that phosphorylate tau and thus reduces NFT formation. If NFT do form, melatonin scavenges the radicals they generate. Clearly, melatonin seems an important agent to prevent oxidative stress to the brain that is associated with AD. The indoleamine reduces two of the major features that are known to be associated with AD.

Another neurodegenerative condition with high free radical production is PD. PD results due to loss of the dopaminergic neurons in the substantia nigra. A toxin which is known to cause PD in humans is MPTP. It can also be used to produce PD-like symptoms in animals including destruction of dopaminergic neurons. When given to animals, MPTP is taken up by brain cells and metabolized to  $MPP^+$ .  $MPP^+$  then enters dopaminergic neurons where it kills them via free radical mechanisms thereby producing signs of PD. Since MPTP toxicity is a result of free radical damage, melatonin readily neutralizes the toxicity of  $MPP^+$  and reduces the severity of PD in animal models of this condition.

Collectively, to date all experimental and limited clinical data indicates that exogenously administered melatonin may be effective in forestalling the onset of AD and PD. Interestingly, these conditions develop late in life, a time at which endogenous melatonin production in humans is greatly attenuated.

## Objectives & Notes

**Klaus-Dietrich Runow, M.D.**

Date of talk: Sunday, June 10, 2007, 9:00pm

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Board Certifications:	Internat. Board of Environmental Medicine (USA)
Other Information: (including titles of books or articles you have recently written):	Books: Klinische Oekologie (Clinical Ecology) and Nervenschutz durch Entgiftung (Nerve Protection by Detoxification). First honorary member of the German Society for Environmental and Human Toxicology; The society for Threatened Peoples of Goettingen, Germany, brought me to Kosovo to test for toxic heavy metals in refugee camps near Mitrovica. Received in May 2006 the renowned <i>Environmental Award 2006</i> (B.A.U.M. Umweltpreis 2006). Signed by the Federal Minister for Environment (Sigmar Gabriel), Prof. Gege (President of B.A.U.M.) and Uwe Moeller, General Secretary, Club of Rome. B.A.U.M. means: German Society For Ecological Management.

### SPEECH TITLE: “Detoxification of Heavy Metal Intoxication in Children”

At the end of this Presentation, the participant should be able to:

1. Estimate the first results and effectiveness of the detoxification treatment regarding the first group of highly lead contaminated children out of the refugee camp in Mitrovica/Kosovo. The therapy took place in summer 2006.

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## **Detoxification of Heavy Metal Intoxication in Children - Lead Poisoning in a Refugee Camp in Kosovo -**

Klaus-Dietrich Runow<sup>1</sup>, Klaus Michael Weber<sup>2</sup>  
Speech # 2

Since they were burned out of their homes during the Kosovo war in 1999 a group of more than 500 Roma people living at the edge of the derelict Trepca mines near Mitrovica / North Kosovo. The toxic load comes from the slag heaps that waft clouds of heavy metal containing dust into the air, water and soil. Over 30 people died during the last six years. Medical investigations showed extreme high levels of heavy metals. In 2004 WHO conducted blood tests on several children in the camps after a four-year-old Romani girl died of lead poisoning. All children tested had dangerously high lead levels. Although the WHO recommended immediate evacuation the people still live in the polluted area.

Medical treatment for the first highly contaminated patients out of the refugee camp started in April 2006 in our clinic in Bad Emstal/Germany. The lead level in the hair of the 7-year-old boy Denis was 1.200 times over the reference range. He has been treated together with his father and sisters. The effectiveness of the detoxification program has been verified by Spiroergometry Tests. After a 4 week period in a safe environmental, organic food and a treatment with i.v. Glutathion, Alpha-Lipoic Acid, Coenzym Q10, Vitamins and Minerals the oxygenutilisation in the cells was one third higher than before. This was surprising because for achieving such an increase of oxygenutilisation a sportsman needs a one year continuous daily training. Hair, blood and urine tests showed a quick improvement – some went to normal. After 5 months we observed a decrease of the lead levels in the erythrocytes up to 70 % and in hair samples up to 90%. Blood test results and urine organic acids improved in most of the individuals.

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### **Special thanks :**

Society for Threatened People (GfbV), Goettingen,Germany, www.gfbv.de : Tilman Zülch, Jasna Causevic, Frank Witte and Paul Polansky who is living in Kosovo and has been struggling since over 6 years for the evacuation of the polluted camps.

The Detoxification therapy has been supported through the program “A heart for children” from BILD hilft e.V. (a non-profit organization connected to the German newspaper BILD).Special thanks: Frau Martina Krueger + Frau Hildegard Kottusch.

Medical Analysis have been supported by US- Laboratories: Doctors Data, Genova Diagnostics and Metametrix.  
Medical Treatment has been supported by Felix Henrichs from Supplementa B.V., Winschoten, Netherlands.

## Objectives & Notes

**Kaye H. Kilburn, M.D.**

Date of talk: Sunday, June 10, 2007, 9:30am

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Internship:	Western Reserve Hospitals – Cleveland
Residency:	University of Utah: Medicine and pathology, Duke: cardiopulmonary, London: cardiology
Board Certifications:	Am Board Internal Medicine, Am Board Preventive Medicine, occupational Health
Other Information: (including titles of books or articles you have recently written):	“Chemical Brain Injury,” NY: John Wiley 1998; “Mold and Mycotoxins,” editor Washington DC: Heldref 2004; “Endangered Brains” Princeton Scientific Press 2004

### SPEECH TITLE: “Mold/Mycotoxins Impair Neurobehavioral Function”

At the end of this Presentation, the participant should be able to:

1. Mold/mycotoxin effects on neurobehavioral functions are indistinguishable from the effects of other chemical brain toxins
2. Spontaneous recoveries of functions are rare but sulfa hydral and redox therapies are helpful
3. Mold/mycotoxins cause movement disorders that may imitate multiple sclerosis, Alzheimer’s, Parkinson’s disease, stroke and muscle weakness
4. Rare associations are with chorioathetosis, transverse myelitis, nodularthyroiditis - hypothyroidism

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# MOLD ASSOCIATED IMPAIRMENT OF BRAIN AND LUNG IN CHILDREN AND MOTHERS

**Kaye H. Kilburn, M.D.**

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## **Abstract**

### **Background**

Children in schools and homes where visible or odorous mold has been found had burning eyes and throats, mucous membranes airways and skin, fatigue and decreased memory, concentration, attention and poor performance in schools that raised the question were they neurobehavioral impaired. Their, mothers had similar symptoms.

### **Objective**

First; to determine whether children from moldy homes had decreased performance on standardized neurobehavioral and pulmonary tests compared to referents. Second; to compare impairment in children and their mothers.

### **Design**

Children with symptoms from mold exposed homes were compared to unexposed children and to their mothers.

### **Patients and Methods**

Thirty-five children, 19 girls and 16 boys, age 6-16 (a convenience sample) had neurobehavioral performance compared to 163 unexposed children. Measurements were balance, reaction time, color, grip, visual fields, hearing, blink reflex, and vibration, verbal recall, problem solving, vocabulary, peg placement, and trail making A and B, fingertip numbers and long term memory. For a nested match 29 children were compared to their 24 mothers. Mold exposures were confirmed by hypha and colony morphology of samples from indoor air and surfaces and cultures. Measurements of mold exposed children were compared to referent children from a community without mold exposure by using prediction equations for each test. Abnormal scores that were defined as outside the confidence intervals of predicted score's were counted for exposed and unexposed children.

### **Results**

The mean abnormality score of 2.6 for the 35 was different from the mean of 0.9 in unexposed children  $p < .0001$ . Mean values for nine performance scores differed statistically significantly from unexposed children's scores (seven were below predicted): choice reaction time, balance with eyes open and with eyes closed, and blink reflex latency right and nearly on the left, verbal recall immediate and delayed, and digit symbol substitution. Exposed children exceeded unexposed groups on problem solving (Culture Fair) and similarities. Mother's abnormality scores were 7.0 with a ratio to children of 2.7:1. Children were less impaired than mothers on all functions. For peg placement, an executive function, and for cognitive and long term memory where children scores exceeded predicted values, differences from mothers were reversed. Children's vital capacities were significantly reduced and 80% had airways obstruction.

### **Conclusion**

Children from moldy homes noted excessively frequent symptoms. They had hyperactive airway disease. Neurobehavioral abnormalities were 3 fold increased and mean performance scores were decreased compared to unexposed children.

## Objectives & Notes

### Jon Pangborn, Ph.D.

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Board Certifications:	Fellow, Am. Institute of Chemicals
Other Information: (including titles of books or articles you have recently written):	Autism: Effective Biomedical Treatments, J. Pangborn, and S. Beker; Co-Founder: Defeat Autism Now! (DAN!) Organization

### SPEECH TITLE: “Biochemistry-Gone – Wrong in Autism: Some Remedies”

At the end of this Presentation, the participant should be able to:

1. Appreciate that autism is a disease of the body and the brain that includes oxidant stress and inflammation.
2. Understand that autism is a persistent maladaptive state arising from a conflict among epigenetics, toxicity, infection and broken biochemistry.
3. Realize that various remedies can have some success in decreasing or eliminating autistic traits.

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# BIOCHEMISTRY-GONE-WRONG IN AUTISM – SOME REMEDIES

## ABSTRACT

Jon B. Pangborn, Ph.D., F.A.I.C.

Most cases of autism are treatable with potentially significantly positive outcomes. That's because the underlying metabolic problems that most have are due to –

- Acquired toxicities and infectious agents that can be remedied,
- Epigenetic changes that can be reversed by removal of the instigating stressors,
- Genetic traits that become subclinical conditions after toxic and infectious stressors are removed.

Epigenetic changes are changes to gene expression set by methylation. For further information on this, please refer to my other symposium presentation: “Perpetuation of Inflammation by Epigenetic Influences”.

In the 1960s, the incidence of autism was reported to be two or three per 10,000 births. Numerically, three in 10,000 is equivalent to 0.0003. In 2004-5, the US CDC reported an incidence of sixty in 10,000, or 0.006; this is a 20x increase over that of 1960. (Now, 2007, one in 150 or 67 per 10,000 is thought to be the incidence – CDC, Feb 2007.) Admittedly, some of this, perhaps a factor of 2, is due to expansion of the diagnostic and categorization criteria for autism. That still leaves an increase of >10x over a span of only two generations. This is absolutely uncharacteristic of disorders that are primarily genetic. (Genetic disorders have a nearly consistent incidence for a given population or ethnic group.) Hence, the autism we see today (60 to 67 in 10,000) must have a very strong acquired component to its etiology.

We still have a small percentage (<5%) of the total autistic population whose traits are associated with severe inborn errors of metabolism: creatine formation/transport deficiencies, adenylosuccinate lyase deficiency, Angelman syndrome, Fragile X, Rett syndrome, neurofibromatosis, tuberous sclerosis, etc. Biomedical treatments may help the quality of life and cognitive abilities of some of the metabolic-error patients. But it is unlikely that their autistic traits can be reversed with our present intervention capabilities.

Scientists and clinicians collaborating with DAN believe that neuronal network connectivity is disordered in autistic brains. One reason this happens is that the energetics and synchrony of network operation became deficient for periods of time between ages one and two years. This is when interneurons make connections during the brain's self-assembly process. Lack of connectivity of networks makes integration of sensory information (vision, smell, touch, sound, etc.) difficult, and it makes responsive, expressive speech nearly impossible.

One metabolic process that's responsible for energy delivery and synchrony is methionine metabolism. S-adenosylmethionine, “SAM”, forms the energy carrier creatine by methylation. SAM also methylates phospholipids adjacent to the dopamine D<sub>4</sub> receptors, many of which are in interneurons. Brain D<sub>4</sub> receptors are the “attention getters” – they perceive change and relay the information to concerned networks. Methionine metabolism is additionally tasked with antioxidant duty. It leads to cysteine, which, together with glycine and glutamate, makes glutathione, “GSH”. And, methionine metabolism helps detoxication – by methylation (antimony, phenols, catechols, histamine, etc.), and because GSH attaches to toxicants (including mercury) and to wastes to make excretable forms (“mercapturates”). Another related detox mechanism is sulfation (cysteine provides sulfite which becomes sulfate). All these essential tasks depend upon various aspects of methionine, sulfur and methyl group chemistry.

Autism can happen if subclinical impairments associated with methionine metabolism are propelled into major problems by stressors. Subclinical impairments of this type include:

- Suboptimal handling of adenosine
- Folate “traps”
- Limited cobalamin (vitamin B12)
- Rate-limited or phenotypical variants of transmethylase enzymes
- Glutathione transferase or GSH oxidation-reduction cycling problems
- Reduced activity of certain detoxication enzymes/processes

Stressors include: mercury, antimony, lead, arsenic, organophosphate chemicals, phenolics, possibly phenylpyridinium xenobiotics, and paramyxoviruses. Paramyxoviruses include measles and mumps. A persistent measles infection in the brain can stop phospholipids methylation in cells just as drastically as can inhibited SAM-transmethylase enzymes.

Once initiated, autism's persistence is due to two circumstances. One is cell-sequestered toxicants and infectious agents (e.g. measles), which cannot be removed due to dysfunctional detoxication processes and immune dysregulation. The second circumstance is epigenetic changes to enzymes that are brought about by persistent inflammation caused by infections or toxicants. Epigenetic changes result in enzymatic downregulation of methylation and futile upregulation of stalled or inoperable sulfur chemistry. For more than two decades, cyst(e)ine has been reported to be subnormal in 60-70% of autistics. This and excessive oxidized glutathione/active glutathione (GSSG/GSH) ratio persists until appropriate interventions relieve stressor influences and improve the kinetics of methionine becoming cysteine, cysteine becoming glutathione, and SAM doing its methylation duties.

Intervention begins with removal of dietary-source stressors, treatment of gastrointestinal issues, and bolstering basic nutritional status. Often, this starts the process of detoxication as opiate-acting peptides (products of maldigestion) and dysbiotic bowel flora are reduced or eliminated. Medical intervention often is necessary with use of antifungals and antibiotics followed by probiotic supplements. Anti-inflammatory interventions, both nutritional (vitamins C, E, beta-carotene, melatonin, etc.) and medicinals can be beneficial. Full-blown detoxification treatments should await resolution of diet, nutrition and gastrointestinal issues. Metabolic remedies can involve methylcobalamin, folic acid, trimethylglycine, creatine, and other nutrients and cofactors. Antiviral medications and narcotic antagonists (to counter exorphin peptide effects) have been tried with some success and in conjunction with appropriate nutritional supplements.

#### ACKNOWLEDGEMENT AND INFORMATION SOURCES

This presentation reflects the experience of DAN! (Defeat Autism Now!), of which Dr. Pangborn is a cofounder (1995). DAN! Is a cooperative alliance of clinicians, scientists and parents, operated under the auspices of the Autism Research Institute (ARI). A DVD, "Recovered Autistic Children" is available (free) from ARI, 4182 Adams Avenue, San Diego, CA 92116); it won a Gold Remi Award at the 2005 International Film Festival in Houston. Source references for this presentation and additional information are contained in the book: Autism: Effective Biomedical Treatments, J. Pangborn and SM Baker, Sept 2005, available from ARI and Amazon.com.



## Objectives & Notes

### Mohamed B. Abou-Donia, Ph.D.

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Other Information: (including titles of books or articles you have recently written):	Editor of the book "Neurotoxicology," published more than 320 papers.

### SPEECH TITLE: "Autoantibodies Against Nervous System Proteins as Biomarkers for Brain Injury"

At the end of this Presentation, the participant should be able to:

- 1.
- 2.
- 3.

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**Autoantibodies Against Nervous System Proteins as Biomarkers for Brain Injury. Mohamed B. Abou-Donia, Department of Pharmacology and Cancer Biology, Duke University Medical Center, Durham, NC**

Because of exposure of some individuals to chemicals such as pesticides and airplane cabin fumes, results in neurological deficits, development of objective and reliable methods that will permit quantification of brain injury and neuropathological processes has become increasingly important. Our recent studies have demonstrated that autoantibodies against some brain-derived proteins may be clinically useful as biological markers of neurological deficits in caused by exposure to chemicals. The autoantibodies that were quantified in sera of patients were against the (1) axonal-specific neurofilament heavy protein (NFH), Tau protein, tubulin, and myelin basic protein (MBP); (2) dendrite-specific microtubule-associated protein-2 (MAP-2); and (3) astrocyte-specific glial fibrillary acidic protein (GFAP), and S100 protein. The results show that increased levels in patient's sera of autoantibodies against the neuronal markers (NFH, Tau protein, tubulin, MAP-2, and MBP), as well as GFAP (a marker for astrogliosis), correlate well with the diagnosis of neurological disorders following exposure to pesticides. Increased autoantibodies against NFH, Tau, tubulin, and MBP are biomarkers for axonal degeneration in regions such as the cerebral cortex, leads to motor and sensory abnormalities, ataxia, weakness, and loss of strength. Damage to the hippocampal circuitry leads to learning and memory deficits. Increased autoantibodies against MAP-2 indicate degenerative alteration of the dendrite-rich Purkinje cells of the cerebellum, resulting in gait and coordination abnormalities. Neuronal degeneration of the limbic system, corticofugal system, and central motor system (associated with mood, judgment, emotion, posture, locomotion, and skilled movements) results in psychiatric disorders. Our finding of increased autoantibodies against GFAP is consistent with previous reports that individuals with neuropsychiatric disorders have elevated levels of GFAP. Because injured areas of the brain do not regenerate, these symptoms are expected to persist for a long time.

## Objectives & Notes

**Lisa Nagy, M.D.**

Date of talk: Sunday, June 10, 2007, 11:30am

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### SPEECH TITLE: “Why Horse Women are Crazy - Mycotoxicosis”

At the end of this Presentation, the participant should be able to:

1. Evaluate patients by history, physical for mycotoxin exposure at home, work and play
2. Perform an easy work up and environmental testing for mold and mycotoxins.
3. Advise patients how to safely ride horses and avoid mycotoxin exposure.

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## Why Horse Women Are Crazy – Mycotoxicosis

Yes I am a ‘horse woman’. Hence my interest in this topic. Everyone knows that women who spend their lives around horses are a little ‘wacky’. We all think it is endearing and part of why they became involved in this obsessive way with horses in the first place. Actually I think it is quite the opposite. I believe and hope to illustrate that this neurocognitive and behavioral abnormality has at least one discernable cause – that is preventable in part. In order to have riders interested in protecting themselves from exposure to mycotoxins that are emanating from their horses we first have to have proof that there is a causal link between the exposure and the behavioral and other symptoms.

I use my story of exposure to mycotoxins within my home, the subsequent development of; adrenal insufficiency, dysautonomia, apparent ADD and depression -- if not hypomania, mitochondrial myopathy as well as severe chemically and electrical sensitivity to set the stage of my ‘discovery’. After being diagnosed and treated I returned from the Environmental Health Center of Dallas and rode my horse. I had immediate symptoms when I removed the ‘new’ sweaty horse pad from the animal and realized the same toxin that my body had developed an aversion to was also, coincidentally in my horses sweat. I reasoned that it would be in the urine stool and dust that we brush off the horses as well.

I also know that many of my friends and riding instructors had developed sensitivities to chemicals, allergies, mold intolerance, as well as the usual personality traits that apparently go along with prolonged riding careers. I was thinking that if the mycotoxins were a cause of chemical sensitivity in such a large proportion of the patients in Dallas then this must be a possible cause of ill health in people who work closely with animals. Routes of exposure could be inhalation and transdermal, as ingestion and intravenous were not a problem. Having discussed this with colleagues who do research on mycotoxins I learned that it is known that cattle farmers, and those who raise horses and llamas have higher levels of mycotoxins in their urine – presumably because of their proximity to the higher levels of molds in the barns etc.

I thought the practice of washing the horse pads and blankets – which are covered not only with sweat, but also, urine and manure -- must lead to a higher level of mycotoxins in the people sharing the laundry. This could be in one’s home or in a public Laundromat, a communal condominium facility. In fact many ‘mold patients’ can tell that the average Laundromat has a sometimes intolerable level of mycotoxins in the dryers. The reason behind this is two fold: other people who have moldy homes and clothes have used these machines repeatedly over the years and people have washed pet and horse laundry at some point in many of the machines so they don’t have to wash them at home. Unfortunately dryers hold on to their toxins in the drum whereas washers are more easily rinsed with ammonia (clinical observation only) and left clean.

I will present data illustrating the positive Trichothecene mycotoxins levels in people and horses. I will present the results of a pilot study of riders who wash pads at home and their household inhabitants who do not ride but share the laundry. These people must feel confident that they have not recently (20 years) or currently had a moldy home or workplace that would complicate results. Ideally the riders who do not wash the pads at home should have less dermal exposure to Trichothecenes in their clothes and therefore lower urine levels. These levels should be even less in controls who have no exposure to horses, moldy living spaces, or a previously contaminated dryer at home.

Unfortunately, because the dust we brush off of horses also contains the toxin in measurable amounts it is still important to keep exposure to this source while brushing the horse and vacuuming may be a better idea to minimize exposure. Also standing upwind while brushing would be wise. It is my presumption that the amount of toxin exposure in the clothing is more significant than the inhalation of dust. I do, however, think that leaving the barn clothes at the barn and not wearing the clothes with molds and mycotoxins on them in the car and home is a reasonable endeavor. This would be especially important for patients who have chemical sensitivity, chronic fatigue, fibromyalgia and other related illness (Baraniuk 2005), in order to lower their ‘Total Load’ (Rea). Of prime importance is avoidance of exposure to pesticides at any horse facility which is likely to be critical in the decline of any of the aforementioned patients.

Lastly, I refer to the data accumulated by Drs. Kilburn, Simon, Rea, Gray, Heuser, Campbell, Thrasher, and Vojdani ([Mold and Mycotoxins](#)) who have eloquently documented the neurocognitive damage found in patients who are exposed to molds and mycotoxins. The abnormal Spect scans show clear hypoperfusion asymmetrically especially in the temporal lobes. Antibodies to up to 9 components of the nervous system such as myelin basic protein, ganglioside, sulfatide and tubulin indicate that the phenomenon of molecular mimicry is taking place in these

exposed individuals. As I initially mention in my talk, the adrenal hypofunction that often develops in these patients can be associated with anxiety and a struggle to get through the day on adrenaline. Short term memory loss is often profound and the symptoms of attention deficit, distractability, and aggressive behavior may be due solely to adrenal insufficiency ( McBurnette 2000, Shoal 2003) or decrements in neurotransmitters, cerebral blood flow, tissue perfusion (Von Ardenne), or mitochondrial compromise. The autonomic nervous system disruption which occurs in 85% of environmental patients causes much of the anxiety, appearance of struggling to communicate, and the incessant talking in these patients. Dysautonomia (and dysautonomic crisis) often leads to full blown episodes of panic – which is clearly physiologic not psychogenic in origin.

Regardless of the mechanisms of behavioral changes in environmentally exposed individuals – those in this field know well that they exist and that they are treatable by techniques employed in environmental medicine treatment. Because the first principal of treatment is removing the patient from exposure to the prime offending agent, I think it is imperative that women (and men) understand that they are taxing their family's immune, nervous, and endocrine system unnecessarily by washing horse pads filled with mycotoxins in the home laundry system.



**Objectives & Notes**

**Doris Rapp, M.D.**

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Internship:	Buffalo Children’s Hospital
Residency:	Buffalo Children’s Hospital
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**SPEECH TITLE: “The Fetus to Adult, Good, Bad and Ugly in Medicine”**

At the end of this Presentation, the participant should be able to:

1. Current fetus, infant, child, and adult medical challenges.
2. Obesity, teenage vaccines, precocious puberty, and fluoride concerns.
3. What to do and why in relation to the role of phthalates, and electromagnetic energy in their practice.

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## **The Fetus to Adult, Good, Bad, and Ugly in Medicine**

**Doris J. Rapp, M.D.**

Challenges, as well as good and bad decisions, concerning the fetus, infants, children, and teens will be addressed. Some practical aspects of cancer, SIDS, obesity, precocious puberty, forced vaccinations, fluorides and phthalates will also be covered. Some of the “ugly” is incredible. If time permits some very practical aspects of electromagnetic sensitivities will be mentioned.

## Objectives & Notes

### Jesus E. Suarez, M.D.

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Board Certifications:	Mexican Counsel of Otolaryngology and surgery of head and collar/neck

### CASE STUDY: “Young Man Dizziness Treated with Immunotherapy”

At the end of this Presentation, the participant should be able to:

1. Recognize allergies as a cause of dizziness
2. That these cases can be treated with immunotherapy
3. Take another fact for the skin end point titration (SET) as a good method for screening sensitive patients

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## **CASE STUDY: Young Man Dizziness Treated with Immunotherapy**

Allergies not only affects one system, the circulating immune complexes may alter functions elsewhere, thus it may impair functions of the inner ear, it has been found to affect the endolymphatic sac regulatory functions and give rise to dizziness. This could be possible, because the arterial blood supply to the endolymphatic sac comes directly from the external carotid artery via the occipital artery through the posterior menigeal artery; and these vessels are fenestrated and can filter circulating immune complexes.

There is an opportunity to treat these patients with the all the antiallergic armamentarium and of course with immunotherapy. We treated this patient with immunotherapy and controlled his feeding on a rotary basis, and he was well with one onset of dizziness in one year, which means a good result with a significant control of the disease, according to the North American committee for hearing and balance.

