

**22nd Annual International Symposium
on
Man and His Environment in Health and Disease**

Special Focus

**Environmental Aspects and Treatment of Inflammation:
Generators Including Molds, Mycotoxins, Food Infections and
Toxic Chemicals (Metals, Organics)**

**Sponsored by
American Environmental Health Foundation,
Environmental Health Center – Dallas, and
American Academy of Environmental Medicine**

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of the American Academy of Environmental Medicine (AAEM), the American Environmental Health Foundation and the Environmental Health Center - Dallas. The American Academy of Environmental Medicine is accredited by the ACCME to provide continuing medical education for physicians.

The American Academy of Environmental Medicine designates this educational activity for a maximum of 24 hours in Category 1 credit toward the AMA Physician=s Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the activity.

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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 2004 conference will focus on “Environmental Aspects and Treatment of Inflammation: Generators Including Molds, Mycotoxins, Food Infections and Toxic Chemicals (Metals, Organics)”. For this year=s conference, we have assembled a faculty of top international experts for you. 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 you see.
- ! To present new diagnostic and treatment modalities to help you improve the quality of care for your complex patients.
- ! To provide concepts, tools that will enhance your practice.

OBJECTIVES OF THE MEETING

- ! Improve the outcome of treating patients with chronic disease, nutritional problems and chemical sensitivity.
- ! Use new concepts and treatments to help better diagnose and manage many patients with chronic disease, nutritional problems and chemical sensitivity.
- ! Apply the concepts of this conference to your practice by using nutrition and environmental manipulation for the treatment of chronic disease, nutritional problems and chemical sensitivity.
- ! Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness of your practice in relation to chronic disease, nutritional problems and chemical sensitivity.

INTENDED AUDIENCE

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

EDUCATIONAL FORMATS

1. ✂ Plenary
2. ✂ Panels Discussions
3. ✂ Case Studies
4. ✂ 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

Kaye H. Kilburn, M. D.

University of Southern California Medical Center
Keck School of Medicine
Los Angeles, CA

In Memory of Ronald Finn, M.D.

22nd Annual International Symposium on Man and His Environment in Health and Disease

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Thursday, June 24, 2004

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**22nd ANNUAL INTERNATIONAL SYMPOSIUM
ON MAN & HIS ENVIRONMENT**

SCHEDULE

Thursday, June 24, 2004

7:30 – 8:25 a.m. REGISTRATION

8:25 WELCOME: Al Barrier, M.D. & William J. Rea, M.D.

8:30 MODERATOR: Kalpana Patel, M.D.

8:35 Douglas B. Seba, Ph.D., Independent Marine Scientist, Alexandria, VA, *Title: “Environmental Update 2004: Inflammation and Chemicals”*

8:55 Q & A

9:05 William J. Meggs, M.D., Ph.D., Professor of Toxicology, Brody School of Medicine, East Carolina University, Greenville, NC, *Title: “The Inflammation Cure: Diseases & the Environment”*

9:25 Q&A

9:35 Bruce M. Small, P.Eng., Envirosdesic Certification Program, Small & Rubin Ltd., Georgetown, Ontario, Canada, *Title: “Evaluating a Home for Environmental Hazards”*

9:55 Q&A

10:05 BREAK

10:30 Tang Lee, MRAIC, Professor, Faculty of Environmental Design, Calgary, Alberta, Canada, *Title: “Efficacy of Room Type Air Filtration Devices”*

10:50 Q & A

11:00 William J. Rea, M.D., Director, Environmental Health Center - Dallas, Dallas, TX, *Title: “Artificial Implants as Generators of Inflammation”*

11:20 Q & A

11:30 Carmelo Rizzo, M.D., Roma, Italy, *Title: “The Role of Food Intolerance in the I.B.S.”*

11:50 Q & A

12:00 LUNCH

MODERATOR: Jean Monro, M.D.

1:30 Javier Santos, M.D., Hospital General Valle Hebron, Barcelona, Barcelona, Spain, *Title: “Life Stress Promotes Gut Mucosal Inflammation”*

1:50 Q & A

2:00 Professor Kaye H. Kilburn, M.D., Director of Environmental Sciences Lab., University of Southern California, Keck School of Medicine, Alhambra, CA, *Title: “Are Human Brain Functions Impaired from Living Downwind of Massive Hog Confinement?”*

2:20 Q & A

2:30 Jack D. Thrasher, Ph.D., Consultant and Expert Witness, Toxicology and Immunotoxicology, Alto, NM, *Title: “Infections, Cardiovascular and Autoimmune Diseases”*

2:50 Q & A

3:00 BREAK

3:30 Mohamed B. Abou-Donia, Ph.D., Duke University Medical Center, Durham, NC, *Title: “Delayed Neurochemical and Neuropathological Alterations Induced by Sub-Acute Sarin Exposure in the Rat Brain.”*

3:50 Q & A

4:00 Russel J. Reiter, Ph.D., Professor, UT Health Science Center, San Antonio, TX, *Title: “Melatonin: Exogenous and Endogenous Sources and Actions”*

4:20 Q & A

4:30 Panel Discussion: Allan D. Lieberman, M.D., Medical Director, Center for Occupational & Environmental Medicine, PA, North Charleston, SC, **Wallace Rubin, M.D.**, Private Practice, Metairie, LA, *Title: “Inflammatory & Allergic Triggers to the Inner Ear”*

6:00 AJOURN

THURSDAY, JUNE 24, 2004

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

Douglas B. Seba, Ph.D.

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

Date of talk: Thursday, June 24, 2004, 8:35am

Phone: 703/949-1055

Fax:

E-mail:

Medical School/University Attended:
Current Job Description:
Other Information (including titles of books or articles
you have recently written):

University of Miami, Ph.D.
Independent Marine Scientist
Over forty years experience in measuring the fate and
transport of endocrine disruptors in the environment
and their adverse health effects.

Disclosure Statement:

None

SPEECH TITLE: "Environmental Update 2004: Inflammation and Chemicals"

The speaker has provided the information below.

- 1.) Goals and objectives:** To understand the connection between environmental stressors, particularly chemicals such as xenobiotics, and adverse inflammatory processes.
- 2.) Conclusion of what is to be learned:** That adverse health effects can occur at vast distances from their environmental origin and thus place environmental physicians and patients in a constant state of inflammation.
- 3.) References:** Drawn from a mix of media, websites, and scientific publications relevant to the current timeline.

ENVIRONMENTAL UPDATE 2004: INFLAMMATION AND CHEMICALS

Douglas B. Seba

Paradigms are changing. After centuries of tearing nature into ever smaller pieces to try to understand her, ecologists are asking how to put the pieces back together again. Likewise, even the most conventional physicians are beginning to acknowledge that reductionism may not be sufficient to address all the great medical mysteries. Environmental practitioners have been advocating global thinking for decades and inflammation is a perfect example of the whole being greater than the sum of its parts. Chronic inflammatory conditions are now being mentioned in the same sentence with names such as functional genomics and integrative biology. From mainstream media to arcane professional journals, inflammation is suddenly being viewed as the underlying culprit in bewildering array of seemingly unrelated diseases. There is a feeling in the air that maybe, just maybe, this is the dawn of a new era in medical understanding.

For this Conference, the role of chemicals in the inflammatory process has always been a central theme. This review will touch on contemporary xenobiotics, especially endocrine disruptors, and their connection with adverse inflammatory processes. Particular attention will be given to the fact that some of the environmental stresses can come vast distances from their source, be disbursed temporally and geographically, and thus maintain patients in a constant state of inflammation.

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 inflammation processes will be personally applied by the reviewer to ongoing research in wildlife anomalies in the Bitterroot mountains of Montana.

Abstract Information & Notes

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

Brody School of Medicine
East Carolina University
600 Moye Blvd., RM 4W54
Greenville, NC 27858

Date of talk: Thursday, June 24, 2004, 9:05am

Phone: 252/744-2954
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E-mail: meggsw@mail.ecu.edu

Medical School/University Attended:

Internship:

Residency:

Fellowships:

Board Certifications:

University of Miami

University of Rochester

University of Rochester

National Institutes of Health, New York University

Internal Medicine, Emergency Medicine, Allergy,
Medical Toxicology

Professor, Brody School of Medicine

Medical School Professor

Current Faculty Appointments:

Current Job Description:

Other Information (including titles of books or articles
you have recently written):

Author of "The Inflammation Cure", published by
McGraw-Hill, Sept. 2003. Co-Author of "Biomarkers
of Immunotoxicology", published by National
Academy Press; The Physics of Biology Modern
Physics Letters 2003.

Disclosure Statement:

None

SPEECH TITLE: "The Inflammation Cure: Diseases & the Environment"

The speaker has provided the information below.

1.) Goals and objectives: To know the nature of inflammation, the role of inflammation in diseases, and environmental triggers of inflammation can be used to prevent and cure disease

2.) Conclusion of what is to be learned: Understanding the environmental triggers of inflammation can be used to prevent and cure disease

3.) References: William Joel Meggs, M.D. with Carol Svec. The Inflammation Cure. McGraw-Hill, New York, 2003

The Inflammation Cure: Diseases & the Environment

Presenter: William Joel Meggs, MD, PhD
Brody School of Medicine
East Carolina University
Greenville, NC, USA

Inflammation is an abnormal condition of tissue that has been recognized since ancient times. In his *De Medicis*, the Roman physician Aulus Cornelius Celsus described the four cardinal signs of inflammation: *rubor, calor, dolor, tumor* [redness, heat, pain, and swelling]. Under the microscope, inflammation is characterized by cellular infiltrates of neutrophils, lymphocytes, and other inflammatory cells. There is edema and vasodilation. Inflammation is an integral part of the body's defenses and can be triggered by infections, injury, and tumors. Organic chemicals including proteins can trigger inflammation even when there is no danger of infection from these chemicals. Inflammation is integral to a host of diseases. It has long been appreciated that arthritis, asthma, rhinitis, dermatitis of various descriptions are diseases in which an inflammatory response can lead to tissue damage. Over the last decade, we have learned that inflammation plays a crucial role in other diseases such as coronary artery disease, stroke, diabetes, Alzheimer's disease, cancer, and depression. Inflammation is associated with fatigue, malaise, and the frailty syndrome of aging. Environmental factors play a crucial role in activating inflammation and inducing disease. Inflammation is modulated by what we eat [Diet and nutrition], where we live, work and study, our lifestyles, and how we think. Medications have a role in modulating inflammation. Recent advances in understanding the role of inflammation in a host of diseases suggests new options for treating diseases, and suggest that the role of environmental medicine be expanded in new directions.

Abstract Information & Notes

Bruce M. Small, P.Eng.

Small & Rubin Ltd.
100 Rexway Dr.
Georgetown, Ontario L7G 1R5
Canada

Date of talk: Thursday, June 24, 2004, 9:35am

Phone: 905/702-8615
Fax: 905/873-6260
E-mail: brucesmall@envirodesic.com

Medical School/University Attended:

Bachelor of Applied Science degree in Engineering Science from the University of Toronto (1970) and a Master of Science degree in design from the University of Southern Illinois (1975)

Current Job Description:

Director of the *Envirodesic*TM Certification Program, a private sector initiative for developing and promoting buildings, products and services that contributes to healthier indoor environments. Executive Director of *Technology and Health Foundation*, a non-profit research corporation.

Other Information (including titles of books or articles you have recently written):

Has produced general and specialized research studies, for environmentally hypersensitive individuals, is a consultant, and author of two environmental health books, *Sunnyhill – The Health Story of the Eighties* and *The Susceptibility Report*, including an autobiography about his personal experience with the effects of pollution on the health of his own family.

Disclosure Statement:

Icynene Inc. – The Insulation System®
Cogent Environmental Inc. – Ecogent® General Purpose Cleaner

SPEECH TITLES: “Evaluating a Home for Environmental Hazards”

The speaker has provided the information below.

1.) Goals and objectives: The purpose of this talk is to give the medical participants in the symposium some insight as to the detail required in home inspections for persons who may be environmentally sensitive. The information may also be useful for medical staff involved in coaching patients on methods of home environmental diagnosis and control.

2.) Conclusion of what is to be learned: The nature of home environmental evaluations must necessarily vary depending on the purpose. The focus of investigation will be different if a patient is choosing a new home, rather than fine-tuning an existing one. A certain amount of problem identification or elimination can be conducted without a site visit by considering major building characteristics, e.g. presence of mold or a leaky furnace. A detailed site visit typically involves a room-by-room walk-through, guided by some knowledge of the client’s most significant environmental sensitivities. On occasion formal scientific testing may be required to quantify an invisible hazard (e.g. formaldehyde level or pesticides) or as a basis for treatment (e.g. desensitization to residual molds after home cleanup).

3.) References: The author draws upon 27 years of personal experience in home inspection for the environmentally sensitive.

Abstract Information & Notes

Tang G. Lee, MRAIC

Faculty of Environmental Design
The University of Calgary
2500 University Drive NW
Calgary, Alberta T2N 1N4
Canada

Date of talk: Thursday, June 24, 2004, 10:30am

Phone: 403/220-6608
Fax: 403/284-4399
E-mail: lee@ucalgary.ca

Medical School/University Attended:

Case Western Reserve University, Cleveland, The Ohio State University, Columbus, Ryerson University, Toronto

Board Certifications:

Royal Architectural Institute of Canada

Current Faculty Appointments:

The University of Calgary

Current Job Description:

Professor

Other Information (including titles of books or articles you have recently written):

Part owner of and operates one of the largest tropical fish farm in Canada, raising tilapia for consumption. The waste heat, humidity and wastewater discharges into a hydroponics greenhouse.

Disclosure Statement:

Nil

SPEECH TITLE: "Efficacy of Room Type Air Filtration Devices"

The speaker has provided the information below.

- 1.) Goals and objectives:** Understand performance of portable air filters and their limitations. Learn how proper air filtration is achieved to minimize air contaminants in buildings.
- 2.) Conclusion of what is to be learned:** Learn about misleading claims from air filter manufacturers.
- 3.) References:** Lee, T.G., Air quality examination of the TCPL tower for occupants of the Calgary Court of Appeals, unpublished report for Alberta Justice, May 18, 2003

Efficacy of room type air filtration devices

22nd Annual International Symposium on Man and His Environment in Health and Disease. June 24-27, 2004.

Tang G. Lee, MRAIC
The University of Calgary
Phone: 403-220-6608

ABSTRACT

Despite the efficiencies of portable air filter devices, the ability to clean the air in a room is limited. The reason is that any air being supplied into a room will bring a constant flow of potentially polluted air into this space. The air filter device is unable to clean the air in the room, despite its efficiency and airflow rate. Within one metre from the clean air discharge from the unit, the air becomes as polluted as the supply air.

The only method to effectively clean the air in a room is to filter the supply air before it is discharged into the room. The device must be suspended below every ceiling air supply diffuser. However, the suspended air filter units below the ceiling are costly and unsightly. A better method is to install the air filter unit inside the duct supplying air into the room.

The only space that air filter devices are effective is in a room without any ventilation, either from a supply air or from a window. With time, the device continues to clean the air in this sealed room. However, an occupied sealed room without ventilation will soon be depleted of oxygen. Thus this air filter device may clean the airborne particles and even some gasses, but it does not remove carbon dioxide nor provide oxygen.

Portable air filter devices are ineffective in cleaning the air in space that has ventilation. To be effective, the air filter device must intercept and clean the air before it is supplied to the room.

References:

Lee, T.G., Air quality examination of the TCPL tower for occupants of the Calgary Court of Appeal. Unpublished report for Alberta Justice, Edmonton, May 18, 2003.

Abstract Information & Notes

William J. Rea, M.D.

Date of talk: Thursday, June 24, 2004 11:00am

Environmental Health Center - Dallas
8345 Walnut Hill Lane, Suite 220
Dallas, TX 75231

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Medical School/University Attended:

Ohio State University College of Medicine, Columbus, OH

Internship:

Parkland Memorial Hospital, Dallas, TX

Residency:

University of Texas Southwestern Medical School, Dallas, TX

Board Certifications:

American Board of Surgery, American Board of Thoracic Surgery, American Board of Environmental Medicine, American Board Certification, Disability Analyst

Current Faculty Appointments:

Capital University of Integrative Medicine, Washington, D.C.

Current Job Description:

President/M.D. – Environmental Health Center-Dallas

Other Information (including titles of books or articles you have recently written):

Optimum Environments for Optimum Health and Creativity (book); Toxic Exposure to Molds and Mycotoxins

Disclosure Statement:

None

SPEECH TITLE: “Artificial Implants as Generators of Inflammation”

The speaker has provided the information below.

1.) Goals and objectives: To show that implants and non-functioning, dyshomeostatic organs can be generators of inflammation and chronic inflammatory disease states.

2.) Conclusion of what is to be learned: Many implants and dyshomeostatic organs can be generators of inflammation.

3.) References: Randolph, T.G., Human Ecology and Susceptibility to the Chemical Environment, 7th Printing. Charles C. Thomas, Publisher, 1962. Springfield, IL.

ARTIFICIAL IMPLANTS AS GENERATORS OF INFLAMMATION

**William J. Rea, M.D., F.A.C.S., F.A.A.E.M.
Environmental Health Center – Dallas
8345 Walnut Hill Lane, Ste. 220
Dallas, Texas 75231**

There are now over 220 artificial synthetic implants used in the human body. All will initially generate a local inflammatory reaction followed by a fibrotic protective capsule around the implant. Some develop regional or diffuse inflammatory reactions that will cause regional or distal clinical reactions and vasculitis. After a period of time even with removal of the implant, the vasculitis will not stop and the inflammation appears to become autonomic.

Conclusions:

However, frequently, removal helps. Then secondary sensitivity arrives, still triggering the inflammation. These can be any extraneous environmental incitant, inhalant, food, and chemical. These must be treated in order to eliminate the inflammation and return the patient to health. Many implants have to remain in the body and their material has to be neutralized. These include stainless steel, titanium, all other metals, Teflon, Dacron, silicon, etc. Technique will be discussed on how to deal with them.

Goals and Objectives:

1. To present that artificial implants can be generators of inflammation.
2. To aid individuals with implants as part of the generators of inflammation on what to do.
3. To aid the clinician in treating inflammatory disease related to implants.

References:

Rea, William J.: Chemical Sensitivity, Volume 3, Lewis Publishers, Boca Raton, Florida, 1996.

Higuchi, H., Miyata, M., Ishikawa, S., Rea, William J.: (1996) Abnormalities of the Autonomic Nervous System Seen In Patients Who Have Silicon Breast Implants. International Journal of Occupational Medicine, Immunology, and Toxicology, Vol. 5, No. 1. pp. 1-13.

Abstract Information & Notes

Carmelo Rizzo, M.D.

Natural S.r.l. - Viale Ippocrate 93
Roma, 00161
Italy

Date of talk: Thursday, June 24, 2004, 11:30am

Phone: 011/39-6-49380793
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E-mail: natural.roma@flashnet.it

Medical School/University Attended:
Internship:
Current Faculty Appointments:
Current Job Description:
Other Information (including titles of books or articles
you have recently written):

University of Rome
Rome Policlin
University of Urbino
Clinical Ecologist – Nutrition and Allergies
Translations and publication of R. Mackarness “Not all
in the Mind” and “D. Rapp “The Impossible Child” in
Italian. Clinical ecology and food intolerance ed. By
Tecnico Nuove. Founder and President of the Italian
Associates of Clinical Ecology (A.M.I.E.C.).
International Lecturer. Author of many articles on
Food Intolerance.

Disclosure Statement:

None

SPEECH TITLE: “The Role of Food Intolerance in the I.B.S.”

The speaker has provided the information below.

- 1.) Goals and objectives:** Treatment of I.B.S. with the exclusion diet and use of Hydro Colon Therapy
- 2.) Conclusion of what is to be learned:** That the association of a good diagnosis of food intolerance and their exclusion, the use of probiotics and practice of Hydro Colon Therapy all result in a good treatment of this syndrome.
- 3.) References:** The Italian Journal of Gastroentology Vol. 8 Num. 1, March 2003
Study of leukocytes modifications comparing the Cytotoxic method with the analytic approach

THE ROLE OF HYDROCOLONOTHERAPY IN ASSOCIATION WITH PROBIOTICS IN THE THERAPEUTIC APPROACH TO FOOD INTOLERANCES

S. Pedullà – G. Sanfilippo
Endoscopic Surgery - University of Catania
in cooperation with
Prof. Dott. Carmelo Rizzo
University of Urbino

A research was carried on 20 patients (10 men and 10 women, between 20 and 78 years old) affected by a food intolerance between the grade 3rd and 4th.

The aim of our work was to verify, also through the leukocytotoxic test, the efficiency of hydrocolonotherapy, associated or not to probiotics, as a treatment of help in the exclusion diet.

The results obtained proved that hydrocolonotherapy, only supported by the dietetic treatment, allows to achieve a considerable improvement in the symptomatology both somatic and visceral; it also implies a reduction in the grade of reaction against the aliments which are not tolerated by the patient, after only 30 days from the beginning of the therapy.

Furthermore, we found out that patients who have been treated with the combination of both oral probiotics therapy and hydrocolonotherapy showed a significative decrease of the clinical disorders; in all the patients we obtained the total disappearance of the intolerance for at least one of the foods responsible of the adverse reaction.

Therefore, even if this therapy can't be considered the only rational method in the treatment of food intolerances, we can say that the hydrocolonotherapy, associated with the probiotics treatment at high concentrations, represents an important and effective therapeutic approach of this pathology.

*Prof. Dott. Carmelo Rizzo
In cooperation with Dott. La Bruna
Head of the Laboratory Dpt.
Of Garibaldi Hospital of Catania (Italy)*

RESULTS

- We tested 20 patients with a food intolerance to milk and eggs.
- In order to verify the negative answer, we also tested the blood of a healthy subject (not allergic).
- The reading of the Cytotoxic test results showed a 3^o grade intolerance to milk and egg.
- All samples have been tested with the coulter counter before the addition of the allergen (graphs 1 to 5) and also after the addition of 50 µl casein, β-lacto globulin and egg.
- Here follow the blood samples of 5 cases.

Abstract Information & Notes

Javier Santos, M.D., Ph.D.

Hospital General Valle Hebrón
Institut Fundacio Recerca, Digestive Diseases Research
Unit
Paseo Valle Hebrón 119-129
Barcelona, Barcelona 08035
Spain

Date of talk: Thursday, June 24, 2004, 1:30pm

Phone: 93/489-4035
Fax: 93/489-4456
E-mail: jsantos@vhebron.net

Medical School/University Attended:
Internship:

1982-88: Medical School, University of Navarra, Spain
Licensure in General Medicine and Surgery, University
of Navarra, Spain (equivalent to M.D.)

Internship:

1990-1993, Specialization Degree in Gastroenterology,
Hospital General Universitario Valle de Hebrón,
Barcelona, Universidad Autónoma de Barcelona, Spain
Not applicable

Board Certifications:

Current Job Description:

Clinician and Researcher

Other Information (including titles of books or articles
you have recently written):

2000-6: Research contract within the National Health
System (99/3146) supported by the Subdirección
General of Investigación Sanitaria, Instituto de Salud
Carlos III, entitled: "Effect of stress on intestinal
function in patients with irritable bowel syndrome and
development of a new experimental animal model of
irritable bowel syndrome", to develop at Digestive
System research Unit at Hospital General Valle de
Hebrón, Barcelona. Has published several articles.

Disclosure Statement:

None

SPEECH TITLE: "Life Stress Promotes Gut Mucosal Inflammation"

The speaker has provided the information below.

1.) Goals and objectives: To learn how and why life stress is able to induce an inflammatory-prone mucosal microenvironment in the human intestine.

2.) Conclusion of what is to be learned: Life stress may predispose healthy individuals to develop intestinal mucosal inflammation. Characterization of this latent inflammatory condition may be of outmost importance to unravel the pathogenesis of gut functional disorders affecting up to 20% of the general population. Prevention of deleterious effects of life stress may help to achieve a healthy gut and to enhance quality of life.

3.) References: 1) Alonso C., Santos J, Guilarte M. Corticotropin – releasing hormone promotes jejunal proinflammatory responses in IBS patients. Selected for oral presentation at Digestive Disease Week, New Orleans, Louisiana, May 15-20, 2004

2) Hart A, Kamm MA. Mechanisms of initiation and perpetuation of gut inflammation by stress. *Aliment Pharmacol Ther.* 2002; 16:2017-28

3) Mayer EA, Collins SM. Evolving pathophysiologic models of functional gastrointestinal disorders. *Gastroenterology.* 2002; 122:2032-48.

4) Santos J, Perdue MH. Stress and neuroimmune regulation of gut mucosal function. *Gut.* 2004;47 Suppl 4:iv49-51.

5) Vicario M, Santos J, Alonso C. Altered Colonic Barrier and Mitochondrial Function to CRH in chronically-stressed rats.

Life Stress Promotes Gut Mucosal Inflammation

Javier Santos, M.D., Ph.D

Hospital Universitario Valle de Hebron. Digestive System Research Unit. Barcelona, Spain.

Life stress is certainly one of the most important environmental diseases of developed countries affecting more than 50% of workers for over 50% of the time (1). Life stress has been linked to both acute and chronic health problems varying from common cold or increased susceptibility to infections to depression and heart disease. (2-3). In particular, the influence of psychosocial factors on gastrointestinal diseases is being increasingly recognized (4-5). Functional disorders of the gut, including irritable bowel syndrome and functional dyspepsia may affect up to 10-20% of the adult population. Recent data have shown that the clinical evolution and the intensity of symptoms in certain patients with irritable bowel syndrome may depend, to a great extent, on the presence of life stress as a co-morbid factor (6-7). Acute gastroenteritis predisposes to the development of irritable bowel syndrome in certain individuals (8-9). In this group of postinfective irritable bowel patients, psychological factors including life stress most clearly predicted the development of symptoms (8,10).

Experimental evidence indicates that stress alters gastrointestinal motility (11), enhances visceral perception (12), and reactivates naturally occurring (13) as well as chemically-induced colitis (13-14). However, a physiopathological basis linking stress with the development of irritable bowel syndrome in these postinfective patients is still lacking. Previously, we and others have established that both acute and chronic stress induced prominent alterations in rat intestinal epithelial physiology. In particular, we have shown that different stresses increased ion secretion and ion permeability in the rat jejunum and colon of the stress susceptible strain, Wistar-Kyoto (15-18). These changes were paralleled by a marked increase in epithelial macromolecular permeability. These epithelial abnormalities were mimicked by the intraperitoneal injection of corticotropin-releasing hormone, the main stress mediator, through mast cells and neural pathways (19). The magnitude of the changes between the acute and chronic stress models was not been directly compared. However, these epithelial abnormalities lasted for up to 3 days in chronic-stressed rats whereas returned to normal after 24 h in the acute stress study. Such persistent abnormalities could have clinical implications. It has been shown that intestinal permeability is increased in Crohn's disease patients (20) and that germfree rodents do not develop intestinal inflammation (21). Increased uptake of luminal microbial antigens may be required to activate mucosal immune cells leading to an inflammatory response involved in the pathogenesis and/or reactivation of intestinal disorders.

We have also investigated whether enteric infections interact with social stress to promote mucosal inflammation, visceral hypersensitivity and epithelial barrier dysfunction to develop an experimental model of irritable bowel syndrome. In these studies administration of *S. aureus* enterotoxin B triggered persistent visceral hypersensitivity, enhanced epithelial hypersecretion and extended mucosal inflammation induced by social stress in the rat colon (22).

Finally, to close the circle we have recently investigated the effect of corticotropin-releasing hormone, the key stress mediator, on epithelial and sensory responses in irritable bowel syndrome patients and healthy volunteers. The results of this study suggest that stress-mediated corticotropin-releasing hormone release may activate symptomatic irritable bowel syndrome by promoting an inflammatory-prone mucosal microenvironment (23).

References:

1. European Foundation for the Improvement of Living and Working Conditions, Office for Official Publications of the European Communities, 2985 Luxembourg: Second European Survey on Working Conditions-Working Conditions in the European Union. 1995/6.
2. Kivimäki M, et al. Work stress and risk of cardiovascular mortality: Prospective cohort study of industrial employees. *BMJ* 2002, 325, 857-860.
3. Miller GE, Cohen S, Pressman S, Barkin A, Rabin BS, Treanor JJ. Psychological stress and antibody response to influenza vaccination: when is the critical period for stress, and how does it get inside the body? *Psychosom Med.* 2004;66:215-23.
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5. Whitehead WE. Psychosocial aspects of functional gastrointestinal disorders. *Gastroenterol Clin N Am* 1996;25: 21.

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Abstract Information & Notes

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Date of talk: Thursday, June 24, 2004, 2:00pm

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Disclosure Statement:

None

SPEECH TITLE: “Are Human Brain Functions Impaired from Living Downwind of Massive Hog Confinement?”

The speaker has provided the information below.

1.) Goals and objectives: Question: Is there evidence for brain impairment from living near hog confinement)
Goals-Be able to recognize pattern and severity of decreased function due to hydrogen sulfide from pork production the modern way. Your nose knows odors.

Patients exposed to H₂S lose functions.

Objectives-Measure pulmonary and neurobehavioral functions in neighbors of hog farms.

Compare functions to unexposed people nearby and to standard reference people adjusting for age, sex, ed. Level, height and weight.

Tables 1, 2, 3

Figures 1, 2, 3

2.) Conclusion of what is to be learned: Hog confinement generates H₂S.

Manure lagoons release H₂S in bioaerosols and on particles.

H₂S reduces human brain function.

Effects appear irreversible.

Advice-Avoid rotten egg gas.

Summary-Ohio people near hog confinement differed by sway (eyes open), digit symbol substitution, and vocabulary from those further away.

Exposed differed from referents by simple and choice reaction time (SRT, CRT), sway eyes open and closed (SO, SC), color discrimination (right and left), visual fields performance (right and left), digit symbol substitution, vocabulary, verbal recall-immediate and delayed, and picture completion.

There was no difference in pegboard, trail making A and B, information, similarities.

- 3.) References:** Kilburn KH: Brain but not lung functions impaired after a chlorine incident. *Natl Inst. Industrial Health Japan*, 2003; 41:299-305.
Kilburn KH: Effects of hydrogen sulfide (H₂S) on neurobehavioral function. *Southern Medical Journal* 2003; 96:639-646.
Kilburn KH: Evaluating health effects from exposure to hydrogen sulfide: Central Nervous System Dysfunction. *Environ Epidemiol Toxicol* 1:207-216, 1999

Are Human Brain Functions Impaired From Living Downwind of Massive Hog Confinement?

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Background Manure lagoons from massive hog confinement buildings in many states emit methane, hydrogen sulfide (H₂S) and other reduced sulfur gases. Neighbors complain of offensive odors and losses of concentration, memory, and balance.

Method We evaluated a group of neighbors to lagoons in Ohio and compared them to people living 3 or more kilometers away and to controls in a nearby state. Both groups neurobehavioral testing for physiological, cognitive, recall and memory function, and pulmonary function testing was by the same team with distant controls done earlier.

Results The 25 exposed subjects averaged 7.9 abnormalities compared to 5.2 for local Ohio controls and 2.3 in Tennessee controls, and both differences were statistically significant. Comparing mean values, Ohio exposed people differed by 3 abnormalities from Ohio unexposed people: balance with eyes open, digit symbol substitution, and vocabulary. This Ohio exposed group had 13 differences in abnormalities compared to Tennessee unexposed people. The third comparison of Ohio Unexposed people compared to Tennessee controls showed 10 differences. The exposed groups pulmonary function mean forced vital capacity and forced expiratory volume in 1 sec. were reduced significantly compared to local and regional controls. Mood states scores and symptom frequencies were elevated. Distances from the lagoons did not predict neurobehavioral or pulmonary impairment in the Ohio exposed group and durations of residence (exposure) did not vary. Low doses of H₂S may inhibit enzymes metabolizing cysteine in brain tissue. H₂S is suggested to be the 3rd gaseous neurotransmitter after NO and CO.

Conclusions Exposure to hydrogen sulfide from hog enclosures and manure lagoons impaired neurobehavioral and pulmonary functions. Impairment scores in local controls were higher than expected and may reflect exposures to agricultural chemicals that were shared with the H₂S exposed people.

HUMAN BRAIN IMPAIRMENT DOWNWIND OF HOG CONFINEMENT

ABSTRACT

Problem To determine whether neighbors around manure lagoons and massive hog confinement buildings who complained of offensive odors and had symptoms of impaired brain and lung performance.

Method We compared neighbors of lagoons in Ohio to people living beyond 3 kilometers and to controls in a nearby state by testing neurophysiological, cognitive, recall and memory functions, and pulmonary performance.

Results The 25 exposed subjects averaged 7.9 neurobehavioral abnormalities, significantly different from 5.2 for local controls and 2.3 for Tennessee controls. Their mean forced vital capacity and expiratory volume in 1 sec. were reduced significantly compared to local and regional controls.

Neurobehavioral Testing: Comparison of the means of the 25 exposed peoples tests to those of the local unexposed group of 22 showed statistically significant (ss) differences for balance with eyes open was 2.0 abnormalities, digit symbol substitution 1.0 abnormality and vocabulary 1.0 abnormality (Table 1). These abnormalities were not statistically significant after Holm's adjustment for multiple inference. Comparison to the Tennessee (TN) referent group showed 8 physiological test differences: balance measured with eyes open and with eyes closed for 4 abnormalities, simple and choice reaction time 2 abnormalities, color discrimination errors 1 abnormality, and visual field performance 2 abnormalities. For the psychological tests: digit symbol substitution, vocabulary, verbal recall (immediate and delayed), and picture completion were also significantly different. Testing for simultaneous inference (37) reduced the significant differences for both sets of comparisons, but choice reaction time, balance and color remained different compared to TN controls, only simple reaction time was lost. Ohio controls had 6 differences from TN controls.

Profile of Mood States mean scores were elevated at 53.1 in the 25 exposed versus 5.6 in the 33 unexposed, a significant difference (Table 2). Total abnormalities were correlated with symptom frequencies but not Profile of Mood States scores by regression analysis ($p > .007$) with 27.4% of the variance explained (r^2).

Exposure: Indoor air of 11 homes had hydrogen sulfide levels of 2 to 27 ppb. Ranges indoors and outdoors varied less than 50% in one day's spot check samples. Distances varied widely but the inverse of distance squared from hog confinement lagoons did not predict scores or number of abnormalities. Exposure was less than 4 years in only two people.

More of the exposed than control groups had smoked cigarettes: 40% vs. 28% but similar proportions, 16% vs 13% continued to smoke. Regression of total neurobehavioral abnormalities against age, duration of smoking in years and educational attainment showed only age was significant in the exposed and no factor was significant in the unexposed.

The difference in total abnormalities, mean 7.9 ± 4.0 , for the 25 exposed compared to a mean of 5.2 ± 2.6 in the 22 unexposed was statistically significant (by ANOVA $p < .011$). The comparison of the exposed group mean abnormalities to the Tennessee control's mean of 2.3 ± 2.3 was also statistically significant ($p < .0001$), as was the comparison of Ohio controls to Tennessee controls that showed 6 differences and an abnormality score of 8 which was also significant ($p < .0001$).

The exposed group had increased frequencies for shortness of breath when climbing stairs, but not at rest, or while walking nor was wheezing more frequent. Their expiratory flows and vital capacities were significantly decreased (comparisons were adjusted for years of smoking) (Table 3).

Frequencies of 18 of 35 symptoms were statistically significantly elevated in exposed compared to unexposed (Table 4). Mean frequencies were significantly different, 3.2 ± 1.7 in exposed versus 1.9 ± 0.9 in unexposed. There were no differences between the exposed and unexposed groups for rheumatic, lupus erythematosus complaints or

for neurological diseases and psychiatric illnesses. None had substance dependency. The unexposed and exposed groups' did not differ in their occasional exposures to 15 occupations and groups of chemicals.

The 58 Tennessee unexposed people's individual abnormality scores averaged 2.3 as plotted in Figure 1 (13). The comparative abnormality scores for the 25 exposed (mean 7.9) and 22 unexposed (mean 5.2) are plotted in Figures 2 and 3. The distribution of abnormalities of the Tennessee controls was skewed, decreasing sharply from many without any to one or two abnormalities. In contrast, the 25 exposed subjects had a symmetrical distribution around 7.9. The 22 Ohio unexposed subjects also had an unexpectedly symmetrical distribution of abnormalities from a mean of 5.2. The symmetrical distributions of abnormalities in both hydrogen sulfide exposed and unexposed, including the rarity of people with no abnormalities, suggested their exposure to "an unknown Ohio factor".

Conclusions Hog enclosures and manure lagoon gases impaired neurobehavioral and pulmonary functions in neighbors and somewhat in nearby controls. Hydrogen sulfide should be abated. People living near lagoons must avoid rotten egg gas.

TABLE 1

**PAULDING, OHIO--EXPOSED COMPARED TO UNEXPOSED AND TO 58 TENNESSEE REFERENTS.
VALUES ARE AS PERCENT PREDICTED.
MEANS COMPARED BY ANALYSIS OF VARIANCE (ANOVA).**

	A Unexposed	B Exposed	A vs B	C Referents	B vs C	A vs C
	mean±sd	mean±sd	P value	mean±sd	p value	p value
Age (years)	53.6±16.8	53.2±17.3	.938	34.3±13.5	.0001	.121
Educational level (years)	12.3±103	12.8±1.8	.358	12.9±2.3	.817	.236
Simple Reaction Time	100.8±4.0	102.1±4.3	.285	99.6±4.1	.017* .24 ⁺	.277
Choice Reaction Time	101.2±3.0	102.2±3.5	.270	99.8±2.6	.0005* .01 ⁺	.032* .42 ⁺
Balance Sway Speed						
Eyes Open	104.3±21.7	123.4±34.5	.031* .837 ⁺	98.8±21.4	.0002* .004 ⁺	.39
Eyes Closed	109.1±25.5	125.4±45.1	.146	98.9±28.0	.0016* .03 ⁺	.334
Blink Reflex Latency R-1						
Right	91.0±7.4	97.6±14.3	.086	96.5±12.0	.745	.020* .44 ⁺
Left	91.9±10.0	98.9±15.7	.119	99.1±15.8	.963	.177
Color Discrimination Errors						
Right	62.4±45.1	53.0±46.2	.492	94.6±43.1	.0002* .004 ⁺	.0001* .0027 ⁺
Left	55.3±33.9	46.8±39.5	.442	99.4±39.4	.0001* .0027 ⁺	.0001* .0026 ⁺
Visual Field Performance						
Right	120.7±13.9	114.4±12.9	.112	97.0±13.7	.0001* .0026 ⁺	.0001* .0025 ⁺
Left	122.2±19.9	116.5±14.2	.263	97.3±17.5	.0001* .0025 ⁺	.0001* .0024 ⁺
Grip strength						
Right	104.8±14.3	96.7±17.0	.088	98.2±18.3	.733	.160
Left	101.0±16.7	93.4±18.3	.152	94.7±19.3	.784	.639

TABLE 1 (cont'd)

**PAULDING, OHIO--EXPOSED COMPARED TO UNEXPOSED AND TO 58 TENNESSEE REFERENTS.
VALUES ARE AS PERCENT PREDICTED.**

MEANS COMPARED BY ANALYSIS OF VARIANCE (ANOVA).

	A Unexposed	B Exposed	A vs B	C Referents	B vs C	A vs C
	mean±sd	mean±sd	p-value	mean±sd	p value	p value
Culture Fair	105.0±28.1	99.7±19.1	.452	100.6±18.8	.842	.427
Digit Symbol	100.9±12.0	89.6±19.1	.024* .60 ⁺	102.2±9.1	.0001* .0024 ⁺	.790
Vocabulary	91.0±35.9	69.2±29.9	.028* .728 ⁺	90.8±27.0	.002* .03 ⁺	.248
Verbal Recall						
Immediate	81.7±27.9	77.1±20.6	.518	100.5±31.1	.0008* .015 ⁺	.011* .02 ⁺
Delayed	55.1±33.0	53.6±34.7	.883	102.5±40.4	.0001* .0023 ⁺	.0001* .0024 ⁺
Pegboard	115.5±20.6	105.2±20.0	.093	103.9±16.5	.769	.526
Trails A	100.7±7.2	103.6±8.1	.203	100.3±10.1	.149	.821
Trails B	100.8±7.8	100.7±12.5	.968	98.7±8.4	.423	.815
Finger Writing Errors						
Right	95.7±9.0	97.9±7.7	.366	100.4±9.6	.249	.027*
Left	96.9±9.0	101.3±10.1	.132	103.2±10.3	.442	.124
Information	100.0±38.1	89.3±33.5	.317	96.7±37.9	.403	.866
Picture Completion	71.1±35.0	64.9±41.0	.588	90.0±24.6	.0009* .016 ⁺	.0002* .006 ⁺
Similarities	103.6±29.5	90.4±43.5	.242	95.0±41.6	.650	.552

*= P< 0.05

+ = P< 0.05 after Holm's correction for multiple inference

TABLE 2

**PROFILE OF MOOD STATES (POMS) FOR 25 EXPOSED COMPARED TO 22 UNEXPOSED
SUBJECTS IN PAULDING, OHIO.
POMS SCORE IS THE SUM OF TENSION, DEPRESSION, ANGER,
FATIGUE AND CONFUSION MINUS VIGOR**

POMS	25 Exposed mean±sd	22 Unexposed mean±sd	p values
Score	53.1±45.5	5.6±18.6	.0001
Tension	14.9±7.7	7.2±4.0	.0001
Depression	14.7±12.1	4.1±4.1	.0003
Anger	15.4±11.9	4.8±4.5	.0003
Vigor	14.7±7.2	19.8±7.0	.018
Fatigue	12.2±7.6	5.5±3.4	.0004
Confusion	10.6±5.8	3.8±2.5	.0001
Range	-11 to 164	-28 to 46	

TABLE 3
PULMONARY FUNCTION TESTS IN 25 EXPOSED SUBJECTS
COMPARED TO 22 UNEXPOSED IN PAULDING, OHIO AND 58 UNEXPOSED TENNESSEE
SUBJECTS

	A 22 Unexposed	B 25 Exposed	A vs B	C 58 Unexposed	B vs C	A vs C
	Mean±sd	Mean±sd	p values	Mean±sd	p values	p values
FVC	97.0±12.8	87.6±10.7	.014	101.6±15.2	.0001	.180
FEV ₁	94.9±11.7	85.5±15.0	.028	93.6±15.2	.025	.719
FEF ₂₅₋₇₅	102.0±23.7	91.9±30.8	.232	88.1±35.0	.633	.070
FEF ₇₅₋₈₅	93.0±27.1	96.4±54.1	.800	78.1±52.7	.133	.191
FEV ₁ /FVC	77.9±3.3	76.1±7.3	.301	72.8±9.5	.130	.014*

* more normal than Tennessee

Slide 1

Human Brain Impairment Downwind of Hog Confinement

Slide 2

Question: Is there evidence for brain impairment from living near hog confinement?

Do patients exposed to H₂S lose functions?

Goals-Be able to recognize pattern and severity of decreased function due to hydrogen sulfide from pork production the modern way. Your nose knows odors and warns you unless its input is overwhelmed.

Objectives-Measure pulmonary and neurobehavioral functions in neighbors of hog farms.

Compare their functions to unexposed people nearby and to reference people adjusting for age, sex, ed. level, height and weight.

Slide 3

Summary-Ohio people near massive hog confinement and manure lagoons differed by sway (eyes open), digit symbol substitution, and vocabulary from those further away.

Exposed differed from referents by additional impairments of simple and choice reaction time, sway eyes closed, color discrimination (right and left), visual fields performance (right and left), digit symbol substitution, vocabulary, verbal recall-immediate and delayed, and picture completion.

There was no difference in pegboard, trail making A and B, information, and similarities.

Slide 4

Conclusions- Hog confinement generates H₂S.
Manure lagoons release H₂S in bioaerosols and on particles.
H₂S reduces human brain function.
Effects appear irreversible.

Slide 5

Advice
Your nose knows
Avoid rotten egg gas.

Abstract Information & Notes

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Recent Research: (2003) Antibodies to Molds and Satratoxin in Individuals Exposed in Water-Damage buildings; Mixed Mold Exposure: Immunological Changes in Humans with Exposure in Water Damaged Buildings; On the neuropsychological and electrocortical impacts of mixed mold exposure; Neural antigen autoantibodies and neurophysiology abnormalities in patients exposed to mixed-molds in water-damaged buildings; (2004) Abnormalities in Gulf War Veterans: Studies with Lymphocyte Numbers, Lymphocyte Function and Antibodies. *Environmental Health Perspectives*

Disclosure Statement:

Immunosciences Lab, Inc. – Consultant, Medical Center for Immune & Toxic Disorders - Consultant

SPEECH TITLES: “Infections, Cardiovascular and Autoimmune Diseases”

The speaker has provided the information below.

1.) Goals and objectives:

2.) Conclusion of what is to be learned:

3.) References:

INFECTIONS, CARDIOVASCULAR AND AUTOIMMUNE DISEASES

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Cardiovascular disease is predicted to be the most common cause of death worldwide by the year 2020. Half of heart disease patients lack established risk factors. However, previous and recent studies point to a linkage between infection with different bacteria and heart diseases in the other (no risk factors) 50% of observed cases. Pathogenesis of the disease induced by infectious agents is described by three mechanisms of action:

1. Induction of inflammation
2. Release of toxins or superantigens
3. Molecular mimicry or cross-reactivity.

Through the years, many reports have incriminated various infectious agents in the pathogenesis of autoimmune disease. Very recently, The American College of Cardiology issued a list of harmful pathogens as possible links to heart disease.

In addition, evidence suggests that chronic dental infection may be another factor for the development of atherosclerotic heart disease. Patients with poor dentition, especially those with periodontal disease are noted to have frequent episodes of bacteremia. Bacteria in dental plaque may cause blood clots. This plausible cause was described in a study published in the Journal of Periodontology (October 1996) and in three separate articles by Dr. Aristo Vojdani in Laboratory Medicine (March, April and May, 2003).

Based on these articles and a review of 1999 various manuscripts published in scientific journals, we have developed a **Blood Test for Detection of Infectious Agents Involved in Cardiovascular and Autoimmune Diseases**. This test measures IgG antibodies against antigens of more than 15 species of infectious agents. These particular microorganisms (viruses, bacteria, fungi, protozoa) produce a multitude of antigens (proteins) that have immunological cross-reactivity with human proteins. These include human myosin and specific proteins and peptides involved in Lupus and arthritis and will be discussed.

Abstract Information & Notes

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of neurotoxicology.

Disclosure Statement:

None

SPEECH TITLE: "Delayed Neurochemical and Neuropathological Alterations Induced by Sub-Acute Sarin Exposure in the Rat Brain."

The speaker has provided the information below.

1.) Goals and objectives: To investigate the delayed effects, one week after exposure to the nerve agent, sarin after a single intramuscular dose of 1, 0.5, 0.1, or 0.01 X LD50: 1) Plasma butyrylcholinesterase, 2) Brain regions acetylcholinesterase, 3) m2-muscarinic acetylcholine receptor, 4) Permeability of the blood brain barrier, and 5) Histopathological and immunohistochemical changes in the rat brain.

2.) Conclusion of what is to be learned: Exposure to lethal doses of sarin caused seizures and convulsions leading to cell death via necrosis. While, sublethal doses of sarin resulted in delayed neuronal cell death via apoptosis in many areas of the brain.

3.) References: Abel-Rahman, A., A.K. Shett, and M.B. Abou-Donia (2002). Acute Exposure To Sarin Increases Blood Brain barrier Permeability and Induces Neuropathological Changes in the Rat Brain: Dose-Response Relationships. *Neuroscience*, 113:721-741.

Delayed Neurochemical and Neuropathological Alterations Induced by Sub-Acute Sarin Exposure in the Rat Brain

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We previously reported that acute exposure to an LD₅₀ dose of sarin induces seizure and early neuropathological changes in the adult rat brain 24 h after treatment. In the present study, we investigated the effects of a single intramuscular injection of sarin at doses of 1, 0.5, 0.1, and 0.01 X LD₅₀ (100 µg/kg) on the adult, male rat forebrain seven days after treatment. At this time-point both sarin-treated and vehicle-treated (controls) animals were analyzed for: (i) plasma butyrylcholinesterase (BChE) activity; (ii) brain acetylcholinesterase (AChE), (iii) m2 muscarinic acetylcholine receptor (m2mAChR) ligand binding; (iv) histopathological changes in the brain using hematoxylin and eosin (H&E) staining, microtubule-associated protein-2 (MAP-2) and glial fibrillary acidic protein (GFAP) immunostaining, TdT-mediated dUTP nick-end labeling (TUNEL) and ssDNA assays. None of the treatments caused any significant effect of BChE activity at this time-point. Only animals treated with 1 X LD₅₀ sarin exhibited significant inhibition of forebrain and brainstem acetylcholinesterase activity, seven days after treatment. Animals treated with either 1 or 0.5 X LD₅₀ sarin exhibited a significant decrease in forebrain and brainstem m2 mAChR ligand binding. These two doses also caused diffuse neuronal cell death, significant reduction in the number of healthy (or surviving) neurons with reduced MAP-2 immunoreactivity and increased GFAP expression in the motor cortex, hippocampus and the cerebellum. Seven days after treatment with either 1 or 0.5 X LD₅₀ sarin, there was a significant decrease in the density of surviving neurons in layers I-III and V of the motor cortex, granule cells of the dentate gyrus, pyramidal neurons of the hippocampal CA1 and CA3 subfields, and Purkinje of the cerebellum. This decrease in the number of surviving neurons was also associated with an increase in neuronal cell death in animals treated with 0.5 X LD₅₀ sarin compared to animals exposed to 1 X LD₅₀ sarin suggesting that neuronal cell death occurs earlier after higher doses of sarin. Furthermore, animals treated with 0.5 X LD₅₀ sarin exhibited a significant number of degenerating neurons undergoing apoptosis, shown by marked increase in cell positive (TUNEL) and detection of single stranded ssDNA throughout the motor cortex and the hippocampus. In contrast, neither the 0.1 or 0.01 X LD₅₀ treatment exhibited significant changes in the above brain regions. These results indicate that while early neuronal degeneration produced by a lethal dose (an LD₅₀) of sarin is related to its effect on inducing seizures and convulsions caused by its cholinergic and non-cholinergic actions, the non-lethal dose, 0.5 X LD₅₀ sarin results in delayed neuronal death via apoptosis. These changes could lead to severe sensorimotor and neurobehavioral abnormalities. Collectively, the above results indicate that sarin causes neuronal degeneration in many regions of the brain, which is dose-dependent and exacerbated with time. This work was supported by the U.S. Army Medical Research and Materiel Command under Contract Project Order DAMD17-98-C-8027.

Abstract Information & Notes

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Current Job Description:

Biomedical research; teaching medical students

Other Information (including titles of books or articles you have recently written):

10 authored books; 36 edited books; 750 published research articles; 350 published review articles and chapters; 58 research awards and prizes including 3 honorary doctor of medicine degrees

Disclosure Statement:

None

SPEECH TITLES: "Melatonin: Exogenous and Endogenous Sources and Actions"

The speaker has provided the information below.

1.) Goals and objectives: Goal: To identify dietary sources and sites of endogenous melatonin production and to define its effects. Objectives: To present data on the oncostatic actions of melatonin and to define the mechanisms of melatonin's interactions with damaging free radicals.

2.) Conclusion of what is to be learned: Melatonin is a ubiquitously distributed molecule with potent antioxidant and cancer inhibition effects.

3.) References: Allegra M, Reiter RJ, Tan DX, Gentile C, Tesoriere L, Livren MA. The chemistry of melatonin's interaction with reactive species. J Pineal Res 2003; Reiter RJ, Tan OX, Burkhardt S, Manchester LC. Melatonin in plants. Nutr Rev 2001; 59: 286-260.

MELATONIN: EXOGENOUS AND ENDOGENOUS SOURCES AND ACTIONS

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Melatonin is a very widely distributed molecule and is found in plants, unicellular organisms, insects and all vertebrate species including man. When ingested, melatonin is readily absorbed by the gastrointestinal tract and blood levels of the indole as well as urinary excretion of its major metabolite increase. Although relatively few edible foods have been studied as to their melatonin content, some of the foods in which melatonin has been measured in appreciable quantities include Montmorency tart cherries, walnuts, and a number of plants that are classified as medicinal herbs or used as food supplements.

In humans, melatonin is produced in the pineal gland, a small organ in the brain. Additionally, the retinas, gastrointestinal tract, leukocytes and some bone marrow cells produce melatonin. Melatonin, after its synthesis from the essential amino acid tryptophan, is released from the pineal gland in a circadian manner, with much higher levels being discharged during darkness at night than during the day. As a result, circulating blood levels of melatonin in humans are 10-30 times higher than daytime values. This marked circadian rhythm of melatonin strengthens other 24 hour slave oscillations which are essential for optimal health and physiology.

Light detected by the retinas at night, provided it is sufficiently bright and of the proper wavelength, immediately inhibits melatonin synthesis in the pineal gland and blood levels quickly fall to daytime values. Thus, any organ that "reads" the melatonin message is given inappropriate information as to time, i.e., even though it may still be night, the low melatonin levels indicate it is day and the responding organs alter their physiology to daytime levels. This misinformation contributes to malaise, reduced physiological performance and possibly alterations in the sleep/wake cycle. Thus, light is a "drug" by virtue of its ability to suppress melatonin at night. Examples of the gross misuse of light include the exposure of individuals to light during night shift work and transmeridian travel. Women who routinely work at night have an increased incidence of breast cancer and the acute consequences of transmeridian travel, i.e., jet lag, are well known. Whenever possible, light should be avoided at night. Humans evolved in an environment over thousands of years under a clearly defined light/dark cycle. This has been severely disrupted with the introduction of artificial light.

Since melatonin is a potent antioxidant, the misuse of light due to the inhibition of melatonin decreases the total antioxidative capacity of the body and increases the likelihood of molecular damage. Melatonin's antioxidant and free radical scavenging capacity are well documented. Melatonin directly detoxifies a number of oxygen and nitrogen-based radicals and radical-related products including singlet oxygen ($^1\text{O}_2$), hydrogen peroxide (H_2O_2), hydroxyl radical ($\bullet\text{OH}$), hypochlorous acid (HOCl) and the peroxynitrite anion (ONOO^-). These highly toxic agents are persistently produced in the body and several are by-products of oxygen (O_2), which is inhaled with every breath. Thus, while O_2 is necessary for survival, because a small percentage (1-4%) of it is metabolized to toxic metabolites, the use of this molecule gradually causes organisms to deteriorate, i.e., to age, as well as causes age-related diseases.

When melatonin scavenges radicals and related products it is converted to a number of other agents that also function as scavengers. Some of the major products that are formed when melatonin functions as a scavenger include cyclic 3-hydroxymelatonin, *N*¹acetyl-*N*²-formyl-5-methoxykynuramine (AFMK) and *N*¹acetyl-5-methoxykynuramine (AMK). The latter two molecules are now known to also be potent free radical scavengers.

Thus, not only in melatonin itself an efficient detoxifier of radicals and radical products, but its “offspring”, AFMK and AMK, are as well. This is referred to as the antioxidant cascade of melatonin.

Besides melatonin’s ability to function as a direct free radical scavenger, it has other actions which contribute to its ability to reduce the amount of molecular debris resulting from O₂ metabolites. Thus, melatonin stimulates a number of enzymes whose function it is to metabolize toxic reactants to innocuous by-products. Melatonin is known to stimulate the following antioxidative enzymes: glutathione peroxidase, glutathione reductase, catalase and both isoforms of superoxide dismutase. Finally, melatonin may promote the intracellular accumulation of another important antioxidant, glutathione, by stimulating the activity of gamma-glutamylcysteine synthase, the enzyme that rate limits glutathione production.

While melatonin may influence the onset and/or progression of several age-related diseases, its role in cancer will be discussed here. Melatonin has the capability of preventing or inhibiting tumor development and growth via several means. First, because of melatonin’s ability to scavenge free radicals, it protects DNA from oxidative mutilation. It is estimated that 75% of the cancer humans develop was initially the result of DNA damage by free radicals. Given that melatonin limits such damage, the result is a lower chance of developing a cancer. Once tumors are formed, melatonin also stymies their growth via a number of mechanisms.

Thus, melatonin has been shown to inhibit the activity of an enzyme, telomerase, in tumor cells. This enzyme maintains the telomere, extensions on the chromosomes of eukaryotic cells which allow the cells to survive and proliferate. By inhibiting the enzyme telomerase, melatonin may shorten the survival time of cancer cells.

Recent studies have shown that melatonin also causes the differentiation of immature cancer cells into more mature cells. In doing so, melatonin reduces the proliferative rate of cancer cells thereby limiting tumor size and the likelihood of metastasis.

Finally, melatonin, via receptor-mediated mechanisms, prevents the uptake of a tumor growth factor, i.e., linoleic acid by liver cancer cells. By lowering linoleic acid uptake, melatonin reduces its conversion to 13-hydroxyoctadecadienoic acid and the stimulation of mitogen activated protein kinase. When this intracellular cascade is slowed as a result of cancer cells being exposed to melatonin, tumor growth rate is likewise inhibited.

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- Vijayalaxmi, Thomas CR, Reiter RJ, Herman TS. Melatonin: from basic research to cancer treatment clinics. *J Clin Oncol* 2002; 20:2575-2601.

Case Information & Notes

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Disclosure Statement:

None

SPEECH TITLE: "Inflammatory & Allergic Triggers to the Inner Ear"

The speaker has provided the information below.

1.) Goals and objectives: To learn about the biochemical and immunologic influences in relation to patients with hearing loss and dizziness.

2.) Conclusion of what is to be learned: The regulation of inner ear biochemistry and immunology relates to the five body systems; 1) the adrenal gland; 2) the pituitary gland; 3) the hormonal system; 4) the immune system, and 5) the hypothalamus.

3.) References: Rubin, W: Site of lesion vestibular function testing. Laryngoscope 95:386-390, 1985
Rubin, W: How do we use state of the art vestibular testing to diagnose and treat the dizzy patient? An overview of vestibular testing and balance system integration. Neurol Clin 8:225-234, 1990
Rubin, W, Busis SM, Brookler KH: Otoneurologic examination. Otolaryngology 1:1-47, 1989

ENVIRONMENTAL ASPECTS OF INFLAMMATION

Balance and Hearing Symptoms, Evaluation and Treatment

Wallace Rubin, M.D.

The inner ear is a transducer of mechanical to electrical energy for both hearing and balance functions by means of chemicals present within the perilymph and endolymph. Maintenance of normal hearing and balance therefore is dependent upon the availability of the proper chemicals to perform this transduction task. The inner ear functions as an internal body organ because it is chemically dependent upon many body systems.

What are the body systems that are involved in regulating inner ear function? How do we evaluate these systems in relation to inner ear function? Most importantly, how do we apply this information to the treatment of inner ear abnormalities? After instituting treatment, how do we objectively monitor these inner ear problems so as to modify or change the treatment regime?

Five organ and glandular systems are involved in the regulation of inner ear biochemistry: (1) the adrenal gland, (2) the pituitary gland, (3) the hormonal system, (4) the immune system, and (5) the hypothalamus. Each of these organ systems secretes chemical messengers that interact with each other and with the chemicals that are being transported to the inner ear. The chemical environment of the inner ear, therefore, ultimately depends upon what we eat. Some of the chemical ingredients from our food pass through the intestinal wall into the blood stream. Some of the chemicals are then able to pass through the blood-brain barrier into the spinal fluid. Certain necessary chemicals then traverse the endolymphatic duct and sac to the inner ear fluids. The secretory capability of the inner ear contributes to this chemical process. Obviously, this whole mechanism of energy transduction within the inner ear can be faulty, depending on many general biochemical body functions.

Efficient diagnosis and management of hearing and balance problems that originate in the inner ear requires careful evaluation of the patient's complaints. The history is a vital part of the evaluation of a patient experiencing dizziness. It may be suggestive of a clinical diagnosis, but confirmation by physical examination and neurotologic testing is necessary to document and objectively assess an abnormality that is definitive and diagnostic.

Etiologic processes are responsible for auditory and vestibular symptoms. These mechanisms relate to the five interactive organ and glandular systems already named that influence chemical transmission to the inner ear. The functions that affect inner ear biochemistry are adrenal, pituitary, hormonal, immunologic, and hypothalamus abnormalities. These functions need evaluation with the following tests for proper diagnosis and management of neurotologic patients.

Anatomic testing: magnetic resonance imaging (MRI) and brain mapping.

Etiologic testing: biochemical and immunologic assessment.

1. Cholesterol
2. Triglycerides
3. Thyroid
4. Glucose tolerance response, ENG monitored
5. Blood urea nitrogen
6. Serum glutamic-oxaloacetic transaminase (SGOT)
7. Complete blood count (CBC)
8. Fluorescent treponemal antibody absorption (FTA-ABS)
9. Prolactin levels
10. Uric acid
11. Radioallergosorbent test (RAST) immunologic studies
12. Fasting blood sugar (FBS)
13. Circulating Immune Complex—Quantitative (C₁Q)
14. Raji Cell Assay (Immune Complex)
15. C₃,C₄ (Complement 3 and 4, serum)
16. Antinuclear antibodies (ANA), rheumatoid factor

The biochemical, metabolic, hormonal, and neurotransmitter influences, as they relate to hearing and balance problems, have just begun to be explored. The inner ear is, in fact, an internal body organ. The diagnostic and therapeutic direction for the evaluation of the neurotological patient should be oriented to confirm an etiological mechanism. This can be accomplished if our testing modalities are used in a way that is topographically diagnostic. This approach would then logically culminate in the systematic etiological investigation.

Mechanical energy signals that are processed and interpreted as sound, originate in the environment. Other mechanical energy signals occur as a result of body movements. These mechanical energy signals must be converted to electrical energy in order to be transmitted to the appropriate areas of the brain via the VIIIth cranial nerve. This conversion or transduction takes place in the inner ear. The transduction process is accomplished by the chemicals within the inner ear fluids.

Once this role is appreciated, the pathway and mechanisms necessary to transport chemicals ingested in our food to the inner ear become significant. It is this transport through the circulatory system to the cerebrospinal fluid system and then to the inner ear via the endolymphatic duct and sac, that is of significance in our thinking and testing for balance system etiological mechanisms. It is also important to recognize that the adrenal gland, the pituitary gland, the immune system, the hormonal system, and the hypothalamus also influence the chemical constituents of the inner ear fluids, as a result of their interrelated homeostatic mechanisms. These simple concepts but complicated mechanisms are important in deciding which testing procedures are applicable for use in determining etiological diagnoses in balance abnormalities.

The processing of the chemicals that originate in our food, and the transport of the chemicals to the inner ear fluids, involve a three-step obstacle course. These obstacles are the gut wall, the blood-brain barrier, and the endolymphatic sac and duct. At each step along the way, there is differential absorption that allows passage of only the necessary chemicals at the proper concentration for efficient inner ear function.

What then are the tests that need to be performed in order to determine the proper therapeutic regimen? After a complete history and physical examination have been carried out, the confirmatory tests are in the following groups: (1) audiological evaluation; (2) vestibular function testing; (3) imaging; (4) brain mapping; (5) biochemical, immunological, and hormonal testing.

References

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**22ND ANNUAL INTERNATIONAL SYMPOSIUM
ON MAN & HIS ENVIRONMENT**

SCHEDULE

Friday, June 25, 2004

- 8:25 **ANNOUNCEMENTS/MODERATOR: William J. Meggs, M.D., Ph.D.**
- 8:30 **Alexander Sivakou, M.D.**, Acupuncture Department, Minsk, Belarus, *Title: "Application of Electroacupuncture with Fluctuating current in Clinical Practice"*
- 8:50 Q & A
- 9:00 **Martha Stark, M.D.**, Department of Psychiatry, Harvard University Medical School, Newton Centre, MA, *Title: "The Role of Neuroinflammation in Depression"*
- 9:20 Q & A
- 9:30 **Jean Monro, M.D.**, Medical Director of Breakspear Hospital, Hemel Hempstead, Herts, England, *Title: "Causes of Inflammation Observed at Breakspear Hospital"*
- 9:50 Q & A
- 10:00 BREAK WITH EXHIBITORS**
- 10:30 **Savely Yurkovsky, M.D.**, Consultant in Cardiology, Internal & Bio-Energetic Medicine, Chappaqua, NY, *Title: "Toxicological and Biological Agents in the Pathogenesis of Cardiovascular and Chronic Diseases"*
- 10:50 Q & A
- 11:00 **Jack D. Thrasher, Ph.D.**, Consultant and Expert Witness, Toxicology and Immunotoxicology, Alto, NM, *Title: "Neural and Immune Abnormalities in Humans with a chronic and ongoing Exposure to Mixed Toxigenic Molds in a Water-Damaged Building"*
- 11:20 Q & A
- 11:30 **Andrew Campbell, M.D.**, Medical Director, Medical Center for Immune & Toxic Disorders, Spring, TX, *Title: "Inflammation and Immune Reactions Caused by Non Orthopedic Medical Implantable Devices"*
- 11:50 Q & A
- 12:00n OPEN LUNCH**
- MODERATOR: Wallace Rubin, M.D.**
- 1:30 **Professor Jerry Alter, Ph.D.**, Wright State University, Dept. of Biochemistry and Molecular Biology, Dayton, OH, *Title: "Scavenging Enzymes and Chemical Sensitivity"*
- 1:50 Q & A
- 2:00 **Bryan W. Brooks, Ph.D.**, Department of Environmental Studies, Waco, TX, *Title: "Pharmaceuticals as Contaminants of Aquatic Ecosystems"*
- 2:20 Q & A
- 2:30 **Savely Yurkovsky, M.D.**, Consultant in Cardiology, Internal & Bio-Energetic Medicine, Chappaqua, NY, *Title: "Novel Bio-Energetic System in the Diagnosis and Removal of Chronic Disease Pathogens"*
- 2:50 Q & A
- 3:00 BREAK WITH EXHIBITORS**
- 3:30 **Allan D. Lieberman, M.D.**, Medical Director, Center for Occupational & Environmental Medicine, PA, North Charleston, SC, *Title: "Manipulation of Cytokines in the Treatment of Environmental Disease"*
- 3:50 Q & A
- 4:00 **Larry Wolford, D.M.D.**, Academics, Private Practice, Research, Dallas, TX, *Title: "Metal Hypersensitivity in Complex TMJ Patients"*
- 4:20 Q & A
- 4:30 Panel Discussion: **Allan D. Lieberman, M.D.**, Medical Director, Center for Occupational & Environmental Medicine, PA, North Charleston, SC, **Richard G. Jaeckle, Ph.D.**, Private Practice of Psychiatry and Environmental Medicine, Presenting two case studies.
- 6:00 RECEPTION WITH THE EXHIBITORS**

FRIDAY, JUNE 25, 2004

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

Alexander Sivakou, M.D.

Date of talk: Friday, June 25, 2004, 8:30am

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Belarus. -2001.-~~1~~3.-P.23.
Patent 1046 A61N 1/32, 1/18. Method of Pain
Syndrome Treatment

Current Job Description:
Other Information (including titles of books or articles
you have recently written):

Disclosure Statement:

None

SPEECH TITLE: "Application of Electroacupuncture with Fluctuating Current in Clinical Practice"

The speaker has provided the information below.

1.) Goals and objectives: The main aim of the research was the development and approbation of the new way of physiotherapy - electroacupuncture of fluctuating current (EAFC). The essence of this method is in simultaneous effecting by the needle (acupuncture method) and fluctuating current (FC).

2.) Conclusion of what is to be learned: On the base of morphological, physiological and clinico-instrumental data a new method of complex effecting by acupuncture and fluctuating current was invented and developed, which in it's treating and physiological effecting differs from acupuncture and fluctuarisation as well. The EAFC method with outgoing modulation up to 24 V and duration of 20 min doesn't harm the soft tissues and nerve fibers at the place of the injected needle.

3.) References: None

Application of Electroacupuncture with Fluctuating Current in Clinical Practice

Alexander Sivakou, M.D.

The usage of physical factors in acupuncture (the so called physiopuncture) can be regarded as one of the perspective and special ways of effect increase in contemporary physiotherapy. Moreover, it's worth to be mentioned, that a limited number of physical factors is used for physiopuncture nowadays, which narrows the possibilities of this method. Said above lets us to consider the development of new ways of physiopuncture methods and methods of optimization for medical usage to be one of the very actual problems in physiotherapy, which has defined the aim of this work to a big extend.

The main aim of the research was the development and approbation of the new way of physiotherapy - electroacupuncture of fluctuating current (EAFC). The essence of this method is in summultaneous effecting by the needle (acupuncture method) and fluctuating current (FC). The steel needles are injected till the canonical depth, until the foreseen feelings were gained in the patients acupuncture points. The effecting lasts for 10 - 25 minutes with the going out modulation up to 12 V. FC is a special kind of electrical signal in which frequency and amplitude changes chaotically. Neuromuscular apparatus didn't adaptated to such kind of electrical signal, that is why the FC is very effective in pain syndroms treatment.

For this purpose we produced a special apparatus "ALFA - ELECTRO" (Salde 1). This apparatus has the following electrical regimes: 1) fluctuating current, 2) non-modulated impulse current and 3) sinusoidal-modulative current with low frequency. This apparatus also has laserpuncture regimes. Range of modulation frequency for electroacupuncture and laserpuncture is: from 0 to 9,9 Hz (with the step 0,1 Hz - it gives the possibility to provide electroacupuncture treatment according to R.Voll method); from 11 to 999 Hz (with step 1 Hz).

Conventional signs on Slide 1:

- 1 - emitter of laser radiation;
- 2 - on/ off button (also uses to start procedure);
- 3 - on/off indication button;
- 4 - parameter set button (two identical);
- 5 - value of parameters (+1 and - 1) buttons;
- 6 - indicator;
- 7 - external line supply connector - 6 - 18 V
- 8 - output for electroacupuncture and electropuncture.

The following tasks have been regarded, while completing the research :

- 1) to learn and substantiate the possibility and necessity of the complex usage of the acupuncture and fluctuating current;
- 2) to state the morphological electrical safety and anti-inflammatory effect of this methods;
- 3) to define the reaction of the peripheral nerve, while using acupuncture and EAFC;
- 4) to research the anesthetic EAFC effect;
- 5) to investigate clinically the method at the patients with the pain syndrome, related to vertebrogenic lumbo-sacral radiculitis.

According to the stated aims we performed morphological experiments (76 rats, 10040 researches), physiological researches at healthy people and clinical researches at 100 patients with vertebrogenic lumbo-sacral radiculitis. The morphological picture after the EAFC with outgoing voltage modulation of 10 - 24 V was the following. The blood fullness of the vessels was observed at the animals (Slide 2), also a little swelling of perimyrium. The blood fullness of the vessels was observed at the most at the third day (You can see it at this slide) and reduces till the end of the 5-th day. (Slide 3) Nerve fibers at the 1st 3rd and the 5th days after the effecting by EAFC, as you can see at this slide, have normal structure. Their contours are smooth, vivid, without swellings. (Slide 4) While effecting by EAFC with the outgoing modulation of 45 V, at the 1-st days the blood fullness of the vessels is more vivid, swelling perimyrium also. (Slide 5) Diffusive inflammation infiltration appears at that period. (Slide 6) Dystrophic changes slightly increased and were shown more vivid by the end of the 3-rd day (EAFC with the modulation 60 V). In this period of time, as you can see at this slide, (Slide 7) a swelling of endomyrium and perimyrium are vivid, sarcoplasm of the muscle fibers become cloudy, (Slide 8) and the horizontal striation hardly distinguished, the nucleus were pale. (Slide 9) In some spots the fragmentation of the muscle fibers was observed. The phenomenon of necrobiosis were kept even at the 5-th day. The nerve fibers of this group of the animals stayed normal, as a rule. (Slide 10) Only sometimes, very rarely, among those at the 3-rd day after the effecting, right at the place of the injection of the needle, fibers with the active signs can be met as uneven contours. (Slide 11) While effecting by EAFC with the outgoing voltage modulation of 90 V, more strong dystrophic changes were shown. Immediately after effecting the necrosis of muscle and nerve fibers appears. All soft tissues in effecting zone was coagulated. So, the absence of inevitable morphological changes of the soft tissues in the region of the injected needle and good antiinflammation effect after EAFC with the outgoing modulation of 10 - 24 V served as an experimental base for clinical approbation of this method of treatment.

The anesthetic influence of the electroacupuncture is widely known. The method of EAFC, as it seems to us, is the modification of this method. For the statement of the anesthetic influence of EAFC at the physiological stage of the research, made at healthy volunteers, the range of the skin sensitivity towards electrical current at the place of acupuncture and EAFC effectings was studied. While choosing the parameters we were led by the results of the morphological research and the literature data on acupuncture analgesia possessed by us. While EAFC the points with the general innervation were used VB-34 and VB-39. (Slide 12) The results of this research is presented at this slide. It is very well seen that after placebo effecting (curve 1) there was a tendency see to a slight increase and then after acupuncture effect (curve 2) to a slight decrease of the sensitivity and the zone the influence. The dynamics of the sensometric after the EAFC (curve 3) was characterized by the reliable vivid reduction of the sensitivity of the effecting place with the gradual return to its base level in 120 - 150 minutes. Consequently, according to sensometrical research, EAFC causes more vivid skin sensitivity reduce at the effected place than classical acupuncture.

According to the results of the next stage of physiological research (Slide 13) acupuncture and EAFC change basic functional state of the peripheral nerve, which is proved by the decreasing of the amplitude after effecting. Acupuncture influence to the point MC-6 (projection of the nervus medianus) decreases the amplitude of electrical potential till $15\pm 3\%$ (first to impulses on slide) and the effecting by the EAFC - till $76\pm 4\%$ (the last impulse on slide).

The proposed above results of experimental research give as a ground expedition of the EAFC applying with the outgoing modulation up to 12 V for pain syndroms treatment.

The clinical stage of the research was provided at 100 patients with vertebrogenial lumbo-sacral radiculitis with one-sides showing L5 - S1 roots. While objective examinations of the patients different disorders of the reflects, sensitivity and moving spheres were found out, which be typical for this illness. Sensitivity disorders, more often as hypesthesia or paresthesia, scoliosis were presented with 80% of the patients, positive symptom of Lassegas - with 94%. The results with the X-ray and with a functional load and CT revealed the signs of spine-osteochondrosis with 100 of the patients. At 69 patients before and after treatment the studying of the bioelectrical activation of brain and hemodynamics of the lower limbs was provided. The examination of blood circulation at the microcirculative level was done with the radionuclear diagnostics and the macrocirculative level with the reovazographic method. To define the optimal parameters of the effecting all the patients were divided into 3 groups. In the 1st and the 2nd groups EAFC with outgoing modulation 12 to 24 V was applied and at the 3rd (control group) only acupuncture was used. It is important to mention, that in all 3 groups effecting was applied only to the limbs with the pain syndrome.

The complex acupuncture and fluctuation current improved the major blood circulation data (pulse volume) and microcirculation (period of semi leading out Na 131 J) as the limb with the pain syndrome and the limb without the pain syndrome as well (Slide 14). At this slide you can observe that after EAFC the period of semi leading out at the limbs with the pain syndrome decrease from 13 to 6 min, and at the limb without the pain syndrome - from 9.5 min to 6.5 min. Together with the tissue blood circulation improvement at this example a positive dynamics of neurological symptomatics is observed.

On the base of morphological, physiological and clinico-instrumental data a new method of complex effecting by acupuncture and fluctuating current was invented and developed, which in it's treating and physiological effecting differs from acupuncture and fluctuarisation as well. The EAFC method with outgoing modulation up to 24 V and duration of 20 min doesn't harm the soft tissues and nerve fibers at the place of the injected needle. The method can be recommended for treatment of patients with the neurological signs of the lumbo-sacral radiculites. The comparison data of the clinico- neurological examination, multicriteria analysis of the hemodynamics of the lower limbs make it possible to think, that for these patients it is very reasonable to apply EAFC with the outgoing current with the 12 V. The method of EAFC is characterized as an easy, accessible, well handled by the patients, highly therapeutically effective, it deserves to be introduced in the wide medical practice.

Abstract Information & Notes

Martha Stark, M.D.

Date of talk: Friday, June 25, 2004, 9:00am

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Current Faculty Appointments:

American Association of Psychiatric Medicine
Harvard Medical School; Center for Psychoanalytic Studies, Massachusetts General Hospital; Boston Psychoanalytic Institute; Massachusetts Institute for Psychoanalysis

Current Job Description:

teaching and full private practice in psychiatric medicine and psychoanalysis

Other Information (including titles of books or articles you have recently written):

Stark, M. (1994). **Working with Resistance**. Northvale, NJ: Jason Aronson, Inc. Stark, M. (1994). **A Primer on Working with Resistance**. Northvale, NJ: Jason Aronson, Inc. Stark, M. (1999). **Modes of Therapeutic Action: Enhancement of Knowledge, Provision of Experience, and Engagement in Relationship**. Northvale, NJ: Jason Aronson, Inc.

Disclosure Statement:

None

SPEECH TITLE: “The Role of Neuroinflammation in Depression”

The speaker has provided the information below.

1.) Goals and objectives: (a) understand the role of an activated hypothalamic-pituitary-adrenal axis and elevated levels of cortisol in neuroinflammation and depression; (b) explicate the significance of pro-inflammatory cytokines in the etiology of depression; (c) recognize the importance of abnormal neuronal transmission in the induction of depression appreciate the myriad interrelationships between / amongst the endocrine system, the immune system, and the nervous system in the development of depression

2.) Conclusion of what is to be learned: (a) understand that (endogenous) depression is a very real medical problem that involves alteration in brain function and metabolism; (b) appreciate the role played by stress (both the “presence of bad” and the “absence of good”) in the etiology of depression; (c) recognize the positive correlation between an activated immune response, hypersecretion of cortisol, chronic low-level neuroinflammation, and chronic depression; (d) appreciate that depression speaks, ultimately, to a state of dyshomeostasis, whereby the body’s homeostatic regulatory mechanisms have become so disrupted (because of the chronic low-level stress to which the hypothalamus is being subjected) that the brain is no longer able to function properly; (e) explicate the implications for treatment

3.) References:

Bland J. **Genetic Nutritioneering**. Lincolnwood, IL: Keats Publishing; 1999.
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THE ROLE OF NEUROINFLAMMATION IN DEPRESSION

Martha Stark, M.D. - Friday, June 25, 2004

distinction between **depression (a disease serious enough to be associated with actual biochemical changes in the brain)** and the mental anguish that accompanies the transient "downs" that are an inevitable part of life's journey or that is associated with, say, the grief of bereavement

depression - just as real as chemical sensitivity and environmental illness

some public health experts are predicting that **depression**, currently the fourth major cause of disability in the United States, will become the **second major cause of disability by the year 2020** (US Dept of Health and Human Services 1993)

we don't understand the internal workings of the brain nearly as well as we understand the internal workings of the body

in the literature on stress and depression, reference is repeatedly made to **the association between stress, depression, inflammation, and elevated levels of cortisol**

in response to stress, the immune system is mobilized, macrophages (in the body) and microglia (in the brain) are activated, and copious amounts of proinflammatory cytokines are released -- resulting in inflammation (in the body) and neuroinflammation (in the brain)

but also in response to stress, the endocrine system is mobilized, the HPA axis is activated, and corticosteroids are released -- resulting in elevated levels of the body's most powerful anti-inflammatory hormone, namely, cortisol

the **net result** (at least as it relates to the brain) is **depression**

but **how to understand this?** -- the **simultaneous presence (in stress-induced depression) of excess proinflammatory chemical mediators and excess anti-inflammatory chemical mediators**

is this a manifestation of homeostasis or, perhaps, homeostasis gone awry, namely, dyshomeostasis?

adaptation or, perhaps, maladaptation?

compensation or, perhaps, decompensation?

as if this weren't confusing enough, **sometimes stress-induced depression is correlated with these increased levels of cortisol and sometimes with decreased levels of cortisol** - how to account for this?

also the issue of chicken? or egg? what causes what?

is depression cause, correlate, or consequence of inflammation?

is it cause, correlate, or consequence of aberrant levels of cortisol?

also, with respect to the inflammation to which stress gives rise: **ordinarily, inflammation is thought to be an ingenious adaptation that allows the body to defend itself against real or imagined danger** -- even something like sunburn is the body's attempt to repair damage inflicted by ultraviolet radiation and fever is the body's attempt to kill off invading microorganisms by "burning them alive"

so **inflammation**, as with all the body's defenses, has a **piece that is adaptive, but if it continues unabated (especially in the brain), doesn't inflammation, in time, also become part of a larger problem that may lead, ultimately, to chronic disease and degeneration?**

in other words, whereas the inflammation was initially part of the body's stress response to a pre-existing stressor, in time doesn't the inflammation itself become an additional stressor?

and the **cortisol** mobilized in response to stress -- **should it really be designated a "stress" hormone or, perhaps more accurately, an "anti-stress"**

hormone, inasmuch as its function is to help the body deal with the stress it is experiencing -- although here too, with time, the cortisol may itself become part of the problem

and then there's the **mystery shrouding the relationship between depression and the immune response** -- it would seem that **depression is associated with both immune suppression (in the form, say, of decreased neutrophil phagocytosis and natural killer cell activity) (Wallenstein 2003) and immune stimulation (in the form of**

elevated levels of macrophages, elevated levels of proinflammatory cytokines, and elevated levels of acute phase proteins)

how to understand this "dual" response to stress of the body's immune system (both suppression and stimulation)?

furthermore, reference is repeatedly made (at least by neuroscientists) to the association between stress, depression, and increased levels of catecholamines but (by psychiatrists) to stress, depression, and decreased levels of catecholamines

on the one hand, we know that in response to stress, the nervous system is mobilized -- more specifically, the sympathetic branch of the autonomic nervous system is activated, which stimulates the release of epinephrine (adrenaline) and norepinephrine (noradrenaline) from the adrenal medulla

but then there is the well-established catecholamine hypothesis of depression, which has it that depression is often correlated with decreased levels of catecholamines -- a theory in existence for almost 40 years and the very foundation upon which rests the treatment of depression with the classic antidepressants (which increase the "functional" levels of the body's monoamines by either preventing their breakdown, preventing their reuptake, or increasing their synthesis)

in fact, the original 1965 paper by Joseph Schildkraut, in which he advanced these seminal ideas, is the most frequently cited of all articles ever published in the American Journal of Psychiatry and one of the most frequently cited papers in all of psychiatry

does this apparent discrepancy between increased levels of catecholamines on the one hand and decreased levels on the other speak, perhaps, to different kinds of depression? (the first involving agitation, the second involving depletion)

or, maybe, to different time frames? (the increased levels of catecholamines reflecting an earlier stage when the body is still fighting to restore its balance, the decreased levels reflecting a later stage when the body, exhausted from its efforts, has given up)

as I envision it, when the body is functioning well, it is in a state of dynamic equilibrium, opposing but more or less equal "forces" enabling it to operate within an optimal (but tightly regulated) range

proinflammatory chemical mediators in a delicate balance with anti-inflammatory chemical mediators

sympathetic activation of the body's organ systems in a delicate balance with their parasympathetic activation

homeostasis describes this ongoing regulatory process of compensatory micro-adjustments, whereby a healthy body is enabled to maintain (or, if lost, to restore) a steady internal biochemical and physiological balance -- for every action, a reaction

if there is either "too much" or "too little" of a particular variable, then the body will experience stress and, in its infinite wisdom, will activate whatever compensatory mechanisms it must in order to bring that variable back into line

in other words, when stressors are encountered that cause the body to deviate, then homeostatic processes (involving, often, a sequence of "cascading" actions and compensatory reactions) will become mobilized so that the body can correct for the aberration

master-minded by the hypothalamus (a pea-sized but extraordinarily important neural structure centrally located in the floor of the brain) and implemented by an intricate array of highly sophisticated interconnecting networks and complex positive and negative feedback loops between (and within) the immune, the endocrine, and the nervous systems, these homeostatic adjustments enable the body to return to a state of optimal functioning (McEwen 1987, Chalmers 1993)

this multi-directionality of influence - possible because the cells of all three regulatory systems have receptor sites on their membranes, which enable their activities to be modified by immune mediators (cytokines), endocrine mediators (hormones and releasing factors), and neural mediators (neurotransmitters)

particularly disruptive to homeostasis is the impact of cumulative stress

and if "too much" or "too little" of something persists for "too long," then there may come a time when the body (once able to adapt to the stress by mobilizing all its reserves) is now no longer able to compensate

the net result of this chronic stress: dyshomeostasis, maladaptation, depletion, exhaustion, organ dysfunction, chronic disease, degeneration

in mainstream medicine, the focus tends to be on the patient's symptoms, the goal being alleviation of those symptoms (by way of such symptom-relieving medications as antipyretics, antitussives, and antihypertensives, to name a few)

in environmental medicine, however, the focus tends to be on what underlies the patient's symptoms, the goal now being alleviation of the environmental stresses (both those externally derived and those internally generated--both those psychosocial and those physical) that have given rise to the patient's symptoms -- so that the body's optimal homeostatic functioning can once again be restored

in my own (psychoanalytic) writings on the subject of the impact of environmental stresses on a person's mental health, I have found it **useful to conceptualize this impact as involving both the "presence of bad" (that is, too much that is disease-promoting) and the "absence of good" (that is, not enough that is health-promoting)**

I believe that this paradigm is equally useful for conceptualizing the "environmental stresses" with which environmental medicine is concerned, namely, both the "presence of bad" (too much that is "toxic") and the "absence of good" (not enough that replenishes the body's resources)

the recovery of health and homeostatic equilibrium will involve the alleviation of stress by way of both ridding the system of that which is "bad" and supplementing the system with that which is "good"

but **whether the "event" involves too much that is "bad" (which contributes to the total body load) or not enough that is "good" (which contributes to the total body deficit), the body will experience the event as a stressor -- to which the body must attempt to adapt in order to restore internal equilibrium and homeostasis**

if this cannot be accomplished, dyshomeostasis, maladaptation, and chronic illness result

Hans Selye's 1936 "adaptation to stress" model speaks directly to the body's homeostatic efforts to manage the stressors to which it is being exposed

first is the **alarm stage of the acute stress response (characterized by heightened arousal and mobilization of the body's defenses either to fight or to flee)**

if the stress persists, then the body transitions into a stage of resistance (characterized by the body's mobilization of all its defenses in an effort to "adapt" to the stress)

in this stage of adaptation, the body, although compromised in its functioning, is still putting up a good **fight to correct the imbalance and recover its internal homeostasis**

but if the stress continues indefinitely, there comes a time when the body, despite its most heroic efforts, "defaults" into a stage of exhaustion (characterized by the body's depletion of resources)

at this point, the body is no longer able to defend, no longer able to resist, no longer able to adapt -- and simply collapses, exhausted -- because overstimulation leads ultimately and inevitably to inhibition of function **chronic disease sets in, and we speak of the body as being in a depleted, dyshomeostatic state**

with respect to the role played by neuroinflammation in depression:

I would like now to present the **theory of depression advanced by the immunologist Robert Smith (at the University of Chester) to explain its etiology**

in 1991, Smith put forth his "macrophage theory of depression," in which he postulated the following cascade of inflammatory events:

a stressor stimulates the immune system (macrophages in the body, microglia in the brain) to proliferate and to release proinflammatory cytokines (especially IL-1, IL-6, and TNF-alpha) into the body and brain

these chemical messengers (the proinflammatory cytokines) also activate the HPA axis, stimulating release of CRF by the hypothalamus, which is transported in the bloodstream to the anterior lobe of the pituitary gland, where CRF stimulates the release of ACTH, which is transported in the bloodstream to the adrenal cortex, where ACTH stimulates the release of cortisol (synthesized from cholesterol) into the bloodstream

Smith's macrophage theory of depression has it that the immunological abnormalities are primary, the neuroendocrine abnormalities secondary, and depression the net result

but this still doesn't explain **why depression is also sometimes correlated with decreased levels of cortisol**

furthermore, we still have **no way to understand why depression would seem to be correlated both with sympathetic nervous system activation (and elevated levels of catecholamines) and with depletion of the sympathetic nervous system (and depressed levels of catecholamines)**

in order to explain the pathogenesis of what would seem to be two different kinds of depression, **I have therefore formulated my own theory**, one that draws directly upon both Smith's ideas and Selye's ideas:

Stage 1, the alarm stage of stress, is an acute stage, characterized by activation of the immune, the endocrine, and the nervous systems

activation of the immune system results in an acute inflammatory process

activation of the HPA axis results in elevated levels of cortisol (secreted by a stimulated adrenal cortex) and activation of the sympathetic nervous system results, amongst other things, in elevated levels of catecholamines (secreted by a stimulated adrenal medulla)

whereas the catecholamines are short-acting stress hormones, the corticosteroids are long-acting stress hormones

interestingly, it has been hypothesized that **stressful early life experiences may predispose to the development of depression in later life by "sculpting" specific pathways in the brain such that the HPA axis becomes "entrained" to override the "negative feedback inhibition" ordinarily provided by the cortisol to the brain -- the result of which is chronic hyperactivation of the HPA axis, habitualized oversecretion of cortisol, and chronic depression**

in any event, **during the first stage, we have (simply) acute stress, arousal, acute inflammation, an activated HPA axis, elevated cortisol, excess sympathetic nervous system activity, and elevated catecholamines**

if the stress continues, the **body moves into the resistance stage of stress, a stage of adaptation and compensation, designed to maintain homeostasis by way of mobilizing all the body's defenses, all the body's reserves**

this is the "early chronic stage" of stress, a transitional stage that can last from weeks to months to years and is characterized by chronic stress, chronic low-grade inflammation, a chronically activated HPA axis, chronically elevated cortisol, a chronically activated sympathetic nervous system, chronically elevated catecholamines, and now, I believe, an agitated depression with psychomotor agitation, anxiety, mood swings, and irritability

eventually, **if the stress persists long enough and the body becomes tired enough, the body may transition into the third stage, the "late chronic stage," characterized by maladaptation to chronic stress, depletion, and a gradual exhaustion of the body's compensatory mechanisms**

whereas the second stage was marked by "dysfunction" in the levels of chemical messengers released by the various organ systems, the third stage is marked by "dysfunction" in the organs themselves

this third and final stage, then, is a stage of adrenal exhaustion and therefore depleted levels of cortisol and a stage of sympathetic nervous system exhaustion and therefore depleted levels of catecholamines

although not often discussed in the literature, this stage of exhaustion is, I believe, also often associated with dysfunction in the thyroid and therefore depleted levels of thyroid hormones -- which would explain why thyroid supplementation can sometimes have a significant impact on treatment-resistant depressions, potentiating the effects of antidepressants (both orthodox and unorthodox)

in any event, **whereas the early chronic stage of stress was associated with an agitated depression (secondary to neuroinflammation and elevated levels of cortisol and catecholamines), this late chronic stage of stress is associated, I contend, with a depleted depression (secondary still to neuroinflammation but now depleted levels of cortisol and catecholamines) -- thus the psychomotor retardation, anhedonia, vegetative symptoms and signs, lethargy, and despair**

and **whereas in the second stage of adaptation, the brain was still struggling to recover its internal equilibrium, in this third stage of maladaptation, the brain collapses into dyshomeostasis characterized by both chronic neuroinflammation and, now, neurodegeneration**

Robert Sapolsky - studied the detrimental effect on the hippocampus of prolonged exposure to elevated levels of cortisol

the hippocampus, part of the temporal lobe, is the memory center of the brain and home to the largest number of glucocorticoid receptors in the body

chronic overstimulation by cortisol can serve as a slow-acting nerve toxin, resulting in compromised function of the hippocampal neurons and their eventual suicide by way of apoptosis

brain imaging studies have demonstrated actual shrinkage in size of the hippocampus

neurodegeneration of part of the brain as a result of chronic stress and hypersecretion of cortisol -- with resultant learning and short-term memory problems (Sapolsky R and McEwen B 1998)

in any event, this way of conceptualizing the two kinds of depression (as representing different stages in the body's response to stress and therefore as represented by different underlying biochemical markers) would also account for why depression is said to be correlated with both psychomotor agitation and psychomotor retardation, increased appetite and decreased appetite, weight gain and weight loss, hypersomnia and insomnia -- crucial distinctions with profound treatment implications, crucial distinctions too often overlooked by psychiatrists

this model would also explain why the **classic antidepressants (TCAs, MAOIs, and SSRIs) are more effective with some depressions than with others**

indeed, research findings confirm that TCAs, MAOIs, and SSRIs -- all of which enhance monoamine activity -- are more useful in depleted depressions (characterized, as they are, by depressed levels of monoamines) than in agitated depressions (with their elevated levels)

in the psychiatric literature, depression is often presented as a monolithic syndrome

but of note is the fact that **sometimes a clinical distinction is made by psychopharmacologists between "melancholic" depressions (which respond well to antidepressant medications) and "atypical" depressions (so-named because they are particularly treatment-resistant)**

bear in mind (Kirsch and Antonuccio) that even when used for depleted depressions, more than half the clinical trials sponsored by the pharmaceutical companies themselves have failed to demonstrate significant differences in effectiveness between antidepressants and placebos, a fact referred to as a "dirty little secret" by several clinical trial researchers (Hollon) but considered by FDA officials to be "of no practical value to either patient or prescriber" (Leber)

in any event, if, indeed, stress in the brain gives rise to a stress response culminating in depression and, along the way, involves (1) activation of the immune system and neuroinflammation; (2) activation of the endocrine system resulting in elevated levels of cortisol, and (3) activation of the sympathetic nervous system and elevated levels of catecholamines, then this "cascading" sequence of events will generate a number of options for novel and unorthodox approaches to treatment (and prevention) of depression

to be effective, **each such approach must target either some aspect of the original stress or some aspect of the response to the stress, which, as we have seen, becomes itself an added stress if it goes unchecked for too long. In other words, although adaptive in the short term, inflammation, elevated levels of cortisol, and sympathetic overactivation will, in the long run, become maladaptive**

in closing: to return to what we'd said earlier, **every strategic intervention should attempt either to decrease the "bad" or to increase the "good"**

by way of example, important to cut back on the consumption of proinflammatory omega-6 fatty acids at the same time that the storehouses of anti-inflammatory omega-3 fatty acids are being replenished

I will close with a comment about my **two favorite strategies for combating depression -- sleep deprivation and aerobic exercise**

neuroscientists don't really know how to explain the mystery of why depriving yourself of half a night's sleep once a week (preferably the second half of the night) would have such a beneficial effect on depression -- as has been demonstrated by numerous studies (Leibenluft 1992)

it has been hypothesized, however, that **interrupting normal sleep patterns may "resynchronize disturbed circadian rhythms," producing a rapid and sustained recovery from depression, an effect potentiated by aerobic activity (Pflug 1976, Ratey 2004)**

in fact, in 1999, a **team of researchers at the Duke University Medical Center** demonstrated that (in the middle aged and the elderly) **aerobic exercise is at least as effective as medication in treating major depression**

but, interestingly, they discovered an **additional benefit** as well--namely, **improved cognitive ability, particularly in the frontal and prefrontal regions of the brain** as Woody Allen once remarked, "My brain is my second favorite organ."

References for THE ROLE OF NEUROINFLAMMATION IN DEPRESSION
Martha Stark, M.D. - Friday, June 24, 2004

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THE ROLE OF NEUROINFLAMMATION IN DEPRESSION

Martha Stark, M.D. - Friday, June 24, 2004

Goals and objectives:

By the end of the presentation, each participant should be able to

- a. understand the role of an activated hypothalamic-pituitary-adrenal axis and elevated levels of cortisol in neuroinflammation and depression**
- b. explicate the significance of pro-inflammatory cytokines in the etiology of depression**
- c. recognize the importance of abnormal neuronal transmission in the induction of depression**
- d. appreciate the myriad interrelationships between / amongst the endocrine system, the immune system, and the nervous system in the development of depression**

Conclusion of what is to be learned:

- a. understand that (endogenous) depression is a very real medical problem that involves alterations in brain function and metabolism**
- b. appreciate the role played by stress (both the "presence of bad" and the "absence of good") in the etiology of depression**
- c. recognize the positive correlation between an activated immune response, hypersecretion of cortisol, chronic low-level neuroinflammation, and chronic depression**
- d. appreciate that depression speaks, ultimately, to a state of dyshomeostasis, whereby the body's homeostatic regulatory mechanisms have become so disrupted (because of the chronic low-level stress to which the hypothalamus is being subjected) that the brain is no longer able to function properly**
- e. explicate the implications for treatment**

Abstract Information & Notes

Jean Monro, M.D.

Date of talk: Friday, June 25, 2004, 9:30am

Breakspear Hospital
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Medical School/University Attended:

London Hospital Medical School, England

Residency:

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Board Certifications:

MB, BS, MRCS, LRCP, RAAEM, DipIBEM, MACOEM

Current Faculty Appointments:

Professor in the Dept. of Integrative Medicine, Capital University of Integrative Medicine, Washington, D.C.

Current Job Description:

Medical Director of Breakspear Hospital, England

Disclosure Statement:

A pilot study using the ALCAT system was paid for by the MiniMag distributors.

SPEECH TITLE: “Causes of Inflammation Observed at Breakspear Hospital”

The speaker has provided the information below.

1.) Goals and objectives: To illustrate that food sensitivities are not merely immunological abnormalities, but are heightened sensitivities, which can be mitigated by electromagnetic frequencies. All people have the ability to perceive electromagnetic frequencies and are influenced by these. Equally everyone is affected by food and other environmental impacts.

2.) Conclusion of what is to be learned: The management of the combined effects of the above can be neutralizing therapy and/or magnetic field therapy.

3.) References: 1. Miller JB. Food allergy: provocative testing and injection therapy. Springfield, IL: C.C. Thomas; 1972.

2. Monro JA. Biological effects of neutralizing vaccines. In: Clements-Croome D, ed. Electromagnetic environments and health in buildings. London: spon Press; 2004. p.267-80.

3. Fell PJ, Soulsby S, Brostoff J. Cellular responses to food in irritable bowel syndrome – an investigation of the ALCAT test. J Nutr Med 1991;2:143-9.

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ABSTRACT FOR 22ND ANNUAL INTERNATIONAL SYMPOSIUM ON

JUNE 24 – 27 2004

Patients presenting at Breakspear Hospital have a huge variety of reactions to environmental incitants, which result in chronic inflammatory disease. Inflammation is, of course, mediated through the immune system and biochemical responses, but food sensitivities themselves may not strictly be an immunological response discerned by an antibody evaluation.

The ALCAT system has been shown to determine food sensitivities, as opposed to food allergies (see Reference 1).

The patients at Breakspear Hospital have sometimes been shown to have positive RAST tests, sometimes positive reactions by elimination and challenge, or sometimes positive reactions to the neutralising technique (see Reference 2). This will be illustrated.

We used the ALCAT test to identify sensitivities and then to prove that there can be an electromagnetic component. We used an electromagnetic field therapy (“MiniMag” magnetic field therapy apparatus) in a group of patients to show mitigation of the food sensitivity responses. We were able to show a change in reaction after 1 hour and a further change following 1 week of continued therapy.

Pulsed electromagnetic fields are able to rectify the abnormal responses that people have to everyday food and chemical encounters. This illustrates a fundamental component of the nature of man’s responses to his environment and, therefore, is critical in the management of patients with chronic inflammatory disorders.

Jean A. Monro

Medical Director

Abstract Information & Notes

Savely Yurkovsky, M.D.

Date of talk: Friday, June 25, 2004, 10:30am

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Medical School/University Attended:
Internship:
Residency:
Board Certifications:
Current Job Description:
Other Information (including titles of books or articles
you have recently written):

II Moscow State Medical Institute, USSR
Coney Island Hospital
Coney Island Hospital
Internal Medicine
Private Practice; Teaching
Book: Biological, Chemical, and Nuclear Warfare –
Protecting Yourself and Your Loved Ones: The Power
of Digital Medicine. Articles: Multiple Chemical
Sensitivity – From Treatments to Cure: The Spleen
Epidemic and Non-Disease Treatment of Diseases;
Thyroid Gland: Cures, Fallacies and Fixes; Infectious
Diseases: What Are We Treating?; Autism, ADD,
ADHD, LD and The Power of Digital Medicine;
Guided Digital Medicine, The Law of Unintended
Consequences and Non-Disease Treatment of
Diseases. In the process of authoring a textbook on
Scientific Principles for the practice of Integrative
Medicine, “A Textbook of New Medicine.”

Disclosure Statement:

“SYY Integrated Health Systems, Ltd” – a distributor
of educational materials & bio-energetic diagnostic
items.

SPEECH TITLE: “Toxicological and Biological Agents in the Pathogenesis of Cardiovascular and Chronic Diseases”

The speaker has provided the information below.

1.) Goals and objectives: To apprise of the multisystemic invasive nature of both the toxicological agents and their inevitable consequence – opportunistic fungal infections. “Trojan horse” phenomenon – fungal organisms as the inconspicuous reservoirs and carriers of heavy metals in the body.

2.) Conclusion of what is to be learned: Toxicological and/or fungal infections as playing a preeminent role in the pathogenesis of the majority of chronic, degenerative and acute diseases. High potential for serious iatrogenicity from the current prevailing therapies of candidiasis.

3.) References: Chang, Louis W., “Toxicology of Metals,” CRC Press, Inc., 1996;
Casarett and Doull’s “Toxicology: The Basic Science of Poisons,” Curtis D. Klaassen, Curtis, 1995, 5th Ed., McGraw-Hill; Calderone, Richard A., “Candida & Candidiasis, ASM Press, 2002; Yurkovsky, Savely, <http://www.yurkovsky.com/>, “The Slow AIDS – The Epidemic of Multisystem Candidiasis and Dangers of its Current Treatments.” (Due April 2004).

Toxicological and Biological Agents in the Pathogenesis of Cardiovascular and Chronic Diseases

I. BRIEF OVERVIEW OF VASCULAR PHYSIOLOGY

Cell Types Implicated in the Vasculotoxic Response

CELL TYPE	FUNCTION
Endothelial cells	First barrier to blood borne toxins; synthesis and release of endothelium-derived relaxing factor; synthesis of pro- and antiaggregatory factors; attachment and recruitment of inflammatory cells; synthesis of connective tissue proteins; generation of oxygen-derived free radicals and other radical entities
Smooth muscle cells	Maintenance of vasomotor tone; synthesis of extracellular matrix proteins, including collagen and elastin; synthesis of prostaglandins and other biologically active lipids; regulation of monocyte function; formation of free radicals
Fibroblasts	Synthesis of extracellular matrix proteins, including collagens; structural support to the vessel wall
Monocytes/macrophages	Scavenger potential; synthesis of macrophage-derived growth factor; generation of reactive oxygen species; lymphocyte activation; progenitor of foam cells
Platelets	Synthesis of proaggregatory substances and smooth muscle mitogens such as platelet-derived growth factor
Lymphocytes	Release of activated oxygen species; cellular immunity; production of immunoglobulins

II. VASCULAR PATHOGENESIS, MAJOR MECHANISMS:

1. Direct endothelial cell injury
2. Luminal medial smooth muscle cells
3. Small vessel injury
4. Combined

III. VASCULOTOXIC AGENTS

1. Alkylamines
 - a) allylamine
 - b) aldehydes: acrolein, acetaldehyde, formaldehyde
2. Heavy metals and metals

- Arsenic
- Beryllium
- Cadmium
- Chromium
- Cobalt
- Copper
- Indium
- Lead
- Magnesium
- Manganese
- Mercury
- Selenium
- Thallium
- Vanadium
- Zinc
- 3. Aromatic Hydrocarbons
- 4. Gases
 - Carbon Monoxide
 - Oxygen
 - Carbon Disulfide
 - 1,3-Butadiene
- 5. **Pharmaceuticals**
 - a) Antibacterial-Antimitotic agents
 - Cyclophosphamide
 - 5-Fluorodeoxyuridine
 - Gentamicin
 - b) Sympathomimetic amines:
 - epinephrine
 - norepinephrine
 - dopamine
 - isoproterinol
 - amphetamines
 - c) Cocaine
 - d) Antineoplastic agents
 - 5-fluorouracil
 - doxorubicin
 - mitomycin
 - e) Analgesics and non-steroidal anti-inflammatory agents
 - f) Oral contraceptives
 - g) Contrast dyes
 - h) Phosphodiesterase inhibitors
- 6. Natural products
 - a) Bacterial Endotoxins
 - b) Hydrazinobenzoic Acid
 - c) Trichotece Mycotoxins
- 7. Opportunistic Fungal infections

IV. RELEVANT MULTI-ORGAN EFFECTS OF TOXICANTS::

- 1. Brain
- 2. Myocardium
- 3. Kidney
- 4. Liver
- 5. Lungs
- 6. Others

V. “SOFT” SCIENCE BEHIND “SAFE LEVELS” OF TOXICOLOGICAL AGENTS

- 1) Individual meaning
- 2) Dose-effect and dose-response relationships
- 3) Inherent limitations of regulatory toxicology

VI. LIMITATIONS OF THE CURRENT DIAGNOSTIC TOXICOLOGICAL ASSESSMENTS

- 1) Inaccessibility of the relevant tissues
- 2) Potentially misleading nature of the accessible samples
- 3) Issue of the key or dominant toxin

VII. LIMITATIONS OF THE CURRENT DETOXIFYING THERAPEUTIC APPROACHES

- 1) Lack of sufficient tissue penetration
- 2) Inadequate organ support
- 3) Lack of specific therapeutics for many intoxicants

VIII. CONCEPT OF HUMAN PHYSIOLOGY AS ONE OF A BIOLOGICAL QUANTUM SYSTEM

IX. BIO-RESONANCE DIAGNOSIS IN THE DIAGNOSIS OF TOXICOLOGICAL AND BIOLOGICAL AGENTS.

- 1) modalities
- 2) advantages
- 3) practitioner-related limitations

X. HOMEOPATHY IN THE TREATMENT OF TOXICOLOGICAL AND BIOLOGICAL AGENTS

The use of:

- 1) Isodes – energetic imprints of the actual pathogenic agents
- 2) Autoisodes – energetic imprints of the intoxicated or infected bodily fluids
- 3) Sarcodes – energetic imprints of organs and tissues
- 4) Other beneficial actions

XI. PRESENTATION OF DOCUMENTED CLINICAL CASES

CONCLUSIONS

1. The current prevailing diagnostic methods suffer from formidable limitations in establishing the key intoxicants within the body of an individual and, as importantly, the extent of their presence.
2. The prevailing therapeutics, likewise, lack in specificity and effectiveness.
3. Skillful applications of bio-resonance testing affords rapid, safe, low cost and non-invasive diagnosis of the relevant pathogens that play a major role in cardiovascular and other degenerative diseases.
4. Homeopathic system presented renders very effective, safe and low cost therapy in the management of acute and chronic toxicants and a wide range of chronic diseases.

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Abstract Information & Notes

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Medical School/University Attended:

UCLA School of Medicine, Department of Anatomy, Ph.D., 1964

Faculty Positions:

University of Colorado, School of Medicine, Department of Anatomy, 1964-1966; UCLA School of Medicine, Department of Anatomy; 1966-1972

Current Faculty Appointments:

Retired from Teaching

Current Job Description:

Consultant and Expert Witness, Toxicology and Immunotoxicology

Other Information (including titles of books or articles you have recently written):

Book: (1990) *The Poisoning of Our Homes and Workplaces: The Indoor Formaldehyde Crisis*. Santa Ana, Seadora, Inc.

Recent Research: (2003) Antibodies to Molds and Satratoxin in Individuals Exposed in Water-Damage buildings; Mixed Mold Exposure: Immunological Changes in Humans with Exposure in Water Damaged Buildings; On the neuropsychological and electrocortical impacts of mixed mold exposure; Neural antigen autoantibodies and neurophysiology abnormalities in patients exposed to mixed-molds in water-damaged buildings; (2004) Abnormalities in Gulf War Veterans: Studies with Lymphocyte Numbers, Lymphocyte Function and Antibodies. *Environmental Health Perspectives*

Disclosure Statement:

Immunosciences Lab, Inc. – Consultant, Medical Center for Immune & Toxic Disorders - Consultant

SPEECH TITLES: “Neural and Immune Abnormalities in Humans with a chronic and ongoing Exposure to Mixed Toxicogenic Molds in a Water-Damaged Building”

The speaker has provided the information below.

1.) Goals and objectives:

2.) Conclusion of what is to be learned:

3.) References:

Neural and Immune Abnormalities in Humans with a chronic and ongoing Exposure to Mixed Toxicogenic Molds in a Water-Damaged Building.

Jack D. Thrasher¹
Andrew Campbell²
Aristo Vojdani³
Roberta A. Madison⁴
Michael R. Gray⁵

First Draft of Manuscript

¹*Sam-1 Trust, Alto, New Mexico*

²*Medical Center for Immune and Toxic Disorders, Spring, Texas*

³*Immunosciences Laboratories, Beverly Hills, California*

⁴*Department of Health Sciences, California State University, Northridge*

⁵*Progressived Health Care Group, Benson Arizona*

Corresponding Author:

Jack D. Thrasher, PH.D., Sam-1 Trust, P.O. Box 874, Alto, New Mexico 88312, (505) 336-8317

Abstract

33 patients (48.2±10.3 yrs, 7 males, 26 females) underwent physical exams, electro-diagnostic and immunologic testing concurrent to and within days (32.8±14.6 days) following their chronic exposure (39.6±3.3 months) to mixed molds in a water-damaged building. The patients had a preponderance of neurological symptoms, e.g. 100 % with CNS and PNS complaints. Other symptoms included severe fatigue (75%), shortness of breath and chest tightness (75%) and recurrent flu-like illness (60.7%). Neurological testing revealed abnormalities as follows: sensory neuropathy (43.5%) sensory motor polyneuropathy (30.4%), brainstem auditory evoked response (BAER) abnormalities (55.5%); optic nerve dysfunction (9.8%) and abnormal EEGs (9.8%). Several of the patients had a combination of neurological dysfunction. Immune tests revealed autoantibodies against neural antigens: myelin basic protein (MBP), ganglioside GM1 and sulfatide. Comparison to health controls revealed the following immune abnormalities: 1) increased C3 and C4 complements and immune complexes (IgG, IgM and IgA) compatible with inflammatory conditions; 2) Increased T and B cells markers and helper/suppressor ratio, indicating a relative lymphocytosis and immune activation; 3) Mitogenesis to PHA, ConA, PWM and LPS was significantly elevated over controls. However, three distinct mitogen responses were found as follows: suppression, elevation and highly elevated mitogenesis to ConA, PWM and LPS. Natural Killer cell activity was suppressed in 42.4% of the patients. It is concluded that these patients with an ongoing exposure to molds had developed neurological dysfunction and pathology, while at the same time were undergoing continuous antigenic stimulation resulting in the above immune findings.

Abstract Information & Notes

Andrew Campbell, M.D.

Date of talk: Friday, June 25, 2004, 11:30am

Medical Center for Immune & Toxic Disorders
25010 Oakhurst, Ste. 200
Spring, TX 77386

Phone: 281/681-8989
Fax: 281/681-8787
E-mail: md@immunotoxicology.com

Medical School/University Attended:

Universidad Autónoma de Guadalajara, México

Internship:

Orlando Regional Medical Center, Orlando, Florida

Residency:

Medical College of Georgia, Augusta, Georgia

Board Certifications:

ABFP, ABME, ABFM

Current Job Description:

Medical Director, Medical Center for Immune & Toxic Disorder

Other Information (including titles of books or articles you have recently written):

Published four articles for **The Scientific World Journal**, in 2003, and has published many other articles for various publications since 1992. A list will be available upon request.

Disclosure Statement:

None

SPEECH TITLE: "Inflammation and Immune Reactions Caused by Non Orthopedic Medical Implantable Devices"

The speaker has provided the information below.

1.) Goals and objectives:

2.) Conclusion of what is to be learned:

3.) References:

Inflammation and Immune Reactions Caused by Non Orthopedic Medical Implantable Devices

Andrew W. Campbell, M.D.

Medical Director

Medical Center for Immune and Toxic Disorders

25010 Oakhurst Drive

Spring, Texas 77386

281 681-8989

md@immunotoxicology.com

Medical Implants:

Medical implants are used in various parts of the body to replace diseased joints or enhance appearances. The materials used in the manufacturing of medical implantable devices have been known to cause immune reactions, some of them severe. In this presentation, the inflammatory and immunologic responses to silicone implants and silicone gel implants will be discussed.

Abstract Information & Notes

Jerry Alter, Ph.D.

Date of talk: Friday, June 25, 2004, 1:30am

Wright State University, Dept. of Biochemistry and
Molecular Biology
School of Medicine, College of Science and Mathematics
234 Biological Sciences
Dayton, OH 45435-0001

Phone: 937/775-2504
Fax: 937/775-3730
E-mail:

Medical School/University Attended:

Ph.D., Washington State University, 1975
Postdoc, Harvard Medical School, 1975-1978

Current Faculty Appointments:

Associate Professor of Biochemistry

Current Job Description:

Director, Biomedical Sciences Ph.D. Program
Coordinator, Initiative for Biological Computation

Disclosure Statement:

None

SPEECH TITLE: "Scavenging Enzymes and Chemical Sensitivity"

The speaker has provided the information below.

1.) Goals and objectives: We have investigated Multiple Chemical Sensitivity in humans and measured the activity of organophosphate and formaldehyde metabolizing enzymes in the blood fractions of control and chemically sensitive populations.

2.) Conclusion of what is to be learned: We have obtained evidence indicating that chemically sensitive individuals can be distinguished from control individuals by the activities of the enzymes under investigation. Further, we can distinguish between formaldehyde and organophosphate sensitive and control populations based on the activity of selected esterase and dehydrogenase enzymes.

3.) References:

Scavenging Enzymes and Chemical Sensitivity

Jerry Alter, Ph.D.

We have hypothesized that Multiple Chemical Sensitivity (MCS) in humans is linked with abnormal activity levels of the enzymes capable of metabolizing those specific compounds. To test this assertion for formaldehyde sensitivity and organophosphate sensitivity, we have examined the activity of four enzymes in both chemically sensitive and control populations. The enzyme activities examined which are important for detoxifying and metabolizing formaldehyde and organophosphates include: aldehyde dehydrogenase (ALDH), chi alcohol dehydrogenase (χ ADH), paraoxonase (PON), and aryl esterase (AE). In a pilot study, we collected blood, hair, and saliva samples from individuals to determine the best, readily accessible tissues in which to monitor the levels of our targeted activities. Blood samples were separated into fractions enriched in RBCs, WBCs, and serum prior to analyses. Methods and conditions for quantifying activities of each enzyme in each fraction and sample were identified. These assays were based on continuous spectroscopic monitoring of NAD production of proton release during the respective enzymatic reactions. Preliminary studies indicated that blood was the only suitable tissue for such an analysis. The same studies indicated in our assays were capable of reproducibly measuring each individual's enzyme activities with standard deviations less than 10% of the average activity. However, we also observed substantial variation among individuals, preventing clear delineation between activities of sensitive and control individuals on the basis of the pilot study. Based upon results from the study, we collected blood samples from 69 individuals, 20 individuals in a control population and 49 individuals in test populations, using a double blind protocol. Thirty three individuals in the test population were screened for sensitivity by the Environmental Health Foundation in Dallas Texas, and sixteen individuals were diagnosed with chemical sensitivity at clinics in either Dayton, Ohio, or Buffalo, New York.

Results for the latter study indicate that enzyme activities we measured do not correlate with gender, ethnicity (within the limited groups we analyzed), or age. Similarly, chemical sensitivity did not seem to segregate based on these factors. Plots of each individual's activities in all fractions that we measured, i.e., "enzyme profiles", indicate that chemically sensitive individuals generally have lower activity levels than control group individuals. Further, the shape of these profiles among chemically sensitive and control group individuals, is somewhat different, indicating relative activities may differ among these groups. However, plots of individual's chi alcohol dehydrogenase activity versus aldehyde dehydrogenase activity clearly discriminate between normal and formaldehyde sensitive groups. Similarly, plots of individual's aryl esterase versus paraoxonase activities discriminate between normal and organophosphate sensitive groups. These results establish a link between the clinically accessible biochemical markers, i.e., specific blood enzyme activities, and chemical sensitivity. Further, our results indicate different chemical sensitivities, i.e., formaldehyde as opposed to organophosphate, are linked to different enzymatic markers. Finally, the enzymatic markers identified are rational in terms of the metabolism of the chemical toxicants we studied. This information may be clinically useful to help identify or confirm diagnosis of chemical sensitivity. Our results also suggest possible links between the expression of chemical sensitivity and the micro nutrient state of individuals.

Abstract Information & Notes

Bryan W. Brooks, Ph.D.

Date of talk: Friday, June 25, 2004, 2:00pm

Baylor University
Department of Environmental Studies
P.O. Box 97266
Waco, TX 76798

Phone: 254/710-6553
Fax: 254/710-3409
E-mail: Bryan_Brooks@Baylor.edu

Medical School/University Attended:

Ph.D. – University of North Texas; MS – University of Mississippi

Current Faculty Appointments:

Baylor University, Waco, TX

Current Job Description:

Assistant Professor of Environmental Studies

Other Information (including titles of books or articles you have recently written):

Co-author of 3 articles currently at press in 2004.

Disclosure Statement:

None

SPEECH TITLE: “Pharmaceuticals as Contaminants of Aquatic Ecosystems”

The speaker has provided the information below.

1.) Goals and objectives: Present a review of knowledge on pharmaceuticals and personal care products as environmental contaminants. Identify major sources, environmental disposition, and effects of PPCPs in aquatic systems.

2.) Conclusion of what is to be learned: Prioritization of critical research areas in study of pharmaceuticals as emerging environmental contaminants.

3.) References:

- Boxall, A.B.A., Fogg, L.A., Blackwell, P.A., Kay, P., Pemberton, E.J. & A. Croxford, 2004. Veterinary medicines in the environment. *Rev Environ Contamin Toxicol* 180:1-91.
- Brooks, B.W., Foran, C.M., Richards, S., Weston, J.J., Turner, P.K., Stanley, J.K., Solomon, K., Slattery, M. & T.W. La Point, 2003. Aquatic ecotoxicology of fluoxetine. *Toxicol Lett* 142: 169-83.
- Daughton, C.G. & T.L. Jones-Lepp (Eds), 2001. *Pharmaceutical and Personal Care Products in the Environment: Scientific and Regulatory Issues*. ACS symposium series 791, American Chemical Society, Washington, DC. 396 pp.
- Daughton, C.G. & T.A., Ternes, 1999. Pharmaceuticals and personal care products in the environment: agents of subtle change? *Environ Health Perspect* 107 (Suppl. 6): 907-938.
- Huggett DB, Cook JC, Ericson JF, Williams RT. 2003. A theoretical model for utilizing mammalian pharmacology and safety data to prioritize potential impacts of human pharmaceuticals to fish. *Human Ecol Risk Assess* 9: 1801-1812.
- Koplin DW, Furlong ET, Meyer MT, Thurman EM, Zaugg SD, Barber LB, Buxton HT. 2002. Pharmaceuticals, hormones, and other organic wastewater contaminants in US streams, 1999-2000: A national reconnaissance. *Environ Sci Technol* 36: 1202-1211.
- Kummerer, K. (Ed.), 2001. *Pharmaceuticals in the Environment: Sources, Fate, Effects and Risks*. Springer, Berlin. 265pp.
- Metcalf CD, Miao XS, Koenig BG, Struger J. 2003. Distribution of acidic and neutral drugs in the surface waters near sewage treatment plants in the lower great lakes, Canada. *Environ Toxicol Chem* 22: 2881-2889.
- Schulman, L.J., Sargent, E.V., Naumann, B.D., Faria, E.C., Dolan, D.G. & J.P. Wargo, 2002. A human health risk assessment of pharmaceuticals in the aquatic environment. *Human Ecol Risk Assess* 8: 657-680.
- Webb, S., Ternes, T., Gilbert, M., & K. Olejniczak, 2003. Indirect human exposure to pharmaceuticals via drinking water. *Toxicol Lett* 142: 157-167.

Title: Pharmaceuticals as Contaminants of Aquatic Ecosystems

Author: Bryan W. Brooks, Ph.D.

Abstract:

I. Goals and Objectives

The objective of this presentation is to provide a state-of-the-science review on pharmaceuticals and personal care products (PPCPs) as environmental contaminants, including information on major sources, environmental fate and disposition, and effects of PPCPs in aquatic systems. The goal is to provide timely information on an emerging area in environmental science to environmental health professionals.

II. Outline

A. This presentation will review:

1. Water quality regulations in the U.S.
2. Water quantity considerations in the south central and southwestern U.S.
3. Assessment of contaminant impacts on aquatic organisms
4. Current regulatory approaches for pharmaceuticals as environmental contaminants
5. “Hot spots” of PPCP introduction to the environment, specifically focusing on municipal effluent-dominated streams in the south central U.S.
6. Information on occurrence and fate of PPCPs in surface waters

B. This presentation will review several studies by our research group:

1. Identification of estrogenic compounds in Denton TX treated effluent
2. Effects of β -adrenergic receptor blockers on aquatic invertebrates and fish
3. Effects of the selective serotonin reuptake inhibitor fluoxetine on microorganisms, aquatic invertebrates and fish
4. Determination of select SSRIs in aquatic organisms

C. This presentation will identify major research gaps in the study of environmental PPCPs

1. Regulatory gaps
2. Research gaps
 - A. Risk Assessment procedures
 - B. Analytical procedures
 - C. “Non-target” consequences
 - D. Ecosystem responses: structural, functional
3. Education needs

III. Conclusions

Given numerous uncertainties regarding PPCPs as environmental contaminants, future studies should focus on environmental occurrence and fate of PPCPs, and their effects at multiple levels of biological organization. Biomarkers of exposure and effect are required to understand mechanistic toxicity of PPCPs to “non-target” aquatic organisms. Regulatory guidance is needed in the U.S. to prioritize immediate and future research.

IV. References

Boxall, A.B.A., Fogg, L.A., Blackwell, P.A., Kay, P., Pemberton, E.J. & A. Croxford. 2004. Veterinary medicines in the environment. *Rev Environ Contamin Toxicol* 180: 1-91.

Brooks, B.W., Riley, T., & R.D. Taylor. 2004. Water quality of effluent-dominated stream ecosystems: ecotoxicological, hydrological, and management considerations. *Hydrobiologia*. In Press.

Brooks, B.W., Richards, S.M., Weston, J., Turner, P.K., Stanley, J.K., La Point, T.W., Brain, R., Glidewell, E.A., Massengale, A.R.D., Smith, W., Blank, C.L., Solomon, K.R., Slattery, M., & C.M. Foran. 2004. Aquatic ecotoxicology of fluoxetine: a review of recent research. In: *Advances in Pharmacology: Hot Spot Pollutants*. Vol 52, *Pharmaceuticals in the Environment*. D. Dietrich, S. Webb and T. Petry (Eds.). Academic Press. (In Press)

Brooks, B.W., Foran, C.M., Richards, S., Weston, J.J., Turner, P.K., Stanley, J.K., Solomon, K., Slattery, M. & T.W. La Point. 2003. Aquatic ecotoxicology of fluoxetine. *Toxicol Lett* 142: 169-83.

Daughton, C.G. & T.L. Jones-Lepp (Eds). 2001. *Pharmaceutical and Personal Care Products in the Environment: Scientific and Regulatory Issues*. ACS Symposium Series 791, American Chemical Society, Washington, DC. 396 pp.

Daughton, C.G. & T.A. Ternes. 1999. Pharmaceuticals and personal care products in the environment: agents of subtle change? *Environ Health Perspect* 107 (Suppl. 6): 907-938.

Foran, C.M., Weston, J.J., Slattery, M., Brooks, B.W. & D.B.Huggett. 2004. Reproductive assessment of Japanese medaka (*Oryzias latipes*) following a four-week fluoxetine exposure. *Arch Environ Contamin Toxicol* In Press.

Huggett DB, Cook JC, Ericson JF, Williams RT. 2003. A theoretical model for utilizing mammalian pharmacology and safety data to prioritize potential impacts of human pharmaceuticals to fish. *Human Ecol Risk Assess* 9: 1801-1812.

Kolpin DW, Furlong ET, Meyer MT, Thurman EM, Zaugg SD, Barber LB, Buxton HT. 2002. Pharmaceuticals, hormones, and other organic wastewater contaminants in US streams, 1999-2000: A national reconnaissance. *Environ Sci Technol* 36: 1202-1211.

Kummerer, K., (Ed.). 2001. *Pharmaceuticals in the Environment: Sources, Fate, Effects and Risks*. Springer, Berlin. 265 pp.

Metcalf CD, Miao XS, Koenig BG, Struger J. 2003. Distribution of acidic and neutral drugs in the surface waters near sewage treatment plants in the lower great lakes, Canada. *Environ Toxicol Chem* 22: 2881-2889.

Schulman, L.J., Sargent, E.V., Naumann, B.D., Faria, E.C., Dolan, D.G. & J.P. Wargo. 2002. A human health risk assessment of pharmaceuticals in the aquatic environment. *Human Ecol Risk Assess* 8: 657-680.

Webb, S., Ternes, T., Gilbert, M., & K. Olejniczak, 2003. Indirect human exposure to pharmaceuticals via drinking water. *Toxicol Lett* 142: 157-167.

Abstract Information & Notes

Savely Yurkovsky, M.D.

Consultant in Cardiology, Internal & Bio-Energetic
Medicine
37 King Street
Chappaqua, NY 10514

Date of talk: Friday, June 25, 2004, 2:30pm

Phone: 914/861-9161
Fax: 914/861-9160
E-mail: info@yurkovsky.com
Website: www.yurkovsky.com

Medical School/University Attended:
Internship:
Residency:
Board Certifications:
Current Job Description:
Other Information (including titles of books or articles
you have recently written):

II Moscow State Medical Institute, USSR
Coney Island Hospital
Coney Island Hospital
Internal Medicine
Private Practice; Teaching
Book: Biological, Chemical, and Nuclear Warfare –
Protecting Yourself and Your Loved Ones: The Power
of Digital Medicine. Articles: Multiple Chemical
Sensitivity – From Treatments to Cure: The Spleen
Epidemic and Non-Disease Treatment of Diseases;
Thyroid Gland: Cures, Fallacies and Fixes; Infectious
Diseases: What Are We Treating?; Autism, ADD,
ADHD, LD and The Power of Digital Medicine;
Guided Digital Medicine, The Law of Unintended
Consequences and Non-Disease Treatment of
Diseases. In the process of authoring a textbook on
Scientific Principles for the practice of Integrative
Medicine, “A Textbook of New Medicine.”

Disclosure Statement:

“SYY Integrated Health Systems, Ltd” – a distributor
of educational materials & bio-energetic diagnostic
items.

SPEECH TITLE: “Novel Bio-Energetic System in the Diagnosis and Removal of Chronic Disease Pathogens”

The speaker has provided the information below.

1.) Goals and objectives: To present evidence that the true origin of diseases can be effectively diagnosed and treated through the most fundamental homeostatic domain in man – energy. Presentation of the reversed cases with the serious and diverse pathologies.

2.) Conclusion of what is to be learned: Physicians are capable of producing superior clinical results regardless of the site of pathology or nosological entity, upon implementation of the bio-energetic medical system presented.

3.) References: Yurkovsky, Savely, “Biological, Chemical, and Nuclear Warfare – Protecting Yourself and Your Loved Ones: The Power of Digital Medicine, Science of Medicine Publishing, 2003; Tiller, William A., “Science & Human Transformation: Subtle Energies, Intentionality and Consciousness,” Paxior Publishing, 1997; Oshman, James L., “Energy Medicine, The Scientific Basis,” Churchill Livingstone, 2000; McTaggart, Lynne, “The Field: The Quest for the Secret Force of the Universe,” Harper-Collins, 2001; Yurkovsky, Savely, <http://www.yurkovsky.com/>, “Guided Digital Medicine, The Laws of Unintended Consequences and Non-Disease Treatment of Diseases – Part I.”

Abstract Information & Notes

Allan D. Lieberman, M.D.

Date of talk: Friday, June 25, 2004, 3:30pm

Center for Occupational & Environmental Medicine, PA
7510 Northforest Dr.
North Charleston, SC 29420-4297

Phone: 843/572-1600
Fax: 843/572-1795
E-mail: allanl@coem.com

Medical School/University Attended:

Chicago Medical School

Internship:

Mt. Sinai Hospital – Chicago

Residency:

Children’s Memorial Hospital – Chicago

Board Certifications:

Fellow-American Academy of Environmental
Medicine, American Board of Environmental Medicine

Current Faculty Appointments:

Braun University Department of Biochemistry –
Assistant Professor

Current Job Description:

Medical Director of the Center for Occupational &
Environmental Medicine

Other Information (including titles of books or articles
you have recently written):

Disclosure Statement:

None

SPEECH TITLE: “Manipulation of Cytokines in the Treatment of Environmental Disease”

The speaker has provided the information below.

- 1.) Goals and objectives:** To introduce the concept of regulatory systems manipulation using Cytokine Therapy.
- 2.) Conclusion of what is to be learned:** Cytokine manipulation maybe the up and coming state of the art treatment for environmentally triggered disorders.
- 3.) References:**

MANIPULATION OF CYTOKINES IN THE TREATMENT OF ENVIRONMENTAL DISEASE

Empirically, medical science discovered that we could alter disease by using varying molecules, drugs and even neutraceuticals. How this happened was not apparent until modern technology identified the presence of the two systems of the immune response classified as the innate and adaptive responses. The latter consisted of the T cells and B cells which responded to information provided by the innate system made up of macrophages, neutrophils, and natural killer cells by producing molecular signals or Cytokines. These low molecular weight glycoproteins regulate and coordinate the immune responses.

We now know that the naïve CD4 T cell can be directed to go in different directions, each resulting in different consequences. Modulation of each group of cytokines regulates the balance between protection and immunopathology in the host.

The CD4 cell is recognized as being differentiated into 5 identifiable cells with only 3 having major significance:

TH1 – involved primarily in cell mediated immunity

TH2 – involved in production of antibody and proliferation of eosinophiles

TH3 – Produces transforming growth factor-beta

The TH1 driven cell mediated immunity protects against intracellular pathogens and viruses but also has a negative side implicated in organ specific autoimmunity. While the TH2 driven humoral immunity produces antibody for local or barrier immunity it too has a negative side inducing an allergic response. Thus it can be seen that a balance must be maintained between their protective and their immunopathologic effects. The cytokines themselves are the dominant regulators of T helper cell differentiation.

My first interest in this complex topic began after reading the paper: Therapeutic Effects of BCG Vaccination in Adult Asthmatic Patients: a randomized controlled trial by CHOI & KOH (Annals of Allergy, Asthma & Immunology 2002; 88:384-591)

The conclusion of this paper was that BCG vaccination improved asthma and was accompanied by a suppressed TH2 type immune response. Manipulating the direction the immune system will go offered a therapeutic benefit in allergic and autoimmune disease.

Cases will be presented to show how influencing cytokine actions could alter some of the complex problems we see in everyday practice.

Abstract Information & Notes

Larry Wolford, D.M.D.

Baylor University School of Dentistry
3409 Worth Street, Suite 400
Dallas, TX 75246

Date of talk: Friday, June 25, 2004, 4:00pm

Phone: 214/828-9115
Fax: 214/828-1714
E-mail: lwolford@swbell.net

Medical School/University Attended:

Temple University School of Dentistry, Philadelphia, PA

Internship:

University of Texas Southwestern Medical School and Parkland Hospital, Dallas, TX

Residency:

Same

Board Certifications:

Oral and Maxillofacial Surgery

Current Faculty Appointments:

Clinical Professor Baylor College of Dentistry

Current Job Description:

Academics, Private Practice, Research

Other Information (including titles of books or articles you have recently written):

Co-Authored 2 text books, written 19 book chapters for other books, published over 145 articles.

Disclosure Statement:

None

SPEECH TITLES: “Metal Hypersensitivity in Complex TMJ Patients”

The speaker has provided the information below.

1.) Goals and objectives: Demonstrate presence of Hypersensitivity in TMJ patients with chronic irresolvable pain. Show difference in outcomes with the use of different types of TMJ prostheses.

2.) Conclusion of what is to be learned: Chemical Sensitivity to constituent metals in total joint prosthesis TMJ patients is highly prevalent in patients with chronic irresolvable pain. Metal on metal articulating prostheses contribute to poor treatment outcomes.

3.) References: See abstract

Metal Hypersensitivity in Complex TMJ Patients

By

Larry M. Wolford, D.M.D.
Pushkar Mebra, B.D.S., D.M.D.
William Rea, M.D.

Purpose: To evaluate hypersensitivity to constituent materials in TMJ total joint prostheses as a contributory factor for irresolvable chronic pain in patients with total joint prostheses.

Patients and Methods: The study sample consisted of 26 female patients with an average age of 42.6 years (range 20 to 55) with chronic irresolvable pain. Twenty patients had preexisting Christensen total joint TMJ prostheses, 3 patients had Vitek-Kent prostheses, 1 patient had a Techmedica total joint prosthesis, and 2 patients had no previous TMJ surgery. The Christensen and Vitek-Kent total joint prostheses are cast from chromium-cobalt alloy, which contains chromium, cobalt, molybdenum, nickel, and other trace elements. The TMJ Concepts (previously Techmedica) total joint prosthesis is made of commercially pure wrought titanium and wrought titanium alloy, with articulating surfaces of polyethylene and wrought chromium cobalt alloy. All 26 patients were tested for chemical sensitivity by intradermal injections of each specific element using a serial dilution titration technique. Intradermal injection of normal saline was used as a negative control, and histamine was used as a positive control. Testing was performed for each of the following materials: Chromium, Cobalt, Molybdenum, Nickel, Titanium, Aluminum, Vanadium, and Polyethylene. Following testing, patients with pre-existing prostheses had the devices removed. All 26 patients were reconstructed with the TMJ Concepts total joint prosthesis and fat grafts. Pre and post reconstruction pain levels were assessed with visual analog scale (VAS) scores.

Results: Severe hypersensitivity to nickel was seen in 25 to 26 patients (96%); 9 of 26 patients (35%) were moderately sensitive to chromium; 5 of 26 patients (19%) were mildly sensitive to titanium, and 3 patients (12%) were mildly sensitive to titanium, and 3 patients (12%) were mildly sensitive to aluminum. No patients were hypersensitive to cobalt, vanadium, molybdenum, or polyethylene. Average length of follow-up after reconstruction was 16 months (range 11 to 34). Average subjective VAS TMJ pain score prior to TMJ reconstruction with TMJ Concepts prosthesis and fat grafting was 8.2 (range 6 to 10) where 0 = no pain and 10 = worst pain imaginable. After reconstruction, the average TMJ pain score was 4.9 (range 3 to 8).

Conclusions: Chemical hypersensitivity to constituent metals, especially nickel, in total joint prostheses is highly prevalent in patients with chronic irresolvable pain. Prostheses made of cast chromium-cobalt alloy containing nickel (Christensen and Vitek-Kent devices) exposes patients to these materials, and may contribute towards poor treatment results. The use of prostheses made with wrought biocompatible metals and minimal nickel content, combined with peri-implant fat grafting to decrease direct tissue exposure to metals, improves treatment outcomes.

References:

1. Rea WJ: Chemical Sensitivity. CRC Press Inc., Florida. Vol 3, pages 1727-1884, 1997
2. Wolford LM: Temporomandibular Joint Devices: Treatment factors and outcomes. Oral Surg Oral Med Oral Path 83 (1): 143, 1997

Case Information & Notes

Richard G. Jaeckle, Ph.D.

8220 Walnut Hill Lane, Suite 404
Dallas, TX 75231

Date of talk: Friday, June 25, 2004, 4:30 pm

Phone: 214/696-0964

Fax:

E-mail: rgjmd@airmail.net

Medical School/University Attended:

Internship:

Residency:

Board Certifications:

Current Job Description:

University of Texas Southwestern Medical School

Veterans Administration Hospital, Dallas, TX

Psychiatry: St Louis Univ Hospitals & Child

Psychiatry: Washington Univ Child Guidance Clinic

AmerBdPsyNeurol:Psychiatry; AmerBdPsyNeurol:

Child Psychiatry; AmerBdEnvironMed

Private Practice of Psychiatry and Environmental
Medicine

Disclosure Statement:

CASE PRESENTATIONS

Richard G Jaeckle, MD

Dallas, Texas

Case #1

Dementia Praecox was a term first used by Morel in 1857 and later popularized by Kraepelin to describe mental deterioration early in life. Later Bleuler coined the term Schizophrenia and elucidated the classical description of its onset in a teenage girl. The case of an 18y/o girl is presented in which the onset and severity of the Schizophrenic psychosis was parallel to Group A Streptococcus (GAS) toxin. This enlarges considerably the syndrome of PANDAS (Pediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcus), first used in 1994 with sudden onset Obsessive-Compulsive Disorder (OCD) in children following a GAS infection.

Case #2

An environmental physician considered himself in good health having thoroughly chelated himself to reduce his body burden of toxic metals. While participating in an AAEM food course, he received an intradermal skin test of the glycerin control (0.05cc of 10% glycerin in saline). A severe reaction followed characterized by jack-hammer like palpitations and tachycardia of 120/min in a severe panic reaction which put him prostrate on the floor for about 40 minutes. He repeatedly asked for epinephrine, but permitted us to use neutralization until the episode was terminated with the #6 dilution. He was left exhausted by the experience and rested another day until the course ended. He then followed this presenter to Dallas where he was evaluated and treated at the EHCD during the next two weeks. During P/N testing, the episode was repeated in less intense fashion with a petroleum product. Risperdal was very useful in stabilization of his anxiety disorder. He improved considerably, but upon return to his home state and entry of his residence, he was again prostrate within an hour. He was able to get to his office where he recovered slowly. He has returned to normal with the daily use of oxygen therapy, replacement of his home's petroleum based heating unit and supplements.

22ND ANNUAL INTERNATIONAL SYMPOSIUM
ON MAN & HIS ENVIRONMENT

SCHEDULE

Saturday, June 26, 2004

- 8:25 **ANNOUNCEMENTS/MODERATOR: Kaye H. Kilburn, M.D.**
- 8:30 **Bruce M. Small, P.Eng.**, Envirosesic Certification Program, Small & Rubin Ltd., Georgetown, Ontario, Canada, *Title: "Building Safe Homes for the Environmentally Sensitive"*
- 8:50 Q & A
- 9:00 **Carmelo Rizzo, M.D.**, Roma, Italy, *Title: "Leukocytes Morphologic Modifications in Food Intolerances"*
- 9:20 Q & A
- 9:30 **Bertie Griffiths, Ph.D.**, Director of Environmental Health Center – Dallas Laboratory, Dallas, TX, *Title: "Effect of Gamma Gloublin in Chronically Sensitized Patients"*
- 9:50 Q & A
- 10:00 BREAK WITH EXHIBITORS**
- 10:30 **Donald P. Dennis, M.D.**, Private Practice, Atlanta Center for ENT & Facial Plastic Surgery Atlanta, GA, *Title: "Mechanism of Immune Inflammation in Sinusitis & Systemic Fungal Symptoms"*
- 11:00 Q & A
- 11:10 **William J. Meggs, M.D., Ph.D.**, Professor of Toxicology, Brody School of Medicine, East Carolina University, Greenville, NC, *Title: "Inflammation & Aging: Delaying the Inevitable"*
- 11:30 Q&A
- 11:40 **William J. Rea, M.D.**, Director, Environmental Health Center - Dallas, Dallas, TX, *Title: "The Treatment of Chronic Inflammation"*
- 12:00n Q & A
- 12:10n BUFFET LUNCH WITH EXHIBITORS**
- MODERATOR: Bertie Griffiths, Ph.D.**
- 1:30 **Kou Sakabe, M.D., Ph.D.**, Environmental Medical Center, Setagaya, Tokyo, Japan, *Title: "A Low Dose Effect of Environmental Endocrine Disruptors on Immune System"*
- 1:50 Q & A
- 2:00 **Alexander Sivakou, M.D.**, Acupuncture Department, Minsk, Belarus, *Title: "Combination of Magnetoacupuncture and Extracorporeal Autohemomagneto therapy in Clinical Practice"*
- 2:20 Q & A
- 2:30 **Donald P. Dennis, M.D.**, Private Practice, Atlanta Center for ENT & Facial Plastic Surgery Atlanta, GA, *Title: "Treatment of Sinusitis and Systemic Fungal Symptoms"*
- 2:50 Q & A
- 3:00 BREAK WITH EXHIBITORS**
- 3:30 **Theodore R. Simon, M.D.**, Private Practice - Nuclear Medicine, Functional Imaging of Texas, PA, Dallas, TX, *Title: "Inflammation from a Nuclear Medicine Perspective"*
- 3:50 Q & A
- 4:00 **Javier Santos, M.D.**, Hospital General Valle Hebron, Barcelona, Barcelona, Spain, *Title: "Neuro-Immune Control of Gut Inflammation: The Mast Cell"*
- 4:20 Q & A
- 4:30 CASE STUDIES **Adrienne Buffaloe, M.D.**, Research/Private Practice, New York, NY, *Title: "Immune Modulation Using Hyper Immune Egg"*: **Kalpana Patel, M.D.**, Director of Environmental Health Center Buffalo, Northwest Center for Allergy and Environmental Medicine, Buffalo, NY, *Title: "Inflammation as it Relates to Postpartum Hemorrhage"*
- 6:00 AJOURN**

SATURDAY, JUNE 26, 2004

ABSTRACTS

AND

HANDOUT

Abstract Information & Notes

Bruce M. Small, P.Eng.

Date of talk: Saturday, June 26, 2004, 8:30am

Small & Rubin Ltd.
100 Rexway Dr.
Georgetown, Ontario L7G 1R5
Canada

Phone: 905/702-8615
Fax: 905/873-6260
E-mail: brucesmall@envirodesic.com

Medical School/University Attended:

Bachelor of Applied Science degree in Engineering Science from the University of Toronto (1970) and a Master of Science degree in design from the University of Southern Illinois (1975)

Current Job Description:

Director of the *Envirodesic*TM Certification Program, a private sector initiative for developing and promoting buildings, products and services that contributes to healthier indoor environments. Executive Director of *Technology and Health Foundation*, a non-profit research corporation.

Other Information (including titles of books or articles you have recently written):

Has produced general and specialized research studies, for environmentally hypersensitive individuals, is a consultant, and author of two environmental health books, *Sunnyhill – The Health Story of the Eighties* and *The Susceptibility Report*, including an autobiography about his personal experience with the effects of pollution on the health of his own family.

Disclosure Statement:

Icynene Inc. – The Insulation System®
Cogent Environmental Inc. – Ecogent® General Purpose Cleaner

SPEECH TITLE: “Building Safe Homes for the Environmentally Sensitive”

The speaker has provided the information below.

1.) Goals and objectives: The purpose of this talk is to give the participants in the symposium some insight as to the design, construction, commissioning and operating principles that are required to produce an “ecology home” suitable for environmentally sensitive individuals.

2.) Conclusion of what is to be learned: Over a dozen major design factors are reviewed by the author to illustrate the kind of considerations that must be taken into account to create an ecologically acceptable environment for someone who is hypersensitive. As with all quality construction, care must be taken to create a building that will not go moldy over time. Careful material selection is important, but other principles such as simplicity, separation of areas, adequate room size and ventilated storage areas are also important. Protection of materials against moisture on the construction site is also critical. Once operational, an ecology home, like any other, requires preventive maintenance to avoid future environmental problems.

3.) References: The author draws upon 27 years of personal experience in home design for the environmentally sensitive and from his recent lecture presentations to architects on the design of mold-free buildings.

Blood sample of a patient Intolerant to β -lactoglobulin

Blood sample of a patient intolerant to milk casein

Preliminary results are interesting.

As showed in the graphs, we notice in all the samples:

- The distribution curve of white blood cells moves to the left;
- We have the complete modification of the curve, which is replaced by one or more parallelograms.

Moreover, analysing the dispersion diagram, we can notice a drastic decrease of the granularity on the graph.

DISCUSSION

Even if we studied a small number of cases, results of the Cytotoxic test (manually executed) match exactly to the analysis performed with the coulter counter. Leukocytes' modification is clearly pointed out either in the sample of leukocytes suspension or in the dilution sample: 1/10, 1/15 and 1/20; nevertheless we need further research in order to realize the complete standardization.

Abstract Information & Notes

Carmelo Rizzo, M.D.

Date of talk: Saturday, June 26, 2004, 9:00am

Natural S.r.l. - Viale Ippocrate 93
Roma, 00161
Italy

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Medical School/University Attended:
Internship:
Current Faculty Appointments:
Current Job Description:
Other Information (including titles of books or articles you have recently written):

University of Rome
Rome Policlin
University of Urbino
Clinical Ecologist – Nutrition and Allergies
Translations and publication of R. Mackarness “Not all in the Mind” and “D. Rapp “The Impossible Child” in Italian. Clinical ecology and food intolerance ed. By Tecnico Nuove. Founder and President of the Italian Associates of Clinical Ecology (A.M.I.E.C.). International Lecturer. Author of many articles on Food Intolerance.

Disclosure Statement:

None

SPEECH TITLE: “Study of leukocytes modifications comparing the Cytotoxic method with the analytic approach”

The speaker has provided the information below.

- 1.) Goals and objectives:** Comparing the Leukocytes Morphologic Modifications in the Cytotoxic Diagnosis of food intolerance with Leukocyte changes in classical analytic method.
- 2.) Conclusion of what is to be learned:** The clinical trials support our method, but it needs further research.
- 3.) References:** Case reports of the Department of Clinical Pathology of University of Catania (Italy)

Abstract Information & Notes

Bertie Griffiths, Ph.D.

Date of talk: Saturday, June 26, 2004, 9:30am

Environmental Health Center - Dallas
8345 Walnut Hill Lane, Ste. 220
Dallas, TX 75231

Phone: 214/368-4132
Fax: 214/691-8432
E-mail:

Medical School/University Attended:

M.S., B.S., University of Wisconsin, Bacteriology;
Ph.D. University of West Indies, Virology

Post Doctoral:

University of West Indies

Current Job Description:

Director, Environmental Health Center – Dallas
Analytical Lab.

Disclosure Statement:

SPEECH TITLE: “Effect of Gamma Globulin in Chronically Sensitized Patients”

The speaker has provided the information below.

1.) Goals and objectives:

2.) Conclusion of what is to be learned:

3.) References:

Abstract Information & Notes

Donald P. Dennis, M.D.

Date of talk: Saturday, June 26, 2004,
10:30am

ENT & Facial Plastic Surgery, LLC
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Atlanta, GA 30327

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Medical School/University Attended:

Medical College of Georgia

Internship:

Emory University – Medicine

Residency:

ENT & Facial Plastic Surgery Johns Hopkins
Hospital

Board Certifications:

Otolaryngology and Head and Neck Surgery

Current Job Description:

Private Practice Atlanta Center for ENT &
Facial Plastic Surgery

Other Information (including titles of books or
articles you have recently written):

Chronic Sinusitis: Defective T-cells
responding to super antigens treated by
reduction of Fungi in Nose and Air. Accepted
for publication in Archives of Environmental
Health.

Disclosure Statement:

None, Antisiotic and Antifungal Nasal Sprays

SPEECH TITLE: “Mechanism of Immune Inflammation in Sinusitis & Systemic Fungal Symptoms”

The speaker has provided the information below.

1.) Goals and objectives: To purpose the mechanism for Inflammation in sinusitis and fungal systemic symptoms based on current literature.

2.) Conclusion of what is to be learned: Fungi cause inflammation in the sinus and systemically, when the fungal antigen is removed from the patient and air, symptoms resolve.

3.) References: Attached

Title:

Inflammation of Chronic Sinusitis and Systemic Fungal Symptoms: A T-Cell Defect Treated by Nose, Systemic, and Environmental Air Fungal Antigen Reduction

Introduction

In 1999 the Mayo clinic study found 93% of chronic sinusitis patient met the diagnostic criteria of allergic fungal sinusitis (AFS). In 2001, Schubert proposed the hypothesis that most hypertrophic sinusitis may be due to a T-cell receptor defect that allows Superantigens to up regulate T-cells to cause release of cytokines to induce tissue inflammation. **The purpose of the study was to determine if removal of the fungal antigen from the nose, body, and air would reverse the nasal mucosal disease and systemic symptoms associated with fungal exposure.**

Methods

720 patients with AFS were studied. 89 had systemic symptoms of fungus infections. Nasal cultures were placed on SDA agar and compared to environmental 1 hr. gravity plate exposure fungal cultures to determine environmental source. Photographs and sinus mucosa and CT scans were done before and after environmental remediation and patient treatment with antibiotic and antifungal nasal sprays, and saline nasal irrigations. Patients with systemic symptoms completed a symptom questionnaire before and after environmental and systemic treatment with oral antifungals. The environmental treatment consisted of HEPA air filtration, evaporations and/or fogging with a botanical mixture of grapefruit seed extract (GSE), or non ozone producing ionizers.

Results

720 patients were studied. 411 of 720 (57%), reduced their environmental air fungal load to less than 4 colonies on an SDA agar plate per 1 hr. exposure. 45 of the 411 had systemic symptoms. 374 of 411 (91%) showed normal sinus mucosa endoscopically. 365 of 411 (89%) showed marked improvement of sinus CT scans. Of the 46 whose CT scans that did not markedly improve 3 had lymphoma, 2 had IgG subclass deficiencies, 22 had continued positive nasal fungal cultures, and 19 were lost to follow up. Of the 45 who had systemic symptoms and reduced their air fungal counts to below 4 colonies per 1 hr plate exposure, 39 (87%) experienced systemic fungal symptom relief.

Conclusion

Chronic sinusitis and systemic fungal symptoms are likely caused by an immune response to fungal antigen. When the antigen is removed from the patient and the environmental air, the immune reaction stops and the inflammation in the tissues can

Resolve. Exceptions to this are due to underlying disease processes and/or failure to remove the antigen from the patient or environment, or failure to find the source of exposure.

References:

1. Kaliner MA, Osguthorpe JD, et al. Sinusitis: bench to bedside. Current findings, future directions. J allergy Clin Immunol 1997;99:S829-S848.
2. Ponikau JU, Sherris DA, Kern EB, et al. The diagnosis and incidence of allergic fungal sinusitis. Mayo Clin Proc 1999; 74:877-84.
3. Braun H, Buxina W, Freudenschuss, K, et al. Eosinophilic fungal rhinosinusitis: A common disorder in Europe? Laryngoscope 2003;113:264-269.
4. Ponikau JU. Chronic Rhinosinusitis: The War of the Immune System Against Fungi. American Rhinologic Society 2001;Jan.
5. Waxman JE, Sale SR, Spector JG, et al. Allergic *Aspergillus* sinusitis: concepts in diagnosis and treatment of a new clinical entity. Laryngoscope 1987; 97:261-266.
6. Hamilos DL. Chronic sinusitis. J Allergy Clin Immunol 2000; 106:213-227.
7. Oswald, MR, Basic Immunology. Kansas City, Kansas, University of Kansas Medical Center. 2000; 4:72.
8. Schubert, MS. A superantigen hypothesis for the pathogenesis of chronic hypertrophic rhinosinusitis, allergic fungal sinusitis, and related disorders. Ann Allergy, Asthma, Immunol.2001; 4:181-187
9. Kotzin BL, Leung DY, Klapper J, et al. Superantigens and their potential role in human disease. Adv Immunol 1993; 54:99-166
10. Klapper J, Kotzin B, Herron L, et al. V β -specific stimulation of human T cells by staphylococcal toxins. Science 1989;244:811-813.
11. Bernstein JM, Ballow M, Schlievert PM, et al. A superantigen hypothesis for the Pathogenesis of chronic hyperplastic sinusitis with massive nasal polyposis. Am J Rhinol 2004 ; 17:321-326

Abstract Information & Notes

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

Brody School of Medicine
East Carolina University
600 Moye Blvd., RM 4W54
Greenville, NC 27858

Date of talk: Saturday, June 26, 2004, 11:10am

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Medical School/University Attended:

Internship:

Residency:

Fellowships:

Board Certifications:

Current Faculty Appointments:

Current Job Description:

Other Information (including titles of books or articles you have recently written):

University of Miami

University of Rochester

University of Rochester

National Institutes of Health, New York University

Internal Medicine, Emergency Medicine, Allergy,
Medical Toxicology

Professor, Brody School of Medicine

Medical School Professor

Author of "The Inflammation Cure", published by McGraw-Hill, Sept. 2003. Co-Author of "Biomarkers of Immunotoxicology", published by National Academy Press; The Physics of Biology Modern Physics Letters 2003.

Disclosure Statement:

None

SPEECH TITLE: "Inflammation & Aging: Delaying the Inevitable"

The speaker has provided the information below.

1.) Goals and objectives: To know the role of inflammation in the aging process. To know lifestyle and environmental modifications that can moderate aging.

2.) Conclusion of what is to be learned: Inflammation plays a role in the aging process. This knowledge can be used to modulate the rate of aging.

3.) References: William Joel Meggs, MD, with Carol Svec. The Inflammation Cure. McGraw-Hill, New York, 2003.

Inflammation & Aging

Presenter: William Joel Meggs, MD, PhD
Brody School of Medicine
East Carolina University
Greenville, NC, USA

Diseases associated with aging have been linked to inflammation. As people age, pro-inflammatory cytokines such as IL-6 rise in their blood while anti-inflammatory cytokines fall. The stages of life, from birth and development, maturity and reproduction, and decline into senility may be modulated by inflammation. The inflamm-aging doctrine holds that inflammation is a mechanism for self-destruction of the organism. Age is a major risk factor for atherosclerosis and can be a determinant of disease in the absence of any other risk factor. Inflammation plays a role in osteoarthritis, with the destruction of the musculoskeletal system with aging. Alzheimer's disease is now known to be mediated by inflammation. Recent research shows that use of non-steroidal anti-inflammatory medications may reduce the risk of Alzheimer's disease. There are a number of interventions that can be used to delay the inexorable processes of aging, while other interventions accelerate the aging process. From the viewpoint of evolutionary biology, the role of inflammation in aging may be a result of biological economy.

Abstract Information & Notes

William J. Rea, M.D.

Environmental Health Center - Dallas
8345 Walnut Hill Lane, Suite 220
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Date of talk: Saturday, June 26, 2004, 11:40am

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Medical School/University Attended:

Ohio State University College of Medicine, Columbus, OH

Internship:

Parkland Memorial Hospital, Dallas, TX

Residency:

University of Texas Southwestern Medical School, Dallas, TX

Board Certifications:

American Board of Surgery, American Board of Thoracic Surgery, American Board of Environmental Medicine, American Board Certification, Disability Analyst

Current Faculty Appointments:

Capital University of Integrative Medicine, Washington, D.C.

Current Job Description:

President/M.D. – Environmental Health Center-Dallas

Other Information (including titles of books or articles you have recently written):

Optimum Environments for Optimum Health and Creativity (book); Toxic Exposure to Molds and Mycotoxins

Disclosure Statement:

None

SPEECH TITLE: “The Treatment of Chronic Inflammation”

The speaker has provided the information below.

1.) Goals and objectives: To familiarize the clinician with comprehensive treatment modalities which will improve the quality of life for patients with chronic inflammatory diseases.

2.) Conclusion of what is to be learned: There are many treatment modalities which can help the patient with chronic inflammatory disease to have a better quality of life.

3.) References: Heine, H and Pischinger A: Matrix and Matrix Regulation: Basis of a Holistic Theory in Medicine. 1991. Haug International, Brussels.

THE TREATMENT OF CHRONIC INFLAMMATION

**William J. Rea, M.D., F.A.C.S., F.A.A.E.M.
Environmental Health Center – Dallas
8345 Walnut Hill Lane, Ste. 220
Dallas, Texas 75231**

There are many treatments of inflammation, mostly by medication. These are either partially successful, totally unsuccessful or have so many complications that they are hazardous. We have taken a different pathway to eliminate inflammation. These include the avoidance and elimination of the incitants (both primary and secondary), strengthening the detoxification system with nutrition, and mobilization of toxics that are sequestered in the body.

Elaborate techniques have been developed for avoidance of the entering generators of inflammation. These include finding fresh, less polluted air, both inside and out, eating less polluted food, drinking less polluted water, or surgically eliminating areas of inflammation such as implants from the body.

Conclusions:

Treatment of the hypersensitive aspects of the generators of inflammation is essential in most cases. Treatment of nutritional aspects of detoxification is essential. Also, mobilization of toxics by medically supervised sauna, exercise, and physical therapy is essential as is medically supervised oxygen therapy. Immune modulators are frequently necessary. Overall, much inflammation can be reduced objectively by these measures, thus preventing disabling disease.

Goals and Objectives:

1. To define the areas of treatment for inflammation.
2. To make each area of treatment easy for the patient to understand.
3. To make each area of treatment available for the physician in his practice.

References:

Rea, William J: Chemical Sensitivity, Lewis Publishers, Boca Raton, Florida, 1992.

Rea, William J.: Chemical Sensitivity, Volume 2, Lewis Publishers, Boca Raton, Florida, 1994.

Rea, William J.: Chemical Sensitivity, Volume 3, Lewis Publishers, Boca Raton, Florida, 1996.

Rea, William J.: Chemical Sensitivity, Volume 4, Lewis Publishers, Boca Raton, Florida, 1997.

Abstract Information & Notes

Kou Sakabe, M.D., Ph.D.

Environmental Medical Center
The Kitasato Institute, Kitasato University
4-3-18 Seijo
Setagaya, Tokyo 157-0066
Japan

Date of talk: Saturday, June 26, 2004, 1:30pm

Phone: 011/81-3-5490-2366
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Medical School/University Attended:

Internship:

Residency:

Board Certifications:

Tokai University School of Medicine

Tokai University Hospital

Tokai University Hospital

Japanese Society of Industrial and Occupational
Medicine

Japanese Association of Physical Medicine,
Balneology and Climatology

Professor of Clinical Ecology

Current Faculty Appointments:

Current Job Description:

Clinical Ecologist, Environmental Toxicologist

Disclosure Statement:

None

SPEECH TITLE: "A Low Dose Effect of Environmental Endocrine Disruptors on Immune System"

The speaker has provided the information below.

- 1) **Goals and objectives:** Elucidation of the immune disrupting mechanism of environmental endocrine disruptors for health risk assessment.
- 2) **Outline of talk/abstract:** a) an electron microscopic observation indicated Bis-A, DBP or DEHP treatment to bring about apoptosis of thymocytes (thymic T-lymphocytes) which were embraced by stromal cells (i.e. macrophages); b) flow cytometric analysis demonstrated Bis-A, DBP or DEHP to induce the change of lymphocyte subsets: an increase in the helper/inducer (CD4⁺CD8⁻) cells with decrease in the double positive (CD4⁺CD8⁺) cells in the thymus.
- 3) **Conclusion of what is to be learned:** It follows from the above findings that Bis-A, DBP or DEHP may cause morphologic changes in the murine primary immune organ closely related to T-lymphocyte growth, differentiation and function. In addition, these changes appear to derive mainly from Bis-A, DBP- or DEHP-induced tissue-specific gene expression. The results of the present study suggest that we must recognize the possibility that a low-dose Bis-A, DBP or DEHP can affect the various immune responses in the capacity of immune disruptors.
- 4) **References:** 1) Int. J. Immunopharmacol. 20(4-5), 205-212, 1998.
2) Int. J. Immunopharmacol. 21(12), 861-868, 1999.b
3) Pathophysiol. 6(1), 231-236, 2000.
4) Environmental Endocrine Disruptors, edited by L.J. Guillette and D.A. Crain, Taylor & Francis, 1999.
5) Proceeding, International Symposium on Environmental Endocrine Disruptors 2003.

A Low Dose Effect of Environmental Endocrine Disruptors on Immune System

Kou Sakabe, M.D., Ph.D.

Division of Clinical Ecology, Environmental Medical Center-Tokyo, The Kitasato Institute,

Professor, Graduate School of Medical Sciences, Kitasato University

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Natural sex steroids such as 17β -estradiol or 5α -dihydrotestosterone, are well known to modulate immune responses in various animals including humans.

Although this family of natural hormones are steroidal in structure, a variety of exogenous non-steroids has been found to act like estrogens or androgens. In fact, environmental estrogens or androgens, which are one of the endocrine disruptors, have been implicated in a number of human health disorders. These substances are derived from a number of relatively common and abundant sources such as plastics, pesticides and agricultural products. However, little is known about the pharmacological and/or toxicological effects on immunocompetent cells of exposure to these substances especially in reference to the cause of animal and human immune disorders. To address this issue, the present *in vitro* study focuses on the effects of natural and environmental chemicals (ECs) such as bisphenols and phthalic esters on murine thymus.

The effect of ECs on the thymus from castrated female rats (BDII/Han) was examined by molecular biologic, microscopic and flow cytometric techniques. Our findings were as follows : (a) an electron microscopic observation indicated Bis-A, DBP or DEHP treatment to bring about apoptosis of thymocytes (thymic T-lymphocytes) which were embraced by stromal cells (i.e. macrophages) ; (b) flow cytometric analysis demonstrated Bis-A, DBP or DEHP to induce the change of lymphocyte subsets: an increase in the helper/inducer ($CD4^+CD8^-$) cells with decrease in the double positive ($CD4^+CD8^+$) cells in the thymus. It follows from the above findings that Bis-A, DBP or DEHP may cause morphologic changes in the murine primary immune organ closely related to T-lymphocyte growth, differentiation and function. In addition, these changes appear to derive mainly from Bis-A, DBP- or DEHP-induced tissue-specific gene expression. The results of the present study suggest that we must recognize the possibility that a low-dose Bis-A, DBP or DEHP can affect the various immune responses in the capacity of immune disruptors.

Abstract Information & Notes

Alexander Sivakou, M.D.

Date of talk: Saturday, June 26, 2004, 2:00pm

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Belarus, Russia

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Medical School/University Attended:
Internship:
Board Certifications:

Grodno Medical University Republic of Belarus
Neurology, Psychiatry, Acupuncture, Physiotherapy
ICMART (International Council of Medical
Acupuncture and Related Technics), International
Board Member; Honorable Member of the Chinese
Needling and Moxibustion Manipulation society.
Belarusian Medical Academy for Postgraduating
Doctors.

Current Faculty Appointments:

Acupuncture Department
Patent 4088 A61H 39/00, A61N 7/00. Method of
Focus Ultrasound Therapy.// Official bul. Rep. Of
Belarus. -2001.-~~1~~3.-P.23.
Patent 1046 A61N 1/32, 1/18. Method of Pain
Syndrome Treatment

Current Job Description:
Other Information (including titles of books or articles
you have recently written):

Disclosure Statement:

None

SPEECH TITLE: "Combination of Magnetoacupuncture and Extracorporeal Autohemomagnetotherapy in Clinical Practice"

The speaker has provided the information below.

1.) Goals and objectives: The main of this investigation was to substantiate the advisability of arranges and combines affection in clinical practice and sport medicine by methods of autohemomagnetotherapy (AHMT) and magnetopuncture (MP).

2.) Conclusion of what is to be learned: During general assessment of the treatment results a more expressed positive dynamics was observed in the group with AHMT and MP - 78%. In the AHMT group the therapeutic effect was 50%. The results obtained prove the prospects and efficiency of combination AHMT and MP in medical practice and provide grounds for development of new methods of treatment

3.) References: None

COMBINATION OF MAGNETOACUPUNCTURE AND AUTOHEMOMAGNETOTHERAPY IN CLINICAL PRACTICE

A. Sivakou, V. Ostapenko,*A. Razumov, S. Pletnev
Belarus, * Russia

Magnetotherapy today is one of the most prospective therapeutic directions, which is contained in application of different kinds of magnetic field with medicinal and prophylactic purpose. The effectiveness of application of magnetotherapy in clinical practice indicates expediency of searching the new methods and ways of magnetotherapy. The question of studying the interaction of magnet field with biological objects is very important. There is enough number of structures on submolecular, molecular and subcellular level in the organism. The changes of them can easily become transformed into the cellular, tissue and system reaction order, which are defining physiological and medicinal effect of magnetotherapy. Among medicinal effects of magnetotherapy the most proved are immunomodulated, anti-inflammatory, antiedema, sedative, trifiphicital - regenerative, anesthetic, metabolic etc. Sensitivity of the organism the action of magnetic field defines the degree of expressness of the effects which were mentioned earlier. According to the literary data the threshold of sensitivity to impulsive magnetic field is 0.1 militesla (mTl), to variable - 3 mTl, to constant - 8 mTl.

Among the general peculiarities of the action of magnetic field to the organism are: 1) reciprocal reaction of the organism to the influence of magnetic field is characterized by certain phases, that corroborates the expediency if the course treatment; 2) a reaction of the organism to a single procedure of the influence by a magnetic field stays the same for 2 - 6 days and other a series of procedures - for 35 - 40 days; 3) the influence of a magnetic field depends on the initial condition of the organism; 4) reactions of the organism are not only liminal, but also resonant. Magnetotherapy is recommended for the treatment of various diseases, while the contraindications are limited. The contraindications are individual intolerance, system blood and oncological diseases, hemorrhage, hypotension, thyrotoxicosis. According to the given classification different types of the magnetic fields are used. The intensity of the influence of a magnetic field is greatly varied: from weak (microtesla intensity - this intensity can be compared to the influence of the Earth's magnetic field) to strong (the units of Tesla, such intensity may course muscular contraction), thus, the intensity of the influence in therapy can vary to million times. The technique and methods of the procedure have changed for the last years. Internal organs can be influenced (either rectally or vaginally) as well as various skin zones and regions (the projections of the reflexiological zones, internal and endocrine organs). The influence of a magnetic field on the points of acupuncture has some prospects. The classification of the methods of physical factors influence to acupuncture points is presented on the slide. Magnitopuncture belongs to the electroacupuncture group of this classification. These points have anisotropy of magnet quality, which justifies the expediency of the influence of a magnetic field on the points of acupuncture. When influencing the points of acupuncture we can affect purposefully the functioning of different organs and systems connected with them. To use various methods of magnetotherapy we devised a multi - purpose apparatus, which allows to conduct general magnitotherapy, rectal and vaginal magnetotherapy, autohemomagnetotherapy (AHMT) and magnetopuncture (MP). The main of this investigation was to substantiate the advisability of arranging and combines affection in clinical practice and sport medicine by methods of AHMP and MP. AHMT methods consisted in affection by a disc-like inductor on the veins of bend of elbow or inguinal region during 10 min. Then affection was executed on acupuncture points by a special inductor. The choice of the points was carried out taking into consideration the pathologic process localization and the principles generally accepted in acupuncture. Local, zone, segmental and channel affected points received affection. Affection parameters of low frequency pulse magnetic field are the following: complexity form of impulse, the basic frequency - 40-160 Hz, modulate frequency - 10 Hz, quantity of magnet inductance - 100 mTl during AHMT influence and 70 mTl - during MP.

The clinical investigation stage was performed on patients with vertebral lumbosacral radiculitis with lumbar ischialgia, vegetovascular and reflex tonic syndrome. The magnetopuncture was made into local, zone, segmental and channel affected points. Most often the affection was made into the points V24, V25, V26, V27, V36, V57, V40, V60, E36, VB34 and etc. The total time of the procedure is 14 - 20 min. The exposure for the one point is increased from 2 min during the first procedure to 3 min by the third procedure. Prior to start of the treatment course the acupuncture points most sensible to manual palpation are determined and these points are treated first during initial 3 -5 procedures. Later, with decrease of the pain syndrome other acupuncture points selected are treated. Therapy was conducted in two groups of patients suffering from neurological manifestations of osteochondrosis in lumbosacral section of vertebral column. In the first (reference) group therapy was executed by means of autohemomagnetotherapy (AHMT) and the second group was treated by the AHMT and magnetopuncture (MP). The treatment results were estimated by positive dynamics of the clinical picture and decrease of the expression degree of objective disease features measured by means of electroencephalogram, reography and multi - criteria analysis. During general assessment of the treatment results a more expressed positive dynamics was observed in the second group - 78% and was characterized by decrease of pain syndrome, disappearance or decrease of tonic muscle tension, stress symptoms, normalization of parameters of electrophysiological investigation methods was also recorded. In the first, reference group the therapeutic effect

was 50%. The results obtained prove the prospects and efficiency of combination AHMT and MP in medical practice and provide grounds for development of new methods of treatment.

An investigation concerning the application advisability of magnetotherapy in sport medicine. The existing means and methods of recovery are not affective enough working capacity, preserving of health of sportsmen in top sport determines the necessity to search for fundamentally new methods, means and technologies.

The range of training activities in sport, physical overwork refer to the group of factors, bringing about the so-called secondary immunodeficiency. Methods of working capacity rehabilitation and increase, stimulating energy and plastic exchange in a sportsman's organism are considered to be the most perspective ones.

Usage of pharmacological drugs as the means of rehabilitation in sport is limited due to strict restrictions on the part of the medical commission International Olympic Committee and individual peculiarities of an organism. In given circumstances the most perspective and acceptable are non medicamental procedures and technologies of working capacity increase and immunodeficiency reactions. Magnetotherapy belongs to such methods. Examinations of the people suffering from cardiovascular system disorders that were made earlier revealed immunomodulating effect of the method of magnetotherapy that is being presented, which is the reason of its use in the sport medicine. In the group of sportsmen, subjected to the AHMT course (group 1), the working capacity index trustworthily raised for 20%, while in the control group (group 2) stayed without changes.

The results of studying the treatment process of the low frequent AHMT attest to the modulating effect of the course use of these procedures on the erytron (erythropoetic reaction), the blood fluidity, peripheral circulation of the blood and as a result, the improvement of the muscular oxygenation. Besides, the information about immunomodulating effect was given - the increase of the number common T - lymphocytes, T - active lymphocytes and also a drop of the number B - lymphocytes to the norm.

The influence by the method AHMT is carried out in projection of veins on the elbow or inguinal field, over which there is the inductor in the form of disk. During the procedure the patient can stay in the light clothes, with it all there is no need to provide the measures in the prophylaxis of AIDS.

Thus, we should note, that AMT is refers to the category of the universal physiological mechanism of the influence on the organism, has systematic homeostatic character and accompanied specific and non - specific, local and common changes from the side of the different organs and tissues. The program of combined and successive (every other day) influence on sportsman's organism with the help of low frequency AHMT and general AHMT under conditions of twenty one day educational - training session.

The results of research works and practical use of the method when preparing sportsmen, who got magnetotherapy attest to a vivid tendency towards planned rise of the hemoglobin level (from one session to another), while this level isn't stable in the controlled group. Low frequency HMT doesn't influence to a large extent the initial hemoglobin level, though it supports the attained level of hemoglobin in blood for a longer period of the time, not with standing great training work.

The given facts (equally with the rise of average erythrocyte volume the rise of average hemoglobin content in them, the reduction of their deformation index) indirectly attest to the rise of activity an synthesis of endogenic erythropoietin. A stable hemoglobin level allows to fulfill training work in a more efficient way.

The given methods is protected by the Euroasian patent No200100562 dated 04.05.2001. Conclusions. Non - invasion AHMT which is carried out with the use of our devised equipment and technologies raise efficiency of organism that is subjected to active physical work.

One of the mechanisms to raise work efficiency, after non - invasion ANMT course is carried out, is vivid immunomodulating effect, which increases the number of the T - lymphocytes, T - active lymphocytes and reduces the number of B - lymphocytes up to the standard.

These and other facts obtained earlier about increase of indices which characterize erytron, attest to hemopoetic organism function activation as a result of AHMT. It allows to make high hemoglobin level of blood more stable, not with standing physical work, without the use of medical preparations, forbidden by International Olympic Committee. Foregoing facts give us reason to recommend the method of autohemomagnetotherapy with the use of our devised equipment and technology in reconstructive and sporting medicine, including training of elite sportsmen.

Abstract Information & Notes

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Internship:
Residency:
Board Certifications:
Current Job Description:

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Other Information (including titles of books or articles
you have recently written):

Chronic Sinusitis: Defective T-cells responding to
super antigens treated by reduction of Fungi in Nose
and Air. Accepted for publication in Archives of
Environmental Health.

Disclosure Statement:

None, Antisiotic and Antifungal Nasal Sprays

SPEECH TITLE: “Treatment of Sinusitis and Systemic Fungal Symptoms”

The speaker has provided the information below.

- 1.) Goals and objectives:** To show that fungal antigens are responsible for most chronic sinusitis and a host of systemic symptoms
- 2.) Conclusion of what is to be learned:** If fungal antigens are removed from the patient and environmental air, the nasal mucosal returns to normal and systemic symptoms improve or resolve.
- 3.) References:** Attached to abstract

Title:**Inflammation of Chronic Sinusitis and Systemic Fungal Symptoms: A T-Cell Defect Treated by Nose, Systemic, and Environmental Air Fungal Antigen Reduction****Introduction**

In 1999 the Mayo clinic study found 93% of chronic sinusitis patient met the diagnostic criteria of allergic fungal sinusitis (AFS). In 2001, Schubert proposed the hypothesis that most hypertrophic sinusitis may be due to a T-cell receptor defect that allows Superantigens to up regulate T-cells to cause release of cytokines to induce tissue inflammation. **The purpose of the study was to determine if removal of the fungal antigen from the nose, body, and air would reverse the nasal mucosal disease and systemic symptoms associated with fungal exposure.**

Methods

720 patients with AFS were studied. 89 had systemic symptoms of fungus infections. Nasal cultures were placed on SDA agar and compared to environmental 1 hr. gravity plate exposure fungal cultures to determine environmental source. Photographs and sinus mucosa and CT scans were done before and after environmental remediation and patient treatment with antibiotic and antifungal nasal sprays, and saline nasal irrigations. Patients with systemic symptoms completed a symptom questionnaire before and after environmental and systemic treatment with oral antifungals. The environmental treatment consisted of HEPA air filtration, evaporations and/or fogging with a botanical mixture of grapefruit seed extract (GSE), or non ozone producing ionizers.

Results

720 patients were studied. 411 of 720 (57%), reduced their environmental air fungal load to less than 4 colonies on an SDA agar plate per 1 hr. exposure. 45 of the 411 had systemic symptoms. 374 of 411 (91%) showed normal sinus mucosa endoscopically. 365 of 411 (89%) showed marked improvement of sinus CT scans. Of the 46 whose CT scans that did not markedly improve 3 had lymphoma, 2 had IgG subclass deficiencies, 22 had continued positive nasal fungal cultures, and 19 were lost to follow up. Of the 45 who had systemic symptoms and reduced their air fungal counts to below 4 colonies per 1 hr plate exposure, 39 (87%) experienced systemic fungal symptom relief.

Conclusion

Chronic sinusitis and systemic fungal symptoms are likely caused by an immune response to fungal antigen. When the antigen is removed from the patient and the environmental air, the immune reaction stops and the inflammation in the tissues can

Resolve. Exceptions to this are due to underlying disease processes and/or failure to remove the antigen from the patient or environment, or failure to find the source of exposure.

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13. Ponikau JU, Sherris DA, Kern EB, et al. The diagnosis and incidence of allergic fungal sinusitis. Mayo Clin Proc 1999; 74:877-84.
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16. Waxman JE, Sale SR, Spector JG, et al. Allergic *Aspergillus* sinusitis: concepts in diagnosis and treatment of a new clinical entity. Laryngoscope 1987; 97:261-266.
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19. Schubert, MS. A superantigen hypothesis for the pathogenesis of chronic hypertrophic rhinosinusitis, allergic fungal sinusitis, and related disorders. Ann Allergy, Asthma, Immunol.2001; 4:181-187
20. Kotzin BL, Leung DY, Klapper J, et al. Superantigens and their potential role in human disease. Adv Immunol 1993; 54:99-166
21. Klapper J, Kotzin B, Herron L, et al. V β -specific stimulation of human T cells by staphylococcal toxins. Science 1989;244:811-813.
22. Bernstein JM, Ballow M, Schlievert PM, et al. A superantigen hypothesis for the Pathogenesis of chronic hyperplastic sinusitis with massive nasal polyposis. Am J Rhinol 2004 ; 17:321-326

Abstract Information & Notes

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Current Faculty Appointments:

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See CV at www.geocities.com/theodorersimon

Disclosure Statement:

Beebee Foundation

SPEECH TITLE: “Comparison of Neuropsychological Testing with Brain SPECT”

The speaker has provided the information below.

1.) Goals and objectives: The audience should understand the implications of scintigraphic and neuropsychological testing and how these results are complementary in the Environmental Disease arena.

2.) Conclusion of what is to be learned: Both types of testing have strengths and weakness but – together – they are an efficient aid in diagnosing and treating patients.

3.) References:

Inflammation from a Nuclear Medicine Perspective

by Theodore R. Simon, M.D.

Abstract

Goals and Objectives

Nuclear Medicine is a functional modality capable of tracing several aspects of inflammation including increased local metabolic activity, sequestration of iron, increased blood flow, increased capillary permeability, and increased granulocyte traffic. These aspects of inflammation will be illustrated in case studies and discussed in terms of diagnostic and surveillance techniques for the practicing physician.

Conclusions

Nuclear Medicine offers several windows of physiological function through which to identify and quantify inflammation. The window chosen is critical to obtaining the information best suited to the needs of individual patients.

References

These techniques are well established. They are well described in any standard nuclear medicine textbook.

Abstract Information & Notes

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Not applicable

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Board Certifications:

Current Job Description:

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Carlos III, entitled: "Effect of stress on intestinal
function in patients with irritable bowel syndrome and
development of a new experimental animal model of
irritable bowel syndrome", to develop at Digestive
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Other Information (including titles of books or articles
you have recently written):

Disclosure Statement:

None

SPEECH TITLE: "Neuro-Immune Control of Gut Inflammation: The Mast Cell"

The speaker has provided the information below.

1.) Goals and objectives: To learn how crosstalk between resident immune cells in gut mucosa, the mast cell, and nerves is relevant to maintain local microenvironmental homeostasis in the intestine and colon.

2.) Conclusion of what is to be learned: Local neuro-immune regulatory networks, exemplified by neural-mast cell interactions, are key for the maintenance of normal tissue (gut) physiology. Endogenous as well as exogenous events continuously challenge this physiological balance. Loss of this control may precipitate and predispose individuals for chronic inflammatory disorders.

3.) References: 1) Santos J, Saperas E, Mourelle M, et al. *Gastroenterology* 1996; 111:1465-1473.
2) Santos J, Saperas E, Nogueiras C, et al. *Gastroenterology* 1998; 114:640-648.
3) Santos J, Benjamín M, Yang P-C, et al. *Am J Physiol Gastrointest Liver Physiol* 2000; 278:G847-854.
4) Santos J, Yang P-C, Söderholm JD, et al. *Gut* 2001; 48 630-636.
5) Guilarte M, Santos J, Alonso C. *Coricotropin-Releasing Hormone (CRH) Triggers Jejunal Mast Cell and Eosinophil Activation in IBS patients: Selected for oral presentation at Digestive Disease Week, New Orleans, Louisiana, May 15-20, 2004.*

Neuro-Immune Control of Gut Inflammation: The Mast Cell.

Javier Santos, M.D., Ph.D.

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The intestinal epithelium plays an active role in local and systemic immunological and inflammatory events, acting as an antigen presenting system (expressing major histocompatibility class II antigen), influencing the transport of macromolecules, and expressing adhesion molecules, releasing cytokines, chemokines and other regulatory molecules that participate in the trafficking and homing of immune cells in the gut (1,2).

Mast cells are widely distributed in all tissue layers throughout the entire gastrointestinal tract. The intestinal lamina propria contains approximately 20000 mast cells/mm³. The number of mast cells increases in parasitic infections as well as in intestinal disorders such as inflammatory bowel disease, irritable bowel syndrome or food allergy.

Neuroanatomical studies have provided strong evidence for the direct innervation of mast cells. In fact in many somatic and visceral tissues including most organized and non-organized lymphoid compartments like the gastrointestinal mucosa, postganglionic sympathetic as well as peptidergic and vagal afferents are closely associated with mast cells in several species. In the human gastrointestinal mucosa 47 to 78% of mast cells were adjacent to nerve fibers. It seems that those associations increase in number and may be synaptic-like (20-200 nm) in cases where mast cell hyperplasia and tissue inflammation are present whereas non synaptic contacts predominate in normal tissue conditions (3).

The development of *animal models of hypersensitivity* has greatly contributed to the progress in the knowledge of barrier and immune functions of the intestinal epithelia as well as to the neuro-immune regulation of intestinal epithelial transport. These models include mainly enteric parasitic infestation with the nematodes *Nippostrongylus brasiliensis* or *Trichinella spiralis* in rodents or guinea-pigs, active sensitization of rats and mice with egg ovalbumin (OVA) or horseradish peroxidase (HRP), and guinea-pigs with lactoglobulin, and passive sensitization by transferring serum from sensitized animals or injecting IgE antibodies. Findings implicating mast cells as relevant players in neuro-immune regulation of intestinal inflammation will be summarized.

Typically, intestinal allergic reactions to food antigens are characterized by increased water and ion secretion and enhanced permeability. Mast cells contain a vast array of molecules, such as histamine, prostaglandins, nitric oxide, or cytokines that can alter epithelial function. Participation of mast cells in these reactions has been suggested by the lack of secretory response in sensitized jejunal tissues mounted in Ussing chambers after pretreatment with the mast cell stabilizer doxantrazole (4). Studies with parasite models in rats have shown that when active worm expulsion began, increased net secretion of Cl⁻ and Na⁺ was present (5). These events were paralleled by decreased numbers of stained mast cells in the mucosa (4-5) and high serum levels of the specific protease, RMCP II, supporting ongoing mucosal mast cell degranulation. Similar increases in secretion and RMCP II as well as histological abnormalities were demonstrated when intestinal tissue was challenged with worm antigen on day 35 postinfection, when mast cell hyperplasia had developed, further supporting the view that mast cells are critical in ionic transport changes in the inflamed intestine (6-7).

Direct evidence of mast cell involvement in epithelial secretion comes from studies using sensitized mast-deficient mice (W/W^v) (8). In these rodents, a 70% reduction in the secretory response to serosal antigen was shown in comparison to normal congenic mice. Moreover, this response was entirely recovered after reconstitution of mast cells population by injecting bone marrow-derived mast cell precursors.

The final support for mast cell involvement comes from studies on cultured epithelial cells. Using human colonic adenocarcinoma monolayers (HCA-7) sandwiched with peritoneal mast cells from sensitized guinea pigs, Baird et al showed that when mounted on Ussing chambers, antigen challenge increased chloride secretion only when added to the serosal side of the epithelium, where mast cells are normally located *in vivo*, and only in preparations with sensitized mast cells (9). Taken together, these findings strongly argue in favor of mast-cell nerve functional units as important players of the neuroimmune orchestra regulating epithelial physiology.

Both, epithelial and vascular permeability, to small and large molecules has been shown to be increased in many intestinal inflammatory disorders. Using small probes such as ⁵¹Cr-labeled ethylenediaminetetraacetic acid (⁵¹Cr-EDTA) or ⁹⁹Tc-diethylenetriaminopentaacetate (⁹⁹Tc-DTPA), thought to cross the epithelium through the paracellular route, increased baseline gut permeability and overall dramatic increase after antigen challenge or cholinergic stimulation have been shown in the OVA and *N. brasiliensis* rat models (10-11) and in normal rats (12) in association with mast cell activation. The antigen-induced changes were inhibited by serosal application of TTX but mast cell activation was not prevented suggesting that neural activation could be directly related to the release of some mast cell mediators (10). In the *T. spiralis* model antigen challenge was associated with increased permeability to both ⁵¹Cr-EDTA and the larger probe, ¹²⁵I-bovine serum albumin (BSA) (13) indicating mast cell participation in the process.

Other studies have used HRP as a model protein macromolecule to assess permeability changes, since the intact molecule can be measured by a sensitive enzymatic assay and its reaction product can be visualized in tissues and cells by electron microscopy. In normal animals, endosomal epithelial transport of HRP takes 20-30 min, but in rats sensitized to HRP, this process was shown to be more than 10-fold faster and the amount of antigen transported was

also dramatically increased. Using mast-cell deficient (Ws/Ws) rats, it was clearly shown that the initial phase of this transport occurs via endosomes and is mast cell independent, while at later times (20-30 min) neuro-immune interactions appeared to be required for regulation of antigen transport via the paracellular pathway (14).

Finally neural mast cell interactions are also relevant for the control of gut epithelial physiology. Cold pain stimulated mast cell mediator release and water secretion into the jejunum of both normal and food-allergic patients, demonstrating central nervous system ability to modulate intestinal mast cell activity (15). In the colon, acute and chronic stress altered epithelial function and ultrastructural morphology involving both neural pathways and mast cells (16-17).

In summary, increasing evidence is forcing us to accept that comprehensive integration of multidirectional interactions among immune and non-immune cells, intestinal flora, the autonomic, enteric and central nervous systems, is needed to understand gut inflammatory conditions. Progress in this field may help to develop new preventive and therapeutic strategies for immune-regulated gut disorders.

References:

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3. Lin TJ. Mast cells and eosinophils in mucosal defenses and pathogenesis. In *Mucosal Immunology*, Ogra PL, Mestecky J, Lamm ME, Strober, Bienenstock J, McGhee JR, pp 469-482, CRC Press, San Diego, CA, 1999
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8. Perdue MH, Masson S, Wershil BK, Galli SJ. Role of mast cells in ion transport abnormalities associated with intestinal anaphylaxis. Correction of diminished secretory response in genetically mast cell deficient W/W^v mice by bone marrow transplantation. *J Clin Invest* 1991; 87:687-693.
9. Baird AW, Cuthbert AW, McVinish LJ. Type I hypersensitivity reactions in reconstructed tissues using syngeneic cell types. *Br J Pharmacol* 1987; 91:857-869.
10. Crowe SE, Soda K, Stanis AM, Perdue MH. Intestinal permeability in allergic rats: nerve involvement in antigen-induced changes. *Am J Physiol* 1993; 264: G617-623.
11. McKay DM, Bienenstock J. The interaction between mast cells and nerves in the gastrointestinal tract. *Immunol Today* 1994; 15: 533-538.
12. Santos J, Saperas E, Mourelle M, Antolín M, Malagelada J-R. Regulation of intestinal mast cells and luminal protein release by cerebral thyrotropin-releasing hormone in rats. *Gastroenterology* 1996; 111:1465-1473.
13. Ramage JK, Stanis A, Scicchitano R, Hunt RH, Perdue MH. Effect of immunologic reactions on rat intestinal epithelium. Correlation of increased permeability to chromium 51-labeled ethylenediaminetetraacetic acid and ovalbumin during acute inflammation and anaphylaxis. *Gastroenterology* 1988; 94:1368-1375.
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15. Santos J, Saperas E, Nogueiras C, Mourelle M, Antolín M, Cadahia A, Malagelada JR. Release of mast cells mediators into the jejunum by cold stress in humans. *Gastroenterology* 1998; 114: 640-648.
16. Santos J, Benjamin M, Yang P-C, Prior T, Perdue MH. Chronic stress impairs rat growth and jejunal epithelial barrier function. Role of mast cells. *Am J Physiol Gastrointest Liver Physiol* 2000; 278:G847-854.
17. Santos J, Yang P-C, Söderholm JD, M Benjamin, Perdue MH. Role of mast cells in chronic stress-induced colonic epithelial barrier dysfunction in the rat. *Gut* 2001; 48 630-636.

Case Information & Notes

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Residency: Bellevue Hospital Center, New York, NY
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Current Faculty Appointments: NYU School of Medicine, John Jay College of Criminal Justice
Current Job Description: Research/Private Practice
Disclosure Statement: Legacy for Life – Presenter’s Spouse is Distributor

SPEECH TITLE: “**Immune Modulation Using Hyper Immune Egg**”

The speaker has provided the information below.

1.) Goals and objectives: a) To understand the immunology of hyperimmune egg as distinct from hyperimmune milk or colostrums; B) To present clinical trials and case studies in which hyperimmune egg has demonstrated efficacy

2.) Conclusion of what is to be learned: Hyperimmune egg is a nutritional supplement that passively transmits immunoglobulins and other immune mediators that balance the immune system, decrease inflammation, decrease infection, increase energy, and maintain cardio-vascular integrity.

3.) References:

- a) Carlander, D 2002 Avian IgY: in vitro and in Vivo Comprehensive Summaries of Uppsala Dissertations from the Faculty of Medicine. Acta Universitatis, Upsaliensis Upplala 1119:1-53
- b) Greenblatt H et al. 1998 Administration to arthritis patients of a dietary supplement containing immune egg: an open-label pilot study J Medicinal Food 1:171-178
- c) Hatta H et al. 1997 Passive immunization against dental plaque formation in humans: effect of a mouth rinse containing egg yolk antibodies (IgY) specific to streptococcus mutans Caries Research 31:268-74
- d) Karge WH et al 1999 Pilot study on the effect of hyperimmune egg protein on elevated cholesterol levels and cardiovascular risk factors J of Medicinal Food 2:51-63
- e) Melamed I et al 1991 Benefit of oral immune globulin therapy in Patients with immunodeficiency and chronic diarrhea J Pediatrics 119:486-9
- f) Yolken R. et all 1988 Antibodies to rotaviruses in chicken’s eggs: a potential source of antiviral immunoglobins suitable for human consumption Pediatrics 81:291-5

Case Information & Notes

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Board Certifications:

Pediatrics/Environmental Medicine

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Current Job Description:

President American Board of Environmental Medicine;
President Allergy and Environmental Health Center WNY

Other Information (including titles of books or articles you have recently written):

Disclosure Statement:

None

SPEECH TITLE: “**Inflammation as it relates to Post Partum Hemorrhage**”

The speaker has provided the information below.

1.) Goals and objectives: 1) To demonstrate efficacy of environmental approach to diagnose and treat vasculitis resulting in post partum hemorrhage requiring blood transfusion. 2) To demonstrate efficacy of environmental medicine approach to modify genetic susceptibility by providing optimal prenatal treatment for multiple sensitivities and nutritional deficiency.

2.) Outline: · Presentation of a case having prenatal symptoms, followed by postnatal complications who received only bandage treatment with traditional medicine approach.

· Discuss newer methods of comprehensive evaluation and treatment for a patient with history of post partum hemorrhage requiring transfusion.

3.) Conclusion of what is to be learned: 1)Exposure to ambient levels of chemicals in a new home, mold & food sensitivities play a great role as triggers of vasculitis. 2) Nutritional deficiency like magnesium play a great role in premature uterine contraction &/or early termination of pregnancy and abruption placenta. 3) Chemicals, molds and foods when combined with nutritional deficiency have a synergistic effect; play a great role in prenatal, natal and postnatal complications of pregnancy.

4.) References:

Sunday, June 27, 2004

8:25 **ANNOUNCEMENTS/MODERATOR: Douglas B. Seba, Ph.D.**

8:30 **Dennis Hooper, M.D., Ph.D.**, Department of Pathology, Baptist Health Systems, San Antonio, TX,
Title: "Molecular Evaluation of Autopsy tissue in Patients with Fungal Diseases"

8:50 Q & A

9:00 **Professor Kaye H. Kilburn, M.D.**, Director of Environmental Sciences Lab., University of
Southern California, Keck School of Medicine, Alhambra, CA, *Title: "How do Chemical
Exposure, Sensitivity and Impairment Overlap?"*

9:20 Q & A

9:30 **Andrew Campbell, M.D.**, Medical Director, Medical Center for Immune & Toxic Disorders,
Spring, TX, *Title: "Neuroimaging Abnormalities in Mold Exposed Patients"*

9:50 Q & A

10:00 BREAK WITH EXHIBITORS

10:30 **Russel J. Reiter, Ph.D.**, Professor, UT Health Science Center, San Antonio, TX, *Title: "The
Antioxidant Melatonin: Potent Neuroprotective Effects"*

10:50 Q & A

11:00 **Tang Lee, MRAIC**, Professor, Faculty of Environmental Design, Calgary, Alberta, Canada, *Title:
"Worker Exposure in a Wood Panel Plant"*

11:20 Q & A

11:30 **Larry Wolford, D.M.D.**, Academics, Private Practice, Research, Dallas, TX, *Title:
"Chlamydia/Mycoplasma Role Bacteria Affects on the TMJ and Other Body Systems"*

11:50 Q & A

12:00 SUMMARY AND CLOSE: Douglas B. Seba, Ph.D.

SUNDAY, JUNE 27, 2004

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

Dennis G. Hooper, M.D., Ph.D.

Date of talk: Sunday, June 27, 2004,
8:30am

Department of Pathology
UT Health Screening Center
Baptist Health Systems
111 Dallas St.
San Antonio, TX 78205

Phone:
Fax: 210/297-7840
E-mail: 210/527-0495

Medical School/University Attended:

University of Nevada, Reno

Internship:

NAVAL Hospital, San Diego, CA

Residency:

NAVAL Hospital, San Diego, CA

Board Certifications:

Anatomic and Clinical Pathology

Current Faculty Appointments:

Associate Professor, Pathology, University of Texas, Tyler, TX; Associate Clinical Professor, University of Nevada School of Medicine, Dept of Pathology

Current Job Description:

Head of Microbiology/Transfusion Service, Baptist Med. Center, San Antonio, TX; Pathologist and Associate Professor, University of Texas, Tyler, Dept. of Pathology.

Other Information (including titles of books or articles you have recently written):

Published many articles

Disclosure Statement:

SPEECH TITLE: "Molecular Evaluation of Autopsy tissue in Patients with Fungal Diseases"

The speaker has provided the information below.

1.) Goals and objectives:

2.) Conclusion of what is to be learned:

3.) References:

Molecular Evaluation of Autopsy tissue in Patients with Fungal Diseases

Dennis G. Hooper, Ameripath, San Antonio, and Baptist Medical System, Dept. of Pathology,
San Antonio, TX

Patients with histories of exposure to fungal elements or a history of hypersensitivity pneumonitis and taken to autopsy have been evaluated and findings are reported. Lung, brain, and liver tissue have been probed with various DNA probes for fungal elements. DNA probes include but are not limited to 10 various *Aspergillus* species, two *Stachybotryous* species, and many *Penicillium* species. Findings are compared to culture results and histological evaluations. Tissues are also evaluated for various mycotoxin studies from *Aspergillus* sp., *Stachybotryous* sp., and *Penicillium* species. Mycotoxin analysis using Solution Fluorimetry (VICAM), and ELISA techniques are presented. Validation and correlation studies are also presented.

Abstract Information & Notes

Kaye H. Kilburn, M.D.

Date of talk: Sunday, June 27, 2004, 9:00am

University of Southern California
Keck School of Medicine
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Alhambra, CA 91803

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E-mail: kilburn@usc.edu

Medical School/University Attended:
Internship:

University of Utah College of Medicine
Western Reserve, University Hospitals
Cleveland – Internal Medicine

Residency:

University of Utah Medicine, Pathology,
Duke – Cardiopulmonary Physiology and
University of London Cardiology

Board Certifications:

Diplomate American Board of Internal
Medicine and American Board of Preventive
Medicine Occupational Health

Current Job Description:

Ralph Edgington Professor of Internal
Medicine University of Southern California
Keck School of Medicine.

Other Information (including titles of books or
articles you have recently written):

Books: “Chemical Brain Injury”, New York,
John Wiley 1998
“Endangered Brains”, 2004 Princeton Sci
Publishers Co., Inc., Birmingham, AL
“Avoiding Chemical Plagues” in revision

Disclosure Statement:

None

SPEECH TITLE: “How do Chemical Exposure, Sensitivity and Impairment Overlap?”

The speaker has provided the information below.

1.) Goals and objectives: To compare the symptoms and triggers of chemical impairment and chemical sensitivity in 65 mold exposed and 100 chemically exposed to 46 community controls without exposure to chemicals.

2.) Conclusion of what is to be learned: The near identity of symptom frequencies and profiles in people exposed to molds and chemicals, the high similarity of trigger agents and almost the same impairment scores suggests volatile organic agents in molds and in gaseous chemicals damage brain function similarly.

Comment-It is clear that symptom patterns do not distinguish between causes of chemical intolerance. All of these symptoms resemble boats, raised by the tide of chemicals. The opportunity to test children exposed in moldy homes showed children are equally impaired and follow up testing in 16 of 108 exposed people found those impaired seldom regain function.

3.) References: Kilburn KH. Indoor mold exposure associated with neurobehavioral and pulmonary impairment. *Arch Environ Health* 2003; 58:390-398.

Kilburn KH: Endangered Brains, Birmingham, Alabama, Princeton Scientific Publications, 2004.

Kilburn KH. Chemical intolerance from molds versus chemicals indoors. In preparation.

Dales R, Burnett R, Zwanenburg H. Adverse health effects among adults exposed to home dampness and mold. *Am Rev Respir Dis* 1991; 143:505–09

Smoragiewicz W, Cossette B, Boutard A, et al. Trichothecene mycotoxins in the dust of ventilation systems in office buildings. *Int Arch Occup Environ Health* 1993; 65:113–17

Johanning E, Morey P, Jarvis B, et al. Health and immunology study following exposure to toxigenic fungi *Stachybotrys chartarum* in a water damaged office environment. *Int Arch Occup Environ Health* 1996; 68:207–18

Andersson MA, Nikulin M, Kõljalg U, et al. Bacteria, molds and toxins in water-damaged building materials. *Appl Environ Microbiol* 1997; 63:387–93

How do Chemical Exposure, Sensitivity, and Impairment Overlap?

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University of Southern California
Keck School of Medicine
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Alhambra, CA 91803
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Phone: 626-457-4202

The patterns of 16 frequent symptoms and 20 common chemical exposures in 100 consecutive chemical exposed patients and 100 mold exposed patients who had abnormalities of brain function were compared to those of 46 unexposed people from a small desert town. Abnormalities of brain function averaged 10.1, 9.8, and 2.2 respectively. Frequencies of the 16 symptoms were increased 10 times (to 22.5 and 21.1) in both exposed groups above the 2.3 of the town's people controls. These controls most common symptoms were irritation of eyes, throat, and airway followed by headache and lightheadedness. In contrast chemically triggered MCS patients most common listed ones were fatigue, memory loss, concentration, confusion, irritability then throat irritation and headache. The ratio of frequency of symptoms between 100 chemically exposed people and unexposed people was highest for confusion at 70, then 37 for memory loss, 36 for vertigo and 34 for memory dropping to 18 for concentration and 17 for fatigue. For 100 mold/mycotoxin exposed patients the frequencies and the patterns were similar. Almost all people with chemical brain damage were intolerant to chemicals and therefore should be considered chemically sensitive. Acute symptoms stimulated by chemicals often lasted for weeks. Absence of a workable method to measure relative sensitivity makes further comparisons difficult but the ratio of symptoms suggests it is at least 10 to 100 fold. Inhaling mold of home produces similar symptoms to other chemicals which suggest chemicals mediate mold toxicity.

ABSTRACT

How Do Chemical Sensitivity and Impairment Overlap?

Background

The similarity of eye and airway and skin irritation coupled with fatigue and loss of memory, concentration, and balance occurring in people exposed to moldy homes, schools, and offices and exposed by inhalation of chemicals led to comparison of symptoms and inciting agents in groups of chemically exposed, mold exposed, and unexposed people.

Objective

To find shared factors and differentiators in complaints of mold exposed, chemically exposed, and unexposed people.

Groups

100 consecutive patients referred for neurotoxicological evaluations after chemical exposure and 65 referred after mold exposures were contrasted and compared with 46 unexposed community controls.

Method

A pretested questionnaire asked about 20 complaint triggers and frequencies of 16 complaints was completed during neurobehavioral testing of 26 functions for performance score.

Results

Symptoms led by fatigue and losses of memory and concentration had nearly identical profiles and nearly the same frequencies for mold 21.1 (Figure 1) as for chemical exposed 22.5 (Figure 2) that were approximately 10 times the 2.3 (mostly irritation) of unexposed people (top of Figures 1,2). Numbers of triggering agents were similar at 5.9 in mold (Figure 3) and 8.2 in chemically exposed (Figure 4), but differed in that cigarette smoke and perfume led in the mold group whereas paint, diesel exhaust, and gasoline (all hydrocarbons) headed the list for chemically exposed. Both were greatly different from the unexposed. Neurobehavioral abnormalities averaged 9.9 in mold exposed; 10.5 in chemically exposed, versus 3.9 in unexposed people, thus they were parallel (Figure 5,6,7).

Discussion

The patient groups exposed to molds/mycotoxins and to insecticides and to other chemicals had similar profiles and mean values for symptoms that exceeded referent people by 10 fold. Their chemically triggered symptoms were also similar and exceeded and differed from referent people. Their average number of neurobehavioral impairments was similar and 2.5 times referent values. Neither neurobehavioral impairments, profiles of symptoms, nor triggers distinguished those exposed to mold from those chemically exposed.

Elicitation or triggering of many diverse symptoms by chemicals, from mold exposures occurs as frequently and in the same pattern. The symptoms patterns differences were small with fatigue, memory loss, and inability to concentrate leading the list for both. In contrast the unexposed population listed chemical related eye irritation first followed by headache, throat irritation, lightheadedness, airway irritation, and irritability.

The 3 most frequent triggering agents for chemically exposed people were hydrocarbons: paint, diesel exhaust, and gasoline followed by perfume, cigarette smoke, and detergents. It appears that the mold exposed are different listing the top 3 as cigarette smoke, perfume, gasoline, followed by diesel exhaust, the catch-all others and buildings. This pattern was more like a magnified version of the triggers for the unexposed group. Cigarette smoke, perfume, diesel exhaust, paint, insect aerosols, and noise any difference seems minor and the search for clues continues.

This indirect evidence is consistent with the mold disorder being caused by inhaled chemicals, probably mycotoxins such as trichothecenes (T-2) or satratoxin, a dimmer or mold enzymes excreted to digest nutrients such as proteins and cellulose. It suggests that molds are important exposures contributing to or dominating the indoor air or sick building syndrome. When this began is unknown but other community studies and published observations suggest between 1991 and 1997 (Dales et al 1991, Smoragiewicz et al 1993, Johanning et al 1996, Andersson et al 1997).

Conclusions

The near identity of symptom frequencies and profiles in people exposed to molds and chemicals, the high similarity of trigger agents and almost same impairment scores suggests volatile organic agents in molds and in chemicals damage brain function similarly

Slide 1

How Do Chemical Sensitivity and Impairment Overlap?

Slide 2

Goals:

- 1) To compare the symptoms and triggers of chemical impairment and chemical sensitivity in 65 mold exposed and 100 chemically exposed to 46 community controls without exposure to chemicals.
- 2) To examine 35 symptoms in 8 groups for coherence, overlap, and specificity by calculated relative contributions (Eigen values).

Slide 3

Conclusions: 1

The near identity of symptom frequencies and profiles in people exposed to molds and chemicals, the high similarity of triggering agents, and almost the same impairment scores suggests volatile organic agents in molds and gaseous chemicals damage brain function similarly.

Slide 4

Conclusions: 2

The coherence and overlap between symptoms in these patients examined by Eigen values was so high that one symptom group explains 90% of the variance and 2 or 3 groups all of it.

\therefore Symptoms lack specificity.

Abstract Information & Notes

Andrew Campbell, M.D.

Date of talk: Sunday, June 27, 2004, 9:30am

Medical Center for Immune & Toxic
Disorders
25010 Oakhurst, Ste. 200
Spring, TX 77386

Phone: 281/681-8989
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Medical School/University Attended:

Universidad Autónoma de Guadalajara,
México

Internship:

Orlando Regional Medical Center, Orlando,
Florida

Residency:

Medical College of Georgia, Augusta,
Georgia

Board Certifications:

ABFP, ABME, ABFM

Current Job Description:

Medical Director, Medical Center for
Immune & Toxic Disorder

Other Information (including titles of books or
articles you have recently written):

Published four articles for **The Scientific
World Journal**, in 2003, and has published
many other articles for various publications
since 1992. A list will be available upon
request.

Disclosure Statement:

None

SPEECH TITLE: "Neuroimaging Abnormalities in Mold Exposed Patients"

The speaker has provided the information below.

1.) Goals and objectives:

2.) Conclusion of what is to be learned:

3.) References:

Neuro-imaging Abnormalities in Mold and Mycotoxin Exposed Patients.

Andrew W. Campbell, M.D.
Medical Director
Medical Center for Immune and Toxic Disorders
25010 Oakhurst Drive
Spring, Texas 77386
281 681-8989
md@immunotoxicology.com

Exposure to molds and mycotoxins has been documented in the peer-reviewed medical literature to cause neurotoxicity. This is a study of 112 patients who had documented exposure to molds and mycotoxins with neurotoxic symptoms and abnormal neurophysiological studies. These patients underwent MRI imaging and SPECT perfusion scanning of the brain, further documenting the neurotoxicity of molds and mycotoxins when compared to controls.

Abstract Information & Notes

Russel J. Reiter, Ph.D.

UT Health Science Center
7703 Floyd Curl Drive
San Antonio, TX 78229-3900

Date of talk: Sunday, June 27, 2004,
10:30am

Phone:
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E-mail: 210/567-6948
reiter@uthscsa.edu

Medical School/University Attended:	Bowman Gray School of Medicine at Wake Forest University
Current Faculty Appointments:	Professor of Neuroendocrinology, University Texas Health Science Center, San Antonio
Current Job Description:	Biomedical research; teaching medical students
Other Information (including titles of books or articles you have recently written):	10 authored books; 36 edited books; 750 published research articles; 350 published review articles and chapters; 58 research awards and prizes including 3 honorary doctor of medicine degrees
Disclosure Statement:	None

SPEECH TITLE: “**The Antioxidant Melatonin: Potent Neuroprotective Effects**”

The speaker has provided the information below.

1.) Goals and objectives: Goal: To clarify the potential importance of melatonin in deferring the progression of age-related neurodegeneration. Objectives: To review experimental data documenting melatonin’s effects in experimental models of Alzheimer’s and Parkinson’s disease and to review clinical data indicating melatonin’s beneficial actions.

2.) Conclusion of what is to be learned: Exogenously administered melatonin, the production of which is reduced in the aged, may have significant potential in reducing age associated neurobehavioral degeneration.

3.) References: Reiter RJ, Oxidative damage in the central nervous system: Protection by melatonin. Prog Neurobiol 1998; 56:359-384
Tan DX, Manchester LC, Sainz RM, Mayo JC, Alvares FL, Reiter RJ. Antioxidant strategies in protection against neurodegenerative disorders. Exp Opin Ther Patents 2003; 13: 1513-1543.

THE ANTIOXIDANT MELATONIN: POTENT NEUROPROTECTIVE EFFECTS

Russel J. Reiter

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The antioxidant properties of melatonin in the central nervous system (CNS) seem particularly important. Unlike some other well known free radical scavengers, e.g., vitamin E, melatonin quickly penetrates the blood-brain-barrier to enter the CNS. The ability of melatonin to do so presumably stems from its high lipid solubility. Additionally, the concentration of melatonin in the cerebrospinal fluid (CSF) of the third ventricle may be orders of magnitude higher than in the plasma due to the direct release of melatonin into the third ventricle. From this location, melatonin could readily diffuse into the surrounding neural tissue to protect it from free radical damage.

There are a large number of neurodegenerative diseases of the aged that have, as part of their basis, free radical damage which results in neuronal loss. Examples of such diseases include myasthenia gravis, Huntington's disease, Alzheimer's disease and Parkinsonism, among others. Additionally, stroke, i.e., ischemia/reperfusion injury, has a major free radical component.

There have been numerous studies that have investigated the ability of melatonin to limit tissue damage and neuronal loss after a transient interruption of the blood flow to the brain followed by reperfusion with oxygenated blood. In experimental models of ischemia/reperfusion injury, several different methods have been used to deprive the brain, either locally or globally, of oxygenated blood. The most common method has been occlusion of the middle cerebral artery unilaterally to induce a stroke on the lateral surface of one hemisphere. When this is done, the damaged area becomes highly edematous, tissue damage is extensive, neurons and glial elements degenerate, numerous molecules are oxidized and an infarct (dead tissue) develops. If melatonin is administered just prior to arterial occlusion or at the time reperfusion is established the amount of edema, the number of dead neurons and glia, the quantity of oxidized molecules and the infarct volume is significantly reduced. Additional recent studies have also shown that the neurophysiological deficits that develop after an episode of neural ischemia/reperfusion is less severe if the animals are treated with melatonin. Hence, melatonin not only reduces tissue loss but also preserves functions in animals subjected to local ischemia/reperfusion injury (by temporary occlusion of the middle cerebral artery).

The indole is also highly effective in other models of neural ischemia/reperfusion injury. For example, bilateral occlusion of the carotid arteries (in animals that have an incomplete circle of Willis) leads to extensive forebrain damage; conversely, if melatonin is given before the vessels are occluded or in advance of reperfusion, neural loss and neurophysiological decline are limited. Finally, when animals are subjected to a brief period of cardiac arrest, the brain (as well as all other organs) are deprived of oxygenated blood for a period of time. Melatonin has also been used in this situation to reduce molecular damage in the brain and to limit tissue loss. There are now numerous studies showing the protective actions of melatonin in the brain that is

subjected to a temporary interruption of blood flow followed by reperfusion with oxygenated blood.

In experimental models of Parkinson's disease (PD), melatonin has also been shown to reduce brain damage. The two most common drugs used to cause a PD-like condition in experimental animals are 6-hydroxydopamine and 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). When the latter drug is administered to animals (or accidentally taken by humans) it enters astrocytes in the brain where it is metabolized to MPP⁺. MPP⁺ is then released from the astrocytes and, in the substantia nigra, it is taken up via the dopamine transporter into dopaminergic (DA) neurons. Within DA neurons, MPP⁺ generates free radicals that eventually kill these important cells thereby causing PD. Since this is a free radical-related process many attempts have been made to inhibit PD in experimental animal models using melatonin. In each study where MPTP was acutely administered, the concurrent injection of melatonin greatly reduced DA neuron loss and maintained function. Likewise, in one study where low doses of MPTP were given to animals to induce a slowly-developing PD, melatonin, given in advance of each MPTP injection, reduced the loss of dopaminergic neurons in the substantia nigra and preserved neurophysiological function.

Several models of Alzheimer's disease (AD) have also tested the efficacy of melatonin in protecting against this devastating condition. Amyloid β -peptide (A β) is a major culpable toxic agent in the brain of AD patients. When neurons are grown in the presence of A β , intracellular molecular damage is increased, intracellular concentrations of calcium become markedly elevated, free radical generation ensues and neuronal death follows. If cells treated with A β are grown in the presence of melatonin, cellular damage is diminished (as measured by any parameter) and the number of neurons that survive is greatly increased.

Increasing age is usually associated with an accumulation of A β in the brain, particularly in those individuals destined to develop AD. When transgenic mice, transfected with the human amyloid precursor protein (APP), were observed over a major portion of their life-time, A β concentrations in the brain steadily increased and the animals began to die prematurely. If, however, the mice bearing the human APP gene were given melatonin in their drinking water, the amount of A β deposited in the brain was substantially reduced and the death of the animals was significantly delayed.

Finally, besides reducing A β accumulation in the brain and reducing its neuronal toxicity, melatonin was recently reported to inhibit the phosphorylation of the protein tau in neurons. The hyperphosphorylation of tau is another important feature of AD. In summary, melatonin has several actions that may be potentially beneficial in delaying the onset or reducing the severity of AD. This is particularly interesting considering that endogenous melatonin levels gradually wane during aging and AD patients seem to have especially diminished melatonin levels. Thus, melatonin concentrations seem to be lost at the time it would be most beneficial.

References

Antolin I, Mayo JC, Sainz RM, Rodriguez C. Protective effect of melatonin in chronic experimental model of Parkinson's disease. *Brain Res* 2002; 943:162-173.

Cheung RTF. The utility of melatonin in reducing cerebral damage resulting from ischemia and reperfusion. *J Pineal Res* 2003; 34:153-160.

Pappolla MA, Reiter RJ, Bryant-Thomas TK, Poeggeler B. Oxidative mediated neurodegeneration in Alzheimer's disease: melatonin and related antioxidants as neuroprotective agents. *Curr Med Chem* 2003; 3:233-243.

Reiter RJ, Tan DX, Manchester LC, Qi W. Biochemical reactivity of melatonin with reactive oxygen and nitrogen species: a review of the evidence. *Cell Biochem Biophys* 2001; 34:237-256.

Reiter RJ, Tan DX, Manchester LC, El-Sawi MR. Melatonin reduces oxidative damage and promotes mitochondrial respiration: role of oxidative stress. *Ann NY Acad Sci* 2002; 959:238-250.

Reiter RJ, Tan DX. Melatonin: a novel protective agent against oxidative injury of the ischemia/reperfused heart. *Cardiovasc Res* 2003; 58:10-19.

Abstract Information & Notes

Tang G. Lee, MRAIC

Faculty of Environmental Design
The University of Calgary
2500 University Drive NW
Calgary, Alberta T2N 1N4
Canada

Date of talk: Sunday, June 27, 2004,
11:00am

Phone:
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E-mail: 403/284-4399
lee@ucalgary.ca

Medical School/University Attended:

Case Western Reserve University, Cleveland,
The Ohio State University, Columbus,
Ryerson University, Toronto

Board Certifications:

Royal Architectural Institute of Canada

Current Faculty Appointments:

The University of Calgary

Current Job Description:

Professor

Other Information (including titles of books or
articles you have recently written):

Part owner of and operates one of the largest
tropical fish farm in Canada, raising tilapia
for consumption. The waste heat, humidity
and wastewater discharges into a hydroponics
greenhouse.

Disclosure Statement:

Nil

SPEECH TITLE: "Worker Exposure in a Wood Panel Plant"

The speaker has provided the information below.

1.) Goals and objectives: Understand the types of air contaminants in a wood panel plant and its impact on worker health.

2.) Conclusion of what is to be learned: Learn about health symptoms that are caused by airborne particles and moulds.

3.) References: Lee, T.G. Indoor air contaminants in an OSB plant and its effect on worker health, proceedings of World OSB Symposium, Wyndham Chicago Hotel, October 24, 2002.

Comtois, P. Malo, J-L.: The Air Spora of East-Canadian Sawmills. Proc. 5th International Conference on Indoor Air Quality and Climate, Indoor Air '90, Toronto 109-113 (1990)

Cosentino S., Palmas F.: Occurrence of Airborne Fungal Spores in Industrial Working Environments. Dept. Experimental Biology, University of Cagliari, Italy. Proc. 5th International Conference on Indoor Air Quality and Climate, Indoor Air '90, Toronto 115-119(1990)

Duchaine, C., Meriaux, A.: Airborne microfungi from eastern Canadian sawmills. *Canadian Journal of Microbiology*, National Research Council of Canada, **46** (2000)

Herber, F.A., Hessel, P.A., Melenka, L.S., Yoshida, K., Nakaza, M.: Respiratory Consequences of Exposure to Wood Dust and Formaldehyde of Workers Manufacturing Oriented Strand Board. *Archives of Environmental Health*, **49(6)** 465-470 (1994)

Herbert, F.A., Hessel, P.A., Melenka, L.S., Yoshida, K., Nakaza, M.: Pulmonary Effects of Simultaneous Exposures to MDI Formaldehyde and Wood Dust on Workers in an Oriented Strand Board Plant. *Journal of Occupational and Environmental Medicine* **37(4)** 461-465 (1995)

Mandryk, J., Alwis, K.U., Hocking, A.D.: Effects of Personal Exposures on Pulmonary Function and Work-related Symptoms Among Sawmill Workers. *Annual Occupational Hygiene, British Occupational Hygiene Society* Elsevier Science Ltd., **44(4)** 281-289 (2000)

Worker exposure in a wood panel plant

22nd Annual International Symposium on Man and His Environment in Health and Disease. June 24-27, 2004.

Tang G. Lee, MRAIC
The University of Calgary
Phone: 403-220-6608

ABSTRACT

The Human Resources Co-ordinator reported potential air quality problem in a large wood panel plant that may have caused occupational asthma and related illnesses amongst some workers. Several indoor air quality examinations did not find high concentrations of air contaminants and generally comply with occupational health and safety standards. Nevertheless, the problems and symptoms continue to aggravate some staff members.

On December 2001, an indoor air quality assessment of the plant was proposed.

- To determine the cause of occupant symptoms.
- To determine the sources of air contaminants including gasses, airborne particles and micro-organisms.
- To develop a strategy to mitigate the air contaminants.
- To prepare a report outlining the examination and recommendations.

On January 2002, a site examination was conducted at the plant. Interviews were conducted with some staff members and examination of individual medical records. A local medical doctor met with the examination team to provide his medical opinion of the symptoms that he found in his patients. He also provided 133 survey data and tests done on staff before and after working at this plant.

This investigation identified the type, concentration and sources of the air contaminants in this plant. The contaminant of greatest concern is the wood dust and mould spores. However, there are other air contaminants that also should be addressed such as carbon monoxide emissions from the forklifts, floor sweepers, etc. A set of mitigation measures had been developed for this plant that will help alleviate the problems. There are also several operational changes identified. For example, the plant must prevent discharging chemicals into the log pond and disinfecting and replacing the water in the log pond. Wearing approved respirators when the airborne particles exceed certain levels is also necessary.

The morale of the some plant staff has been compromised by reports of illness caused by air contaminants amongst their fellow workers and their own symptoms. While the illness in some workers cannot be linked directly to the air contaminants in this plant, it is prudent to reduce the concentrations of these contaminants as much as possible.

References:

Lee, T.G., Indoor air contaminants in an OSB plant and its effect on work health. Proceedings of World OSB Symposium, Wyndham Chicago Hotel, Chicago, October 2-4, 2002.

Comtois, P. Malo, J-L.: The Air Spora of East-Canadian Sawmills. Proc. 5th International Conference on Indoor Air Quality and Climate, Indoor Air '90, Toronto 109-113 (1990)

Cosentino S., Palmas F.: Occurrence of Airborne Fungal Spores in Industrial Working Environments. Dept. Experimental Biology, University of Cagliari, Italy. Proc. 5th International Conference on Indoor Air Quality and Climate, Indoor Air '90, Toronto 115-119 (1990)

Duchaine, C., Meriaux, A.: Airborne microfungi from eastern Canadian sawmills. *Canadian Journal of Microbiology*, National Research Council of Canada, **46** (2000)

Herbert, F.A., Hessel, P.A., Melenka, L.S., Yoshida, K., Nakaza, M.: Respiratory Consequences of Exposure to Wood Dust and Formaldehyde of Workers Manufacturing Oriented Strand Board. *Archives of Environmental Health*, **49(6)** 465-470 (1994)

Herbert, F.A., Hessel, P.A., Melenka, L.S., Yoshida, K., Nakaza, M.: Pulmonary Effects of Simultaneous Exposures to MDI Formaldehyde and Wood Dust on Workers in an Oriented Strand Board Plant. *Journal of Occupational and Environmental Medicine* **37(4)** 461-465 (1995)

Mandryk, J., Alwis, K.U., Hocking, A.D.: Effects of Personal Exposures on Pulmonary Function and Work-related Symptoms Among Sawmill Workers. *Annual Occupational Hygiene, British Occupational Hygiene Society Elsevier Science Ltd.*, **44(4)** 281-289 (2000)

Abstract Information & Notes

Larry Wolford, D.M.D.

Date of talk: Sunday, June 27, 2004, 8:30am

Baylor University School of Dentistry
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Dallas, TX 75246

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E-mail: lwolford@swbell.net

Medical School/University Attended:

Temple University School of Dentistry,
Philadelphia, PA

Internship:

University of Texas Southwestern Medical
School and Parkland Hospital, Dallas, TX

Residency:

Same

Board Certifications:

Oral and Maxillofacial Surgery

Current Faculty Appointments:

Clinical Professor Baylor College of
Dentistry

Current Job Description:

Academics, Private Practice, Research

Other Information (including titles of books or
articles you have recently written):

Co-Authored 2 text books, written 19 book
chapters for other books, published over 145
articles.

Disclosure Statement:

None

SPEECH TITLE: "Chlamydia/Mycoplasma Affects TMJ and other Systems"

The speaker has provided the information below.

1.) Goals and objectives: Demonstrate Chlamydia/Mycoplasmata affects the TMJ's and other body systems. PCR DNA identification effective means for finding these bacteria. Chlamydia/Mycoplasma bacteria can affect any body system.

2.) Conclusion of what is to be learned: Chlamydia/Mycoplasma bacteria may contribute to TMJ pathosis. The presence of Chlamydia/mycoplasma bacteria in TMJ correlates to increased problems in other body systems

3.) References: See abstract

Chlamydia/Mycoplasma Bacteria Affects on the TMJ and Other Body Systems

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Study #1: We have previously demonstrated the presence of Chlamydia trachomatis, Mycoplasma fermentans, and Mycoplasma genitalium in the bilaminar tissues of the human temporomandibular joint (TMJ). A group of 26 patients (24 F, 2M) underwent TMJ surgery. During the normal course of surgery, tissues usually removed and discarded, were immediately snap frozen and stored until analysis. **Results:** Bacterial DNA was identified in the TMJ tissues as follows: C. trachomatis 11/26 (42%), M. fermentans 6/26 (23%), and M. genitalium 9/26 (35%). Eight of 26 patients had concurrent infections as follows: C. trachomatis with either M. fermentans or M. genitalium in 5/26 (19%), M. fermentans with M. genitalium 2/26 (8%), and all three bacteria in 1/26 (4%). In this study, 19/26 (73% of the patients were positive for the presence of bacteria.

Study #2: The senior author observed that in his TMJ patient population, some of the sickest patients with severe intractable pain had major exposure to birds either currently or in the past. Chlamydia psittaci, a sister bacteria to Chlamydia trachomatis, is an animal pathogen, particularly common in birds, and causes zoonotic disease. A study was performed on 55 patients (52 F, 3 M) who had significant contact with birds by history. Ten other female patients without exposure to birds were used as controls. PCR analysis was performed for DNA identification of C. psittaci in tissues removed from the TMJ area. **Results:** 34 of 55 patients (62% with bird contact, but none of the 10 controls, were PCR positive for Chlamydia psittaci DNA sequences. We also had a veterinary tissue sample from a pet bird owned by one of the patients in the study, and that sample and the patient were PCR positive for the bacteria. Chlamydia psittaci is present in the TMJ bilaminar tissues of some patients with significant exposure to birds and may be involved in the pathogenesis of the TMJ.

Study #3: This study was undertaken to determine the involvement of other body systems in TMJ patients exposed to birds as compared to controls (TMJ patients with no known bird exposure). Data from 100 patients (89 F, 11 M), ages 14 to 65 years were analyzed. There were 60 patients exposed to birds and 40 control patients with exposure to birds had significantly elevated rates of occurrence of problems in other body systems (from 14% to 40% greater) as compared to the control group. This increased occurrence in other body systems may indicate a systemic Psittacosis in the TMJ population with significant exposure to birds.

References:

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