Nineteenth Annual International Symposium
on
Man and His Environment in Health and Disease

Special Focus

The Environmental Aspects of Neurotoxicity

Sponsored by
American Environmental Health Foundation 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 through the joint sponsorship of the American Academy of Environmental Medicine (AAEM) and the American Environmental Health Foundation. 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.

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 2001 conference will focus "The Environmental Aspects of Neurotoxicity", toxic damage to the brain and nerves. 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 Neurotoxicity.
Use new concepts and treatments to help better diagnose and manage many patients with chemical sensitivity and Neurotoxicity.
Apply the concepts of this conference to your practice using nutrition and environmental manipulation for the treatment of Neurotoxicity.
Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness of your practice in relation to Neurotoxicity.

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

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

CONFERENCE FORMAT

The AEHF Committee has selected some of the leading experts in the field of Environmental Aspects of Neurotoxicity.

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.)

Thursday and Saturday afternoon, we will have a case study/panel discussion. This session will consist of various faculty members discussing real cases. The audience is encouraged to participate in these discussions.

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 Griffiths, Ph.D.,
Environmental Health Center - Dallas
Dallas, Texas

Satoshi Ishikawa, M.D.
Clinical Environmental Health Center, Kitasato Hospital
The Kitasato Institute
Machida-shi, Tokyo Japan

Kaye H. Kilburn, M. D.
University of Southern California Medical Center
Keck School of Medicine
Los Angeles, CA

William J. Meggs, M.D., Ph.D.
E. Carolina University School of Medicine
Dept. of Emergency Medicine
Greenville, NC

THURSDAY, JUNE 7, 2001 FACULTY

Pierre Auger, M.D.
Clinique De Santé Au Travail et De Santé Environnementale
Montreal, Quebec Canada
514/849-5201

Deborah Baird, M.D.
Environmental Health Center-Dallas
Rockwall, TX
972/771-1106

C. Malcolm Beck
Garden-Ville, Inc.
San Antonio, TX
201/651-6115

Clement E. Furlong, Ph.D.
Department of Genetics
University of Washington
Seattle, WA
206/543-1193

Kaye H. Kilburn, M. D.
University of Southern California Medical Center
Keck School of Medicine
Los Angeles, CA
323/442-1830

Helmuth Muller-Mohnssen, Ph.D.
Ludwig-Maximilians Univ. Munich
Ismaning/Menich, Germany
011-49-89-969444

William J. Rea, M.D.
Environmental Health Center - Dallas
Dallas, TX
214/3684132

Doug Seba, Ph.D.
Alexandria, VA 22313
703/949-1055

NINETEENTH ANNUAL INTERNATIONAL SYMPOSIUM
ON
MAN AND HIS ENVIRONMENT

SCHEDULE FOR THURSDAY, JUNE 7, 2001

11:00 a.m. REGISTRATION

12:45 p.m. WELCOME/MODERATOR: William J. Rea, M.D., George C. Miller, II, M.D., Gary R. Oberg, M.D.

1:00 Doug Seba, Ph.D. "Environmental Update 2001: A Health Odyssey"
1:20 Q & A

1:30 C. Malcolm Beck "Insects and Our Health"
1:50 Q & A
2:00 Kaye H. Kilburn, M. D. "Neurotoxicity from Chlorine and Hydrogen Sulfide"
2:20 Q & A

2:30 H. Muller-Mohnsen, Ph. D, "Health Effects Caused by Pyrethrine or Pyrethroid Exposure in Civil Aircrew Members"
2:50 Q & A

3:00 BREAK WITH EXHIBITORS

MODERATOR: Jean A. Monro, M.D.

3:30 Clement E. Furlong, Ph.D."The Human Paraoxonase (PON1) Polymorphism: Role in Detoxication of Organophosphorus Insecticides"
3:50 Q & A

4:00 William J. Rea, M.D. "Neurotoxicity-Central Nervous System"
4:20 Q & A

4:30 Pierre L. Auger M.D., "Chronic Toxic Encephalopathies Apparently Related to Exposure to Toxicogenic Fungi"
4:50 Q & A

5:00 CASE STUDIES & PANEL/MODERATOR: Allan D. Lieberman, M.D.

1.) Deborah Baird, M.D. "Seven Year Case Study of Acute Neurotoxicity"

2.) Case Study

6:00 ADJOURN

THURSDAY, JUNE 7, 2001

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

Douglas B. Seba, Ph.D. Date of talk: Thursday, June 7, 1:00 p.m
Phone: 703/949-1055
P.O. Box 1417, #323 Fax: n/a
Alexandria, VA 22313 E-mail: n/a

Medical School Attended: Ph.D., University of Miami
Residency: n/a
Board Certifications: n/a
Current Faculty Appointments: None
Current Job Description: Independent Marine Scientist
Other Information: 35 Years of Experience in Toxicology and Environmental Chemicals

Disclosure Statement: None

SPEECH TITLE: **"Environmental Update 2001: A Health Odyssey"**

The information below has been provided by the speaker.

1.) Goals and objectives: To review major environmental phenomenon as we enter the next millennium

2.) Outline of talk/abstract: Synthetic chemicals, radionuclides, global warming, and increasing dust will be reviewed for contemporary aspects

3.) Conclusion of what is to be learned: That there are a plethora of adverse health effects at vast distances from their environmental origin

4.) References: Drawn from the most recent contemporary science news sources at the time of presentation

ENVIRONMENTAL UPDATE 2001: A HEALTH ODYSSEY

Douglas B. Seba

Objectives
This paper will review major environmental phenomenon as we enter the next thousand years. An odyssey is a long wandering or voyage usually marked by many changes in fortune and that certainly describes human health as we stand on this millennial cusp. This review should help set the perspective for this entire conference.

Abstract
Synthetic chemicals, radionuclides, electronic smog, global warming and increasing dust all vie to be the leading causes of compromised health as we enter this new era. Dust alone carries endocrine disruptors and carcinogens, heavy nuclides and large insects, organic debris and

bacteria, fungi, and viruses. It is a virtually soup of assaults on our health and it is everywhere. As old forgotten plagues once again become the stuff of daily headlines we must quickly embrace the paradigm of environmental health if we are to keep the health gains of the last millennium.

In just the last 50 years or 5% of the millennium the average visibility east of the Mississippi River has decreased by one-half and an area the size of Canada over the Indian Ocean south of India has become covered in constant haze. People lost the ability to see Venus during the day early in the millennia (30%). Thus, no one alive today is breathing air even remotely as clean as our ancestors of a few dozens generations ago. This is a true challenge to our continued health progress.

Conclusion
There are a plethora of adverse health effects at vast distances from their environmental origin. Understanding their fate and transport is essential to patient environmental health.

References
References will be drawn from the most recent contemporary science news sources at the time of presentation.

Abstract Information & Notes

C. Malcolm Beck Date of talk: Thursday, June 7, 1:30 p.m.
Garden-Ville, Inc. Phone: 210/651-6115
7561 East Evans Rd. Fax: 210/651-9231
San Antonio, TX 78266 E-mail: n/a

Medical School Attended: n/a
Current Job Description: Full time researching and giving presentations all over U.S. on understanding nature. Average 80 talks or slide presentations a year to Universities, Colleges, Master Gardeners, Farmers, Ranchers, Home Owners Assn Etc.

Disclosure Statement: None

SPEECH TITLE: **"Insects and Our Health"**

The information below has been provided by the speaker.

1.) Goals and objectives: To change peoples perception of insects, to prove they were all designed for our benefit.

2.) Outline of talk/abstract: Using color slides of insects to show the different life stages of insects, and benefits of each, and how to control troublesome ones without using toxic products.

3.) Conclusion of what is to be learned: If studied from correct point of view you discover that every living creature has something to show, teach or tell us, give us or prevent us from doing stupid things.

4.) References: A life time of playing with bugs and studying insects and their relation to human, animal and plant life, reading many books and publications, mostly, a burning desire to understand why the insects exist.

INSECTS & OUR HEALTH
C. Malcolm Beck

Of all the millions of insect species known, only one percent are considered pests. If we truly understood the role of these so-called bad bugs, however, we wouldn't be so quick to condemn.

Of all the living creatures on this planet, only humans have a free will to love, hate, reason and think logically. Every other living thing is programmed too only be what it is and do what it does.

If we consider ourselves to be the highest and most intelligent creatures on Earth, should not everything else, including the pest, be here to be of service and aid to us, to give us something, does something for us, to teach us something or even prevent us from doing something? If we study from this point of view, we soon discover reasons for pests. Instead we put our money and research into designing toxic ways to destroy pests. More than two billion pounds of pesticide selling for $24 billion are used each year in this country. Discovering reasons, for the existence of pests and nontoxic ways of controlling them would cut severely into the profits of this industry.

Organic gardeners and good farmers have known for years that weak, sick plants attract and can be destroyed by the pest insects and diseases. This is Nature's law, Survival of the Fittest. The so-called bad bugs are here to cull the plants so only the best survive to produce healthy food and seed for generations to come. The lady bugs and other predators and parasitic insects are here acting as a police force to keep the plants' culling insects in check so they can't overdo the culling job. Science has proven this with discovery that lady beetles prefer to feed on pest insects that randomly get on healthy plants. My own research has proven that plants have an immune system, but only when plants are properly grown and are healthy.

The pests are one of the ways Nature speaks to us. They tell us when our soil is becoming weaker and our environment is getting more polluted. We must set our pride aside and listen. Nature is an infallible teacher. She has the answers. But, she doesn't force us to listen. She doesn't force us to learn. We have a free will. She allows us to stupidly ignore her and destroy the environment and ourselves with our pseudo-science, while we try to dominate her.

We either learn to listen and survive, or we will deteriorate to near extinction. Then, Nature can and will renew.

Sources: A lifetime of playing with bugs and studying insects and their relation to human, animal and plant life. Many books and periodic publications. Mostly a burning desire to understand why the insects exist.

Abstract Information & Notes

Kaye H. Kilburn, M. D. Date of talk: Thursday, June 7, 2:00 p.m.

University of Southern California Medical Center Phone: 323/442-1830
Keck School of Medicine Fax: 323/442-1833
2025 Zonal Ave., CSC-201 E-mail: kilburn@usc.edu
Los Angeles, CA 90033

Medical School Attended: University of Utah College of Medicine
Residency: University of Utah Hospitals
Board Certifications: California, Louisiana, North Carolina, Missouri, Wyoming, New York
Current Faculty Appointments: Professor of Medicine, University of Southern California Keck School of Medicine
Current Job Description: Director of Environmental Sciences Lab, Ralph Edgington Professor of Medicine, University of Southern California - Keck School of Medicine
Other Information: Editor-in-Chief, Archives of Environmental Health and President & Director, Neuro-Test, Inc.

Disclosure Statement: None

SPEECH TITLE: **"Neurotoxicity from Chlorine and Hydrogen Sulfide"**
The information below has been provided by the speaker.

1.) Goals and objectives: To visualize and contrast the effects of chlorine and hydrogen sulfide on human brain function from epidemiological studies using neurobehavioral measurements.

2.) Outline of talk/abstract: Abstract furnished

3.) Conclusion of what is to be learned: Chlorine has permanent effects that worsen progressively to atrophy. Inhibition of blink reflex appears reversible. Hydrogen sulfide may hit and run with great damage but low doses insidiously subtract function. A breath or two of ether at industrial concentrations damages the brain irreversible.

4.) References: Kilburn KH and Warshaw RH: Hydrogen sulfide and reduced sulfur gases adversely affect neurophysiological functions. *Toxicol Ind Health* 11:185-197, 1995.

1. Kilburn KH: Effects of a hydrochloric acid spill on neurobehavioral and pulmonary function. *J Occup Environ Med* 38:1018-1025,1996.
2. Kilburn KH: Exposure to reduced sulfur gases impairs neurobehavioral function. *So Med J* 90:997-1006, 1997.
3. Kilburn KH: Evaluating health effects from exposure to hydrogen sulfide: Central Nervous System Dysfunction. *Envion Epidemio Toxicol* 1:207-216, 1999.

NEUROTOXICITY FROM CHLORINE AND HYDROGEN SULFIDE

Kaye H. Kilburn, M.D.
University of Southern California
Keck School of Medicine
2025 Zonal Avenue, CSC 201
Los Angeles, CA 90033
E-mail: kilburn@usc.edu

Chlorine

We tested for effects on the central nervous system (CNS) and lung of chlorine and "spent" potassium creosol (cresylate). A train derailment in a narrow Montana canyon that exposed about 1,000 people at home when tank cars ruptured. Seven weeks after the spill 97 exposed people were evaluated and 57 were studied after 3.3 years. Twenty-six people were tested on both occasions. Balance, reaction time, color discrimination, contrast sensitivity, visual field performance, blink reflex latency, verbal recall, problem solving, and long-term memory including vocabulary were tested. Pulmonary functions were measured by spirometry.

Questionnaires assessed medical histories, mood states, the frequencies of 37 symptoms, other chemical exposures and preexisting disorders. Comparisons were made to 22 unexposed people from a nearby city. Seven weeks after the exposure, five test scores were significantly different from unexposed groups including balance with eyes closed, simple reaction time, numbers of abnormal visual quadrants, vocabulary and information. Moods state scores and symptom frequencies were elevated. After 3.3 years the additional abnormalities included choice reaction time, balance with eyes open, color discrimination errors in both eyes, visual field performance on the right, Culture Fair and verbal recall, immediate and delayed. Exposure at home to chlorine and creosol produced persistent CNS impairment that increased after 3 years.

Hydrogen Sulfide

The aim was to determine whether proximity to Dakota City lagoons generating hydrogen sulfide (H2S) was associated with diminished neurobehavioral function. The 51 Dakota City exposed subjects were compared first to the Dakota City unexposed group, second to a Wickenburg, AZ unexposed (standard control) group and third with a Tennessee unexposed group. We measured color vision, visual fields, balance, reaction time, blink reflex latency, grip strength and hearing. In addition, cognitive functions, perceptual motor speed as in peg placement and trail making, verbal recall and long-term memory (cultural awareness) were tested. Comparisons were made as a group and by individual abnormalities after adjusting for age, sex, education and other factors.

The 51 exposed people averaged 5.8 abnormalities, the "unexposed" group showed 4.3 abnormalities compared to 2.2 in the Wickenburg unexposed group. The local unexposed group was different from Wickenburg unexposed group. Significant differences between the exposed and Wickenburg unexposed group were found for choice reaction time, balance with the eyes open (balance with the eyes closed was close at $p<.07$), blink reflex latency, color vision errors, (vocabulary was close $p<.057$) and fingertip number writing errors, a total of 5 abnormalities. Several years of H2S exposure was associated with neurobehavioral impairment. Other impairment in local controls may be due to atrazine or famphur (a phosphorothiotic ester).

Notes:

Abstract Information & Notes

Helmuth Muller-Mohnssen, Ph.D. Date of talk: Thursday, June 7, 2:30 p.m.

Ludwig-Maximilians Univ. Munich Phone: 011-49-89-969444
Ismaning/Menich Fax: same
Germany E-mail: n/a

Medical School Attended:
Residency:
Board Certifications:
Current Faculty Appointments:
Current Job Description:
Other Information:
Vita
1928 born in Bremen, Germany
1951 state medical examination Marburg/Lahn
1952 MD, Marburg/Lahn, Germany
1968 Habilitation for Physiology Ludwig-Maximilians-University, Munich (LMU)
Educational background:
1952-1956, Institute of Pathology, University of Münster/Westfalen, Germany (Prof. Giese),
1952-1957 1956-1960 Physiological Institute, University of the Saarland (Prof. Stämptli).
1960-1993 Head of Department of Physiology in the federal center of environmental research GSF, Neuherberg/Munich.
1963 Retired
Working Fields

1. Development of collateral circulation induced by stenosing coronary sclerosis.
2. Hydrodynamic mechanisms involved in thrombogensis and atherogenesis,
3. Intravital-microscopical studies of the Node of Ranvier of isolated single motor nerve fiber during its electrophysiological function.
4. The stationary electrophysical basis of impulse-generation by the excitable nerve fiber membrane (Textbook: *"Physics of nervous excilation with an introduction to methodology of physiological research"*, Munich 1979).
5. Epistemological basis of research politics

SPEECH TITLE: "**Health Effects Caused by Pyrethrine or Pyrethroid Exposure In Civilian Air Crew Members"**
The information below has been provided by the speaker.

1.) Goals and objectives: Determination of the occupational stress factor responsible for the increase of illness in civilian air crew members observed since 1988. (20)

2.) Outline of talk/abstract: Application of the usual clinical criteria of cause and effect relationship furnished evidence that in most of these patients pyrethrin/pyrethroid exposure had triggered the onset of the disease and that the disease exhibited the clinical characteristics of pyrethroid intoxication. (39)

3.) Conclusion of what is to be learned: Insecticide exposure is an avoidable stress factor in civilian aviation. Therefore it is up to the decision of the persons responsible for insecticide application in aircraft and contract hotels to counteract the increase of illness. (35)

4.) References:

1. Muller-Mohnssen, H., Hahn, K. (1995): About a method of early recognition of neurotoxic diseases (Exemplified by Pyrethroid intoxication) in German, Gesundheitswesen 57, 214-222.

2. Muller-Mohnssen, H. (1999): Chronic sequelae and irreversible injuries following acute pyrethroid intoxication. Toxicological letters, 107, 161-175 (further references)

HEALTH EFFECTS CAUSED BY PYRETHRINE OR PYRETHROID EXPOSURE IN CIVILIAN AIR CREW MEMBERS

H. Müller-Mohnssen
Ludwig-Maximilians-University
Munich, Germany

Since 1988 it was observed in Germany that there has been a considerable increase in reports of illness with inability to work in crew members of long distance flights. Though the clinical pictures of illness exhibit resemblance for different patients, the symptoms that lead to inability to fly are different as follows: I). Mucocutaneous alterations: Conjunctivitis, alopecia, disfiguring weeping eczema of uncovered skin which flares up after in-flight spray missions, after entering the aircraft or hotel. ii). Cerebro-organic disorders: decrease of intellectual performance, increasing loss of memory, mixup of words, word finding difficulties, narcoleptic attacks alternating enhanced arousal and agitation. Abrupt nausea is observed followed by black out with collapse from seconds up to minutes (person falls down the stairs or, while car driving, wakes up when the car has come to standstill off the road), sudden attacks of clouding of consciousness with complete loss of orientation for 30 to 60 min (persons being at a familiar place suddenly does not know where they are). iii). Movement disturbances: walking "like on cotton", problems with correct estimation of distances (walking into door frames or missing stair steps). Clumsy while serving (when pouring the coffee, missing the cup and spattering the passenger). iiii).

Suspicion of autoimmune diseases: multiple sclerosis, collagenosis, thrombocytopenia. In 80% of the patients the fitness to practice another occupation was obtained one year after termination of flying activity. As a cause of the illness to the flight crew members the occupational stress was considered arising from: 1.) Enhanced psychophysical strain due to irregular way of life, lack of sleep, jet lag, temperature jump, loss of social contacts; 2). Cosmic radiation; 3). Insecticide exposure in aircraft and hotels. The diagram demonstrates the latency period, i.e. the time interval from the beginning of the employment on long distance flights till the complaints exceed the threshold of perception (n=65). Ordinate: year, Abscissa: number of person sorted according to start of flying. Each person is represented by two vertically arranged marks; circles: beginning of occupation on long distance aircraft, filled squares: onset of illness. If the unavoidable components of stress (1 and 2) would cause the illness, the latency should be almost constant, i.e., the upper dotted line should run parallel to the lower.

The results show, however, that the illness begins independently from the date of employment at a fixed date around 1992. Obviously there is no causal relation of illness with the stress components 1 and 2 but with a component firstly appearing between 1960 and 1992. The time span of 4 years between the introduction of pyrethroids for aircraft disinfection (1988) and outbreak of illness in the air crew members corresponds to the latency of 4,7 (±2,5) years of chronic pyrethrine/pyrethroid intoxication after indoor disinfection. The symptoms leading to inability to work depend on which of the three clinical phenotypes of pyrethroid-intoxication is

expressed in the particular patient. In the majority of air crew members the I-(immunmodulatory-) type was found.

Notes:

Abstract Information & Notes

Clement E. Furlong, Ph.D. Date of talk: Thursday, June 7, 3:30 p.m.
Department of Genetics Phone: 206/543-1193
University of Washington Fax: 206/543-0754
Box 357360 E-mail: clem@u.washington.edu
Seattle, WA 98195-7360

Medical School Attended: Ph.D. 1968, Biochemistry, University of California, Davis
Residency: n/a
Board Certifications: n/a
Current Faculty Appointments: Departments of Genetics and Medicine, Division of Medical Genetics
Current Job Description: Research in human biochemical genetics
Other Information:

Disclosure Statement: Bager Corporation (research support)

SPEECH TITLE: **"The Human Paraoxonase (PON1) Polymorphism: Role in Detoxication of Organophosphorus Insecticides"**
The information below has been provided by the speaker.

1). Goals and Objectives: The goals are be to explain the effects on organophosphorus sensitivity of several polymorphisms in the coding and promoter regions of the human paraoxonase (PON1) gene on human chromosome 7. Biochemical, genetic and animal model studies will be described.

2.) Outline of talk:

Outline of Lecture

History of the Human PON1 Polymorphism.
Cloning and Characterization of the Human and Rabbit PON1 cDNA's and the Human PON1 Gene.
Early Animal Model Studies
PON1 Knockout Mouse Model System Studies
The effect of the PON1 Polymorphism on Sensitivity to Diazoxon, Chlorpyrifos oxon and Paraoxon
The Bottom Line, Catalytic Efficiency of the PON1 Q192R Isoforms Determines *in vivo* Efficacy for Detoxications.

3.) Conclusion of what is to be learned: The major point to be learned from lecture #1 is that both the levels of expression of PON1 as well as the isoform expressed can be important in determining risk for exposure to specific organophosphorus (OP) compounds. The second point to be stressed is that the genetic variability in PON1 determines primarily sensitivity to the oxon forms of diazinon and chlorpyrifos and not the parent insecticides themselves (organophosphorothioates). The surprising point to be made is that contrary to what the name of

the enzyme would imply, high levels of the high activity PON1 probably do not provide much protection against exposure to paraoxon (or parathion). The usefulness of the mouse model system for understanding OP metabolism in humans will be discussed

4.) References:

1. Augustinsson KB, Barr M (1963) Age variation in plasma arylesterase activity in children. Clin. Chem. Acta 8:568-573

2. Brophy VH, Jarvik GP, Richter RJ, Rozek LS, Schellenberg GD, Furlong CE (2000) Analysis of paraoxonase (PON1) L55M status requires both genotype and phenotype (In Process Citation). Pharmacogenetics 10:453-60

3. Clendenning JB, Humbert R, Green ED, Wood C, Traver D, Furlong CE (1996) Structural organization of the human PON1 gene. Genomics 35:586-9

4. Costa LG, McDonald BE, Murphy SD, Omenn GS, Richter RJ, Motulsky AG, Furlong CE (1990) Serum paraoxonase and its influence on paraoxon and chlorpyrifos-oxon toxicity in rats. Toxicol Appl Pharmacol 103:66-76

5. Costa LG, Richter RJ, Murphy SD, Omenn GS, Motulsky AG, Furlong CE (1987) Species differences in plasma paraoxonase correlate with sensitivity to paraoxon toxicity. In: Costa LG, Galli CL, Murphy SD (eds) Toxicology of Pesticides: experimental, clinical and regulatory perspectives, Springer-Verlag, Heidelberg, pp 263-266

6. Davies HG, Richter RJ, Keifer M. Broomfield CA, Sowalla J. Furlong CE (1996) The effect of the human serum paraoxonase polymorphism is reversed with diazoxon, soman and sarin. Nat Genet 14:334-6

7. Ecobichon DJ, Stephens DS (1973) Perinatal development of human blood esterases. Clin Pharmacol Ther 14:41-7

8. Hassett C, Richter RJ, Humbert R, Chapline C, Crabb JW, Omiecinski CJ, Furlong CE (1991) Characterization of cDNA clones encoding rabbit and human serum paraoxonase: the mature protein retains its signal sequence. Biochemistry 30:10141-9

9. Li WF, Costa LG, Furlong CE (1993) Serum paraoxonase status: a major factor in determining resistance to organophosphates. J Toxicol Environ Health 40:337-46

10. Li WF, Costa LG, Richter RJ, Hagen T, Shih D, Tward A, Lusis AJ, et al (2000) Catalytic efficiency determines the in-vivo efficacy of PON1 for detoxifying organophosphorus compounds. Pharmacogenetics 10:767-779

11. Li WF, Furlong CE, Costa LG (1995) Paraoxonase protects against chlorpyrifos toxicity in mice. Toxicol Lett 76:219-26

12. Richter RJ, Furlong CE (1999) Determination of paraoxonase (PON1) status requires more than genotyping. Pharmacogenetics 9:745-53

13. Shih DM, Gu L, Xia, YR, Navab M, Li WF, Hama S, Castellani LW, et al (1998) Mice lacking serum paraoxonase are susceptible to organophosphate toxicity and atherosclerosis. Nature 394-284-7

Notes:
Abstract Information & Notes

William J. Rea, M.D. Date of talk: Thursday, June 7, 4:00 p.m

Environmental Health Center - Dallas Phone: 214/368-4132
8345 Walnut Hill Lane, Suite 220 Fax: 214/691-8432
Dallas, TX 75231 E-mail: wjr@ehcd.com

Medical School Attended: Ohio State University College of Medicine
Residency: University of Texas SW Medical School; Parkland Memorial Hospital; Baylor Medical Center, Veteran's Hospital; Children's Medical Center
Board Certifications: American Board of Surgery; American Board of Thoracic Surgery; American Board of Environmental Medicine
Current Faculty Appointments: n/a
Current Job Description: M.D./President - Environmental Health Center - Dallas

Disclosure Statement: None

SPEECH TITLE: **"Neurotoxicity-Central Nervous System"**

The information below has been provided by the speaker.

1.) Goals and objectives: Understand what Neurotoxicity is

2.) Outline of talk/abstract: A series of cases with this problem will be presented, diagnosis and treatment.

3.) Conclusion of what is to be learned: Neurotoxicity is common and should be diagnosed and can be treated.

4.) References: Chemical Sensitivity, Volumes I, II, III, IV

NEUROTOXICITY - CENTRAL NERVOUS SYSTEM
William J. Rea, M.D., F.A.C.S, F.A.A.E.M.
Yaqin Pan, M.D.

Abstract: One hundred patients (ages 12-75; F-61; M-39; average 45), who had chronic chemical exposure at their work place were studied. Exposure source varied but was mainly from solvents in the ambient air. The patients used no protective gear. The main complaints were neurological symptoms 49%; (headaches, migraines, short-term memory loss, inability to concentrate, loss of concentration, vertigo, light headedness), fibromyalgia, fatigue, arthritis, arthralgia, and musculoskeletal symptoms 51%. All patients were not able to stand on their toes with their eyes closed or able to walk a straight line with their eyes closed. Associated symptoms included

respiratory symptoms (shortness of breath, asthma, bronchitis) 35%; ENT (hearing loss, tinnitus, hoarseness, laryngeal edema, dysphasia) 35%; GI (irritable bowel syndrome, malabsorption, diarrhea) 18%; cardiovascular (vasculitis, chest pain, hypertension, cardiomyopathy, angioedema, mitral valve disease) 30%; endocrine (ovarian imbalance, PMS, hypothyroid, thyroiditis) 94%; eye (cataract, vision loss) 2%; skin (rash) 1%. 100% had chemical sensitivity including 6 implants (chin, breast, hip) 2 mercury toxicants; food sensitivity 89%; biological inhalant sensitivity 76%; EMF sensitivity 3%; candidiasis 4%.

Outline: Toxic solvent exposure was evident in 54 patients whose blood was measured with 1,1,1 trichloroethane 45%; benzene in 24%; trimethylbenzene 19%; xylene 9 %; dichloromethane 13%; chloroform 2%; trichloroethylene 7%; tetrachloroethylene 7%; ethylbenzene 2% and dichlorobenzene 2%. Another segment of solvents measured in these patients were the aliphatic hydrocarbon patients who had 3-methylpentane 89%, 2-methylpentane 85% and n-Hexane 75%. One (1) had n-pentane and 7% cyclopentane; 28 patients were measured for chlorinated pesticides. 100% had DDE, 46% trans nonachlor, 25% oxychlorodane, 14% heptachlor epoxide, 31% had hexachlorobenzene, 18% B-BHC, 14% Dieldrin, 98% of the patients were sensitive to food and 93% sensitive to mold, 74% to trees and grasses. 76 patients had intradermal skin tests for chemicals with ethanol 55%; formaldehyde 47%; cologne 96%; cigarette smoke 64%; metal testing performed on 16 patients show at 81% sensitivity to nickel and 69% to zinc sulfate. Double blind inhaled challenge revealed sensitivity to 1,1,1,trichloroethane 20%; formaldehyde 16.7%; toluene 16.9%; 16 organophosphate pesticide 16.7%; phenol 12.5%; chlorine 11.1%, ethanol 6.2%. T-lymphocytes 58% low, T_4 3.5% low and 21% high; T_8 lymphocytes 59% low and 15% high; B-lymphocytes 3% low, 4% high; CMI 3/7 33%; 4/7 27%; 5/7 20%; 6-7 9%; 2/7 13%.

Triple camera brain SPECT scan had 81 patients measured and 81 were positive for Neurotoxicity. Posturography was performed in 78 patients with 79% having abnormal sensory organization and 44 abnormal motor organization. Pupillography showed 84% abnormalities. Heart rate variability was performed on nine patients. All were abnormal. Specific utaneous thermography was performed in 20 patients with 95% rigid (low) response and 5 % hypersensitive response.

Conclusion: Treatment consisted of a massive avoidance of pollutants in air, food and water. Injection therapy, oral and intravenous nutrition, sauna therapy and autogenous lymphocytic factor. There was a significant 82% improvement rate.

Goals:

1. To understand the signs and symptoms of central neuropathy.
2. To be able to diagnose central neuropathy.
3. To be able to treat central neuropathy.

Abstract Information & Notes

Pierre Auger, M.D. Date of talk: Thursday, June 7, 4:30 p.m.

Clinique De Santé Au Travail et De Santé Environnementale Phone: 514/849-5201
3666 St-Urbain Fax: 514/731-0668
Montreal, Quebec H2X 2P4 Canada E-mail: pierrelauger@qc.aira.com

Medical School Attended: Laval University
Residency: Toronto-Quebec, Paris-London
Board Certifications: Occupational Medicine (Royal College)
Current Faculty Appointments: Adjoint Proffessor McGill
Current Job Description: Occupational Environmental Medicine Practice - Clinical Practice and Preventive Medicine.

Disclosure Statement: None

SPEECH TITLE: **"Chronic Toxic Encephalopathies Apparently Related to Exposure to Toxicogenic Fungi"**

The information below has been provided by the speaker.

Authors: Pierre L. Auger, MD, Pierrot Pépin IH, J. David Miller PhD., Manfreid Gareis D.V.M., PhD., Julien Doyon PhD., Rémi Bouchard MD, Marie-France Pinard PhD., Claude Mainville Ing.

1.) Goals and objectives: Description of four cases of toxic encephalopathies possibly caused by a prolonged exposure to toxin producing filamentous fungi in the workplace. We will also demonstrate that complete fungal evaluation with species identification and complementary cytotoxicity testing can be useful to identify possible causes of health effects of mold exposure.

2.) Outline of talk/abstract: Fungal toxins have irritant, immunosuppressive, neurotoxicological and carcinogenetic effects (Henry 1993). Recent report have suggested neurotoxic effects (Croft 1986, Johanning 1996). We report four cases who were diagnosed type 2b solvent type syndrome concurrent with a sensitive neuropathy in one occupant and novel asthma in a second.

First building is a harbour station built over a crawl space with the presence of Aspergillus Fumigatus, A. Versicolor, Penicillium aurantiogriseum. In this building a 42-year old director along with his 32-year old secretary have commenced to suffer from neuropsychological and systemic symptoms and novel asthma in the secretary. Improvement is noticed after 3 - 4 months after removal from exposure. Irreversible sequelae are described 15 months later: slight attention deficit in the director and asthma in the secretary.

Second building is a medical office in which severe flooding occurred during the Winter. In Spring, an environmental evaluation yielded the presence of Penicillium crustosum, P, brevicompactum, Fusarium incarnatum. Important neuropsychological deficits more or less similar in both are found. Improvement is noted upon removal from exposure. The pediatrician died of renal failure because of light chains disease. The female was left with irreversible brain damage.

3.) Conclusion of what is to be learned: The four workers presented with findings similar to solvent encephalopathies (Lindsberg 1995). In an occupational setting, an acute CNS disease with tremor was related to exposure to high levels of Aspergillus (Gordon 1993). The presence of toxicogenic molds in an environment suggests that when two or more occupants of dwelling suffer from neuropsychological symptoms, a detailed exposure analysis should be done with a neuropsychological evaluation on these same occupants.

4.) References:

1. Croft WA, Jarvis BJ, Yatawara CS. 1986 Airborne outbreak of trichothecene toxicosis Atmosph. Environm. 20: 549-558
2. Gordon KE, Masotiti RE, Waddell WR 1993 Tremorgenic encephalopthy: a role of mycotoxins in the production of CNS disease in human? Can. J. Neurol. Science 20: 237-37
3. Henry KM, Cole EC, 1993. A review of mycotoxicosis in indoor air J. Tooxicol Environm. Health 38: 183-93
4. Johanning E., Biagini R., Hull D. Morey P, Jarvis B. Landsbergis P 1996 Health and Immunology Study Following Exposure to Toxicogenic (Stachybotrys chartarum) in a water-damaged office Int. Arch. Occup. Environm. Health 68: 207-218
5. Lundberg I, Hogstedt C, Liden C, Nise CJ 1995. Organic Solvents and Related Compounds In Rosenstock I, Cullen MR editors Textbook of Clinical Occupational and Environmental Medicine Philadelphia Saunders WB 31: 766-84

Abstract Information & Notes

Deborah Baird, M.D. Date of talk: Thursday, June 7, 5:00 p.m.

5915 Volunteer Place Phone: 972/771-1106
Rockwall, TX 75037 Fax: n/a
E-mail: drbaird@msn.com

Medical School Attended: Handemann University of Medicine - Philadelphia
Residency: Psychiatry, Pediatrics, Fellowship in Developmental Disabilities
Board Certifications: American Board of Pediatrics
Current Faculty Appointments: None
Current Job Description: Retired from Private Pediatrics - Doing Locum Tenens - - - and having fun!
Other Information: Currently working on Implant Syndrome book with Dr. Rea.

Disclosure Statement: None

CASE STUDY: **"Seven Year Case Study of Acute Neurotoxicity"**

Notes:

FRIDAY, JUNE 9, 2001 FACULTY

C. Malcolm Beck
Garden-Ville, Inc
San Antonio, TX
210/651-6115

Iris R. Bell, M.D.
Program in Integrative Medicine
University of Arizona
College of Medicine
Tucson, AZ
520/626-3512

Professor Michel Bounias
University of Avignon, France
BioMathematic's and Toxicology Unit,
Chemin du Petit Bosquet
Saint-Christol D'Albion F-84390, France
011 33 490 750 888

Thomas E. Croley, Ph.D.
Ideal Health Clinic of Allen
Allen, TX
972/727-2800

J. Howard Garrett
The Natural Way/WBAP Radio
Dallas, TX

Richard Jaeckle, M.D.
Dallas, TX
214-696-0964

Rima E. Laibow, M.D.
Alexandria Institute of Natural and Integrative Medicine
Croton on Hudson, NY
914/827-9557

William J. Meggs, M.D., Ph.D.
E. Carolina University School of Medicine
Dept. of Emergency Medicine
Greenville, NC
252/816-2954

Eva Millqvist, M.D.
Allergy Centre, Department of Respiratory Medicine
Sahlgrenska University Hospital
Gothenburg, Sweden
011/46-31-3423635

Helmuth Muller-Mohnssen, Ph.D.
Ludwig-Maximilians Univ. Munich
Ismaning/Menich
Germany
011-49-89-969444

Eugene A. Shinn
U.S. Geological Survey
St. Petersburg, FL
727/803-8747 ext.3030

Jonathan V. Wright, M.D.
Tahoma Clinic
Kent, WA
206/631-8920

NINETEENTH ANNUAL INTERNATIONAL SYMPOSIUM ON MAN AND HIS ENVIRONMENT

SCHEDULE FOR FRIDAY, JUNE 8, 2001

8:15 ANNOUNCEMENTS/MODERATOR: William J. Meggs, M.D.

8:30 Iris R. Bell, M.D. "Neural Sensitization Model for Environmental Illness"
8:50 Q & A

9:00 C. Malcolm Beck "Re-mineralizing the Earth"
9:20 Q & A

9:30 Rima E. Laibow, M.D. "Neurotoxicity of Heavy Metals: The Ubiquitous Menace"
9:50 Q & A

10:00 BREAK WITH EXHIBITORS/MODERATOR: Bertie Griffiths, Ph.D.

10:30 Helmuth Muller-Mohnssen, Ph.D. "Pyrethroid Intoxication Showing Three Types of Clinical Expression"
10:50 Q & A
11:00 Eva Millqvist, M.D. "Sensitivity to Chemicals-A Theoretical Background to a Possible Explanation"
11:20 Q & A

11:30 Professor Michel Bounias, Ph.D. "Anticipatory Mental Imaging and 'NeuroBioFeedFarther' in Neurotoxicology"
11:50 Q & A

12:00n BUFFET LUNCH WITH EXHIBITORS

MODERATOR: Charlie Hinshaw Jr., M.D.
1:30 Jonathan V. Wright, M.D. "Modification of Steroid Metabolism with Foods, Minerals, Phytochemicals, and A Vitamin"
2:20 Q & A

2:30 William J. Meggs, M.D. "Controversies in Solvent Neurotoxicity"
2:50 Q & A

3:00 BREAK WITH EXHIBITORS

3:30 Richard Jaeckle, M.D."Neurotoxicity of Molds & Mycotoxins"
3:50 Q & A

4:00 Eugene Shinn, Ph.D."Transoceanic Soil Dust Transport and Medical Implications"
4:20 Q & A

4:30 Thomas E. Croley, Ph.D. "Balancing the Energy Flow of the Autonomic Nervous System"
4:50 Q & A

5:00 Howard Garrett, "BOP, the Basic Organic Program"
5:20 Q & A

5:30 ADJOURN

6:30-8:30 p.m. Tour of Environmental Health Center, American Environmental Health Foundation, and visit with William J. Rea M.D.

FRIDAY, JUNE 8, 2001

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes
Iris R. Bell, M.D. Date of talk: Friday, June 8, 8:30 a.m.

Program in Integrative Medicine Phone: 520/626-3512
University of Arizona Fax: 520/626-3518
College of Medicine E-mail: ibell@u.arizona.edu
P.O. Box 245153
Tucson, AZ 85724

Medical School Attended: Stanford
Residency: U. of California - San Francisco
Board Certifications: Psychiatry with added qualification in Geriatric Psychiatry
Current Faculty Appointments: Associate Professor of Psychiatry
Current Job Description: Director of Research, Program in Integrative Medicine, University of Arizona College of Medicine

Disclosure Statement: None

SPEECH TITLE: **"Neural Sensitization Model for Environmental Illness"**

The information below has been provided by the speaker.

1.) Goals and objectives: To review rationale for the neural sensitization model for environmental illness and laboratory evidence relevant to the model

2.) Outline of talk/abstract: Sensitization is a neurobiological process in which repeated intermittent responses to an exogenous substance or stressor initiate progressive increases in the size of the last response. This talk will review features of sensitization, its release to EI, and physiological data consistent with model.

3.) Conclusion of what is to be learned: Sensitization is a visible hypotheses for CNS/ANS dysfunction in EI for which there is accumulating supportive data.

4.) References:
Bell, IR et al. Neural sensitization model for multiple chemical sensitivity: overview of theory and empirical evidence. Toxicol Indust Health 1999; 15:295-304.

Fernandez M. et al EKG sensitization during chemical exposure in women with and without chemical sensitivity of unknown etiology. Toxicol Indust Health 1999; 15:305-312.

Abstract Information & Notes

C. Malcolm Beck Date of talk: Friday, June 8, 9 a.m.

Garden-Ville, Inc. Phone: 210/651-6115
7561 East Evans Rd. Fax: 210/651-9231
San Antonio, TX 78266 E-mail: n/a

Medical School Attended: n/a
Current Job Description: Full time researching and giving presentations all over U.S. on understanding nature. Average 80 talks or slide presentations a year to Universities, Colleges, Master Gardeners, Farmers, Ranchers, Home Owners Assn Etc.

Disclosure Statement: None

SPEECH TITLE: **"Re-mineralizing the Earth"**
The information below has been provided by the speaker.

1.) Goals and objectives: To show the needs of minerals to create fertile soil, to grow healthy plants.

2.) Outline of talk/abstract: To show with color slides the many test I did with plants in containers grown with and without rock minerals, and field test with & without minerals.

3.) Conclusion of what is to be learned: Using rock sand and dust can grow healthy plants with excellent flavor & nutrition and have immunity to pest and disease, even cold, heat - drought & flood.

4.) References: A life time of growing plants, vegetable, fruit, nuts. In the field and containers, using soil to soilless mixes to study the effects of with & without minerals and a constant study in field of agriculture, biology, botany & geology.

Re-Mineralizing the Earth

C. Malcolm Beck

All life on earth is given birth and sustained by a thin layer of soil that covers the dry lands of this planet. The quality of that thin soil layer determines the quantity and quality of the AIR we breathe, the WATER we drink and the FOOD we eat. If we let the quality of our topsoil degrade to any degree, the life it supports degrades along with it.

Fertile topsoil is made from life forms - living and decaying - in the presence of mineral rocks. The first life on earth was simple and small. Bacteria and fungi could live on rock surfaces, etch away at the rock, exude on it, and die and decompose rocks. These microscopic creatures created the first organic matter which filtered into the cracks and crevices of the rocks to form organic acids that dissolved more rock. The organic matter and rock minerals together became our first soil. The soil building process went on and on until larger, more complex plants could get a foothold. The death and decay of these higher plants built soil even faster.

Green plants collect the sun's energy and combine it with elements from the air and earth to make energy and food for still other forms of life. The living, dying and decaying of all life forms build with added interest to the soil's bank account. Once modern man intervenes, however, the balance changes. Overexposing the soil with bad tillage practices, plus the forced production of conventional agriculture using high analysis fertilizers and pesticides, seriously strain the health of the soil and the quality of the food it produces.

Modern chemical fertilizers contain no energy. The microbial life needed to process the chemicals into proper plant foods have to draw their energy from the soil organic matter and humus reserves. When the soil organic content runs low, the soluble fertilizer is not properly processed. The plants pick up the food in unbalanced ratios, causing them to become stressed, which in turn, invites pest insects and diseases to attack the plants, then toxic pesticides are used.

Meanwhile, some of the soluble fertilizer and pesticides are leaching away to pollute surface and ground water, which sickens more life. As humus runs low and soil life dies, the soil loses vital crumb structure and proper ability to absorb water. Normal rains quickly run off carrying humus and mineral-rich topsoil with it. Exposed subsoil is quickly dried by the sun. During dry spells wind erosion takes place.

As the soil loses mineral and humus, its strength, integrity and ability to produce life with strength and integrity are also lost. But if we have the will, soil can be rebuilt. Humans have intervened to hasten the loss of topsoil. We can intervene to create new topsoil. Nature has supplied us with plenty of mineral rock through deposits such as limestone and greensand and through tectonic exposures of basalt, lava and granite. By spreading the dust and sands of these rocks where needed and by recycling and rebuilding organic matter, we can rebuild the integrity and strength of the soil. Then the health and well-being of life on Mother Earth can be sustained.

Sources: A lifetime of growing plants, vegetables, fruit and nuts, in the field and containers. Using soil and soilless mixes to study the effects of with and without minerals. And a constant study in the fields of agriculture, biology, botany and geology.

Abstract Information & Notes

Rima E. Laibow, M.D. Date of talk: Friday, June 8, 9:30 a.m.

Alexandria Institute of Natural and Integrative Medicine Phone: 914/827-9557
10 Old Post Road South Fax: 914/827-3995
Croton on Hudson, NY 10520-2350 E-mail: laibow@juno.com

Medical School Attended: Albert Einstein College of Medicine
Residency: Lincoln Hospital and St. Luke's Hospital
Board Certifications: 1) Diplomate American Board of Forensic Examiners,
2) Diplomate American Board of Traumatic Stress Studies,
3) Diplomate Neurotherapy Certification Board.
Current Faculty Appointments: N/A
Current Job Description: Medical Director of the Alexandria Institute of Natural and Integrative Medicine
Other Information: 1) Senior Medical Editor Alternative Medicine: The Definitive Guide
2) Author: The Medical Applicatiions of NeuroBioFeedBack in Evans and Aberbanel, Introduction to Quantitatve EEG and NeuroBioFeedBack, Academic Press, 1999
3) President: NeuroTherapy Certification Board
4) Past President: Quantitative EEG Technicians Certification Board
5) Editorial Board, Journal of Neurotherapy.

Disclosure Statement: None

SPEECH TITLE: **"Neurotoxicity of Heavy Metals: The Ubiquitous Menace"**
The information below has been provided by the speaker.

1.) Goals and objectives: To identify common neurotoxins and their effects on the CNS and its regulation of organism wide functions.

2.) Outline of talk/abstract: Neurotoxic metals assault the organism from a variety of sources. Their effects are pervasive and their presence is ubiquitous. Pathogenic effects go well beyond neuropsychiatric dysfunction of mood and cognition, poisoning enzyme systems in the CNS while clinging tightly to their heavy metals represents one of the most significant challenges to robust health in our environment.

3.) Conclusion of what is to be learned: Practitioners will have an increased index of suspicion which will allow them to identify heavy metal neurotoxicity with great precision.

4.) References: (1) "Rea, WM J. Chemical Sensitivity Vol 1, Lewis Publishers, Boca Raton, 1992, Passim."
(2) "Rea, Wm J. Chemical Sensitivity Vol 3, Lewis Publishers, Boca Raton, 1995 Passim."
(3) "Rea, Wm J. Chemical Sensitivity Vol 4, Lewis Publishers, Boca Raton, 1995 Passim."
(4) "Bounais, Michel, Traite'de Toxicologie General: Du Nivgau Moleculaire A\L'echelle Planetaire, Springer, New York, 1999."

Abstract Information & Notes

Helmuth Muller-Mohnssen, Ph.D. Date of talk: Friday, June 8, 10:30 a.m.

Ludwig-Maximilians Univ. Munich Phone: 011-49-89-969444
Ismaning/Menich Fax: same
Germany E-mail: n/a

Medical School Attended:
Residency:
Board Certifications:
Current Faculty Appointments:
Current Job Description:
Other Information:
Vita
1928 born in Bremen, Germany
1951 state medical examination Marburg/Lahn
1952 MD, Marburg/Lahn, Germany
1968 Habilitation for Physiology Ludwig-Maximilians-University, Munich (LMU)
Educational background:
1952-1956, Institute of Pathology, University of Münster/Westfalen, Germany (Prof. Giese),
1952-1957 1956-1960 Physiological Institute, University of the Saarland (Prof. Stämptli).
1960-1993 Head of Department of Physiology in the federal center of environmental research GSF, Neuherberg/Munich.
1963 Retired
Working Fields
1. Development of collateral circulation induced by stenosing coronary sclerosis.
2. Hydrodynamic mechanisms involved in thrombogensis and atherogenesis,
3. Intravital-microscopical studies of the Node of Ranvier of isolated single motor nerve fiber during its electrophysiological function.
4. The stationary electrophysical basis of impulse-generation by the excitable nerve fiber membrane (Textbook: *"Physics of nervous excilation with an introduction to methodology of physiological research"*, Munich 1979).
5. Epistemological basis of research politics

Disclosure Statement: None

SPEECH TITLE: **"Pyrethroid Intoxication Showing Three Types of Clinical Expression"**
The information below has been provided by the speaker.

1.) Goals and objectives: Early detection of new anthropogen diseases with the aid of quantitative analysis of the patients complaints and of exchange of the quantified subjective data between the medical practitioners concerned with the patient (32).

2.) Outline of talk/abstract: In order to collect the minimum number of patients necessary to establish the diagnosis of a new anthropogen disease (35), cooperation of physicians is necessary. Physicians will base their tentative diagnosis mainly on information about complaints and their history obtained by the patient-doctor-dialogue. They will then be able to minimize the diagnostic expenditure by appropriate selection of technical diagnostic procedures to exclude/verify the tentative diagnosis. Because the oral doctor-patient-dialogue is lost for an exchange of data, it

was modeled and recorded by a computer-program which can be installed on a PC and allow to mail and recheck the results by round robins like results of laboratory tests (108).
3.) Conclusion of what is to be learned: The patients' affirmations about their complaints represent data which are reproducible like the results of sensory-physiological tests and referring reliably to the underlying organic disorders (25).

4.) References:
Muller-Mohnssen, H., Hahn, K. (1995): About a method of early recognition of neurotoxic diseases (Exemplified by Pyrethroid intoxication) in German, Gesundheitswesen 57, 214-222.

Muller-Mohnssen, H. (1999): Chronic sequelae and irreversible injuries following acute pyrethroid intoxication. Toxicological letters, 107, 161-175 (further references).

Pyrethroid Intoxication Showing Three Types of Clinical Expression

H. Müller-Mohnssen
Ludwig-Maximilians-University
Munich, Germany

This study investigates whether chronic pyrethroid intoxication including residues after acute pyrethroid intoxication can be diagnosed by showing a characteristic clinical pattern. Pyrethroid exposure was documented by anamnesis, laboratory findings, and a standardized 97 item complaint questionnaire in which a five point scale allowed each patient to rate his or her complaints. Zero denoted the preexposure state. Two pooled collectives comprising patients with clinical evidence of pyrethroid intoxication according to the clinical criterial of causal-relationship between exposure and complaints (table) were included into the study: a): the pyrethroid-collective (n = 145), whose members have been exposed in the interior of houses and b), the air-hostess-collective (n = 57) having been exposed in passenger planes. A control-collective sampled at random and being representative of the average German population completed the same questionnaire (n = 414, including n = 356 persons declaring themselves healthy). Average values of the collective complaint intensities MBM were for a) MBM = 1,53 and for b) MBM = 1,63 against MBM = 0,3 obtained for the control group of healthy persons (MBM = 4,0 is the maximum possible value). The average pyrethroid load expressed in mg/kg house dust or cabin dust was a) 587 and b) 690 respectively versus 0,22 found in the average German household. Complaints referring to psychometrically measurable organic mental disorders (decrement of intellectual performance, emotional condition and affectivity) show nearly the same intensities in all patients of collectives a) and b). Opposed to this the intensities of complaints concerning clinical disorders of the peripheral and autonomic nervous system and disorders of other organs differed in different patients, showing three types of clinical expression:

E-(encephalitis-) type. The disease starts abruptly after a symptomless interval of 4-7 days with pain and other perception disorders on account of an often electrophysiologically verifiable sensible polyneuropathy (PNP), combined with movement disturbances reminiscent to cerebellar ataxia. Depending on the severity of disease the symptoms, which are comparable to those of inflammatory neurological diseases, ascend from peripheral nerves-predominantly lower legs- to central regions of CNS and demand exclusion, for instance, of Polyneuritis, polyradiculitis, Guillain-Barré, multiple sclerosis.

P-(pseudo-dopaminodeficiency) type. This type is observed after long term low dose exposure but also after high dose exposure in persons producing no acute reaction because of higher resistency. The symptoms start imperceptible slow with hypermyotonia and movement disorders, reminiscent to M. Parkinson, correlated with pain syndrome of the locomotor-system as well as

with disorders of gastrointestinal, urogenital and cardiovascular regulation which are stronger than in the other clinical types.

I- (Immunomodulatory) type. This type impresses as worsening of preexistent allergical diathesis and lead to respiratory obstruction, mucocutaneous alterations and immune suppression with deficient defense against acute infection diseases, mainly of respiratory and urinary tract as well as against chronic opportunistic infection (Candida). More rarely are seen autoimmune diseases (thrombocytopenic purpura, hemolytic anemia, sclerodermia, multiple sclerosis). In most cases health condition improves after stopping of exposure.

Table (Pyrethroid Intoxication showing three types of clinical expression)

A causal relationship between pyrethroid exposure and the exposed subject's complaints was assumed if the following criteria were met by the patient:

1. Concomitant diseases were excluded by the physicians in charge and the influence of insecticides other than pyrethroids is negligible.

2. Other persons simultaneously exposed within the same environment fall ill exhibiting similar symptoms

3. Complaints, which firstly appeared after begin of exposure mitigate after interruption of exposure and aggravate after re-exposure.

4. In case of acute intoxication: a high-concentration exposure due to self-application of insecticides (spraying or brushing) can be documented - in case of chronic intoxication: Pyrethroids are detected in the patient's environment and his or her body fluids or tissues: more than 10 mg Pyrethroid/kg house dust, more than 1.5 g pyrethroid metabolite/ 1 urine and more than 0,1 g Pyrethroid/kg hair (values for permethrine). Detection of a specific antigen-antibody reaction by means of Epicutan- and intracutantest. Detection of specific sensibilization by lymphocyte transformation test in the sense of a type-IV-immune response against pyrethroids or pyrethrins.

5. Duration of latency period and severity of the health effects of the person in question correspond to those of a multitude of patients who have been exposed to a similar dose. In case of acute intoxication the latency period between the begin of a documented exposure and the onset of illness was measured, in case of chronic intoxication the time interval between the end of a permanent exposure and the decrease of complaints.

6. The clinical picture resembles that of a multitude of patients who have been exposed to the same chemical substance.

Abstract Information & Notes

Eva Millqvist, M.D. Date of talk: Friday, June 8, 11:00 a. m.
Allergy Centre, Department of Respiratory Medicine Phone: 011/46-31-3423635
Sahlgrenska University Hospital Fax: 011/46-31-417824
Gothenburg, S-413 45, Sweden E-mail: eva.millqvist@medfak.gu.se

Medical School Attended: Karolinska Institute, Stockholm, Sweden
Residency: Gothenburg

Board Certifications: M.D., Ph.D.
Current Faculty Appointments:
Current Job Description: Research in Asthma and Asthma like Conditions. Working with patients sensitive to strong scents and chemicals.
Other Information: Working with developing a new method to test chemical sensitivity.

Disclosure Statement: None

SPEECH TITLE: **"Sensitivity to Chemicals. A Theoretical Background to a Possible Explanation"**

The information below has been provided by the speaker.

1.) Goals and objectives: To present a theoretical background to what may happen in patients with airway sensitivity to chemical irritants like strong scents, smoke and car exhausts.

2.) Outline of talk/abstract: The pathophysiology of airway chemical sensitivity, i.e. asthma-and allergy-like symptoms induced by chemical irritants, may be related to increased sensitivity of free, overreactive nerve endings in the respiratory mucosa because such patients cough more after capsaicin provocation than do healthy subjects and patients with asthma.

3.) Conclusion of what is to be learned: In patients who experience airway symptoms induced by very low levels of chemical irritants, it seems likely that it is not the smell that causes problems but a sensory hyperreactivity of the common chemical sense induced via the trigeminal nerve.

4.) References:

1. Milliqvist E, Bende M, Löwhagen O. Sensory hyperreactivity - a possible mechanism underlying cough and asthma-like symptom. Allergy 1998;53:1208-12.
2. Milliqvist E, Bende M, Löwhagen O. Quality of life and capsaicin sensitivity in patients with sensory airway hyperreactivity. Allergy 2000;55:540-545.
3. Millqvist E. Cough provocation with capsaicin is an objective way to test sensory hyperreactivity in patients with asthma-like symptoms. Allergy 2000;55:546-550.

Abstract Information & Notes

Professor Michel Bounias Date of Talk: Friday, June 8, 11:30 a.m.

University of Avignon, France Phone: 011 33 490 750 888
BioMathematic's and Toxicology Unit, Fax: same
Chemin du Petit Bosquet E-mail: n/a
Saint-Christol D'Albion F-84390, France

Medical School Attended: University of Lyon: INSA School of Engineers; Faculty of Sciences 3rd cycle
Residency: Same as above
Board Certifications: Docteur d'Etates Sciences/DEA (3rd cycle) in Biometrics/Engineer in Biochemistry
Current Faculty Appointments: Professor, Director of Research (Nat'l. Officer), Ministerial Expert

Current Job Description: 1.) Theoretical research: Mathematical foundations of existence of physical universe and biological systems, up to brain and conscious perception functions.
2.) Toxicology (from molecular to Planetary levels): Applications of Mathematical theory.

Other Information: About 400 scientific papers published, 10 books authored
1.) Elaboration of a new deontology for the management of Planet Earth, foundations of a chart of objective rights of all living organisms.
2.) Editorial Activity (CoEditor and chief advisor of Scientific Journals)
3.) Musicologist and Composer
4.) Symphonies, lieders for soprano, etc.)

Disclosure Statement: None

SPEECH TITLE: **"Anticipatory Mental Imaging and ' NeuroBioFeedFarther' in Neurotoxicology"**

The information below has been provided by the speaker.

1.) Goals and objectives: Identification of theoretical foundations of NeuroBioFeedback and neurotoxicological implications, from the mathematics of conscious perceptions.

2.) Outline of talk/abstract: Conditions for existence of a physical space also involve the foundations of conscious perception, up to mental imaging, which exhibits anticipatory properties. Mental images are arguably constructed from both outer and inner information and anticipatory processes underly homeostatic control. Health can thus be restored from altered states by directed feeding of the brain with corrected mental images standing for farther targets of improved status.

3.) Conclusion of what is to be learned: The mechanisms and importance of developmental neurotoxicology and of chemical hypersensitivity, via mental imaging of the unconscious type are emphasized.

4.) References:

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7. Anticipatory Mental Imaging and "NeuroBioFeedFarther" in Neurotoxicology

M. Bounias
Research Director and Professor, University of Avignon and INRA-DSPE,
BioMathematics & Toxicology Unit
Chemin du Petit Bosquet, F-84390
Saint-Christol d' Albion, France

A. Stubblebine and R.E. Laibow
The Alexandria Institute of Natural and Integrative Medicine
Croton-on-Hudson, New York

Summary: A mathematical proof of existence of a physical universe justifies existence of conscious perception, up to mental imaging which exhibits anticipatory properties. Thus directed restoration of correct relationships between homeostasy-related mental images and the control of the organism is possible: this founds NeuroBioFeedback. A set of neurotoxicological targets is derived from the model.

Introduction and preliminaries. Brain is a structure produced by Universe and released in our observable spacetime. Thus neurotoxicity is not independent from the foundations of the existence of Universe, which has been proved to be justified by mappings of topologically closed abstract mathematical subspaces[6-7]. A necessary and sufficient condition for existence of the latter is the existence of the empty set, as a primary axiom[8]. The same conditions also provide justifications for existence of conscious perception[6-9]: neuronal chaining Cauchy-like sequences lead to sets of fixed points of the Banach type, associated with input of information from outside, via perceptive signals representing mathematical paths (Jordan-Veblen's theorem). These sets of fixed points are stable parts standing for mental images. Then the need for self-reference associated with outside perception is fulfilled by the Brouwer's theorem stating that in a closed space all continued functions own a fixed point. Proofs were further given that the mathematical space underlying the functionally of the brain system fulfills the required conditions[10-12] and that the mental imaging also owns fractal properties, and even more importantly that it is an anticipatory process[13].

Brain and anticipatory mental imaging. In anticipatory processes the state of a system at time (t) depends on states at $(t+i)_{i1}$ (i.e. mathematical incursivity or hyperincursivity) [22].Since the brain is able to construct mental images of future situations, it can adjust a succession of decisions and actions to the best possible fitting of the living organism to a given goal. The intersection of the respective sets of Brouwers' type and Banach type fixed points predicts that a kind of mental images is produced by molecular information from inside the organism through neuronal configurations likely to be unconscious at least from the autonomous nervous system. Mental images reflect bursts of outside perceptions, abstracts thoughts, subjective images, but also the state of functioning and metabolic control of homeostasy of the organism through molecular feedback loops from inside the body to brain receptors.

Neuronal foundations of NeuroBioFeedback. Experimental data support the above Proposition. EEG changes reflecting neuronal configurations in the cortex are associated with the stimulation of receptors involved in autonomous metabolic feedback and control[1,2,3,15,16,19,20,23,25,26,29,31,32,34] besides EEG responses elicited by conscious feelings[21,24,30,33] and HPA responses to stress[18]. Sensory perception has thus logically found critical in developmental neurotoxicity[3,4] while developmental toxicity imprints delayed responses[28]. Mentally induced brain changes[5] must not be confused with true chemical sensitivity[27]. Neurotoxicity must thus have to be detected and treated at many target sites of the brain-body reciprocal interactions.

Discussion and conclusions. A disease may fix mental images of altered states; then homeostatic processes turn to wrong control operations. Corrected mental images with anticipatory characteristics must thus be constructed and substituted for the wrong ones in order to turn back the brain functioning progressively to anticipatively corrected goals. This can be farther achieved through feedback loops based on the EEG activity records targeted on improvement of frequencies spectra associated with rehabilitation of patients[14]. Thus: the clinical effects of NeuroBioFeedback treatments find their justification through a NeuroBioFeedFarther' system of anticipatory mental imaging.

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Abstract Information & Notes

Jonathan V. Wright, M.D. Date of talk: Friday, June 8, 1:30 p.m.

Tahoma Clinic Phone: 206/631-8920
515 W. Harrison St., Ste 200 Fax: 253/850-5639
Kent, WA 98032 E-mail: drjvwright@yahoo.com

Medical School Attended: University of Michigan
Residency: Group Health Hospital Family Practice 1969-71
Board Certifications: N/A
Current Faculty Appointments: N/A
Current Job Description: Medical Director Tahoma Clinic

Disclosure Statement: Meridian Valley Laboratories

SPEECH TITLE: **Modification of Steroid Metabolism with Foods, Minerals, Phytochemicals, and A Vitamin**
The information below has been provided by the speaker.

1.) Goals and objectives:

2.) Outline of talk/abstract: Talk will review results of use of foods, cobalt, iodine, Di-Indolylethane & Indole-3-Carbinol, Chrysin, & Vitamin A in modulation of steroid metabolism

3.) Conclusion of what is to be learned: Minerals, botanicals, & vitamins can be used to modify steroid metabolism

4.) References:

"Modification of Steroid Metabolism with Foods, Minerals, Phytochemicals and A Vitamin"

Jonathan V. Wright, M.D.

ABSTRACT

Options available to practitioners for safe, effective modulation of steroid metabolism are increasing. **Brassica vegetables, soy, and flaxseed** have all been shown to favorably influence the 2/16a hydroxyestrogen ratio (a "risk factor" for estrogen related cancers. Supplemental **indole-3-carbinol** and **di-indolylmethane** do the same. **Vitamin A** increases low levels of 17-B estradiol. **Black cohosh** can also stimulate increased estrogen production, while **Vitex** may favorably increase progesterone levels. **Chrysin** in many cases inhibits "abnormal" aromatization of androgens to estrogens. **Boron** raises circulating levels of estrogens and progesterone in postmenopausal women, and testosterone levels in men in their 50s and upwards. respectively. Examples of the little-known observation that **iodine** increases the complete metabolization of estrogens to estriol will be shown.

The novel observation of estrogen hypermetabolization and hyperexcretion, clinical manifestations of this problem, and correction of both symptomatic and laboratory manifestations by **cobalt** will be described, with examples.

Abstract Information & Notes

William J. Meggs, M.D., Ph.D. Date of talk: Friday, June 8, 2:30 p. m.

E. Carolina University School of Medicine Phone: 252/816-2954
Dept. of Emergency Medicine Fax: 252/816-3589
Brody Bldg., Room 4W54 E-mail: meggs@brody.med.ecu.edu
Greenville, NC 27858

Medical School Attended: University of Miami
Residency: Internal Medicine - University of Rochester; Allergy & Immunology - N.I.H.; Medial Toxicology - New York University
Board Certifications: Internal Medicine, Emerg. Med, Medical Toxicology, Allergy & Immunology
Current Faculty Appointments: Brody Medical School at East Carolina Univ.
Current Job Description: Chief, Division of Toxicology; Professor & Vice Chair for Clinical Affairs, Dept. of Emergency Medicine

Disclosure Statement: None

SPEECH TITLE: **"Controversies in Solvent Neurotoxicity"**
The information below has been provided by the speaker.

1.) Goals and objectives: See slides

2.) Outline of talk/abstract: See slides

3.) Conclusion of what is to be learned: See slides

4.) References: See slides

Abstract Information & Notes

Richard Jaeckle, M.D. Date of talk: Friday, June 8, 3:30 p.m.

8220 Walnut Hill Lane, Suite 404 Phone: 214/696-0964
Dallas, TX 75231 Fax: 214/696-1094
E-mail: rgjmd@airmail.net

Medical School Attended: University of Texas Southwestern Medical School
Residency: St. Louis University Hospital - Psychiatry, Fellowship - Washington University Child Guidance Clinic - Child/Adolescent/Psychiatry
Board Certifications: 1) ABPN - Psychiatry
2) ABPN - Child/Adolescent Psychiatry
3) AAEM - Environmental Medicine
Current Job Description: Private Practice

Disclosure Statement: None

SPEECH TITLE: **"Neurotoxicity of Molds & Mycotoxins"**
The information below has been provided by the speaker.

1.) Goals and objectives: To familiarize the audience with the non-allergic / Non-Immunologic effects of molds / mediated by their toxins

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned: Biochemical effects of mold metabolites/toxins may have profound effects on the CNS as well as other organ systems.

4.) References: Murphy JW, Friedman K, Bendinelli M., Fungal Infections and Immune Responses, Plenum Press, New York & London, 1993.

NEUROTOXICITY OF MOLDS & MYCOTOXINS

Richard G. Jaeckle, MD
Dallas, TX

Any living organism takes in nourishment and discharges wastes. Mycotoxins are the wastes or products of the metabolic processes of molds, which are toxic when consumed. Molds and their toxins are present on the product in the field, but multiply during transportation and storage if thorough drying is not accomplished. Conditions favorable for growth are moisture and temperature. Physical damage to the product accelerates the process, but drought stressed plants are more vulnerable. In 1960, worldwide attention was focused on them when an unprecedented

catastrophe occurred. An entire farm of 100,000 turkeys and 20,000 other poults were killed by contaminated peanut meal from Brazil. The culprit was the most poisonous toxin produced by any mold, aflatoxin.

The major mycotoxins include aflatoxin, DON, T-2, zearalenone, fumonisin, and ochratoxins. Agricultural products prone to the development of toxins include peanuts, cottonseed, soy, corn and other grains. For many years veterinarians have observed mycotoxin effects on animals, including allergies, reproductive failure, unthriftiness, loss of appetite, feed refusal, suppression of immune system, decreased feed efficiency, and death. Their effects on organ systems include neuropathy and leucoencephalomalacia, hemorrhage, pulmonary failure, myocardial infarction, liver failure, kidney failure, epidermal and mucosal necrolysis, immunosuppression and bone marrow destruction, and cancer. Biochemical effects include complement lysis, binding of S-H group proteins, cross-linking of proteins, inhibition of DNA and protein synthesis, up or down regulation of macrophages, decreased respiratory function of mitochondria via P-450, and inhibition of sphingolipid synthesis.

Mycotoxicoses in man include St. Anthony's fire, cardiac beriberi, alimentary toxic aleukia, farmer's lung, pulmonary, gastrointestinal and immune insult, and cancer. Cancer in humans has been linked to aflatoxin, sterigmatocystin, zearalenone, patulin, ochratoxin, and fumonisin. Another mycotoxin source for humans are mushrooms, The following represent the major types of reactions. 1) The most common mycotoxin effect is irritation to the gastrointestinal tract, namely nausea, vomiting, cramps, and diarrhea from certain mushrooms. 2) Certain species of mushrooms produce muscarinic effects, usually within 15-30 minutes of ingestion. They may include excessive salivation, sweating tears, lactation (in pregnant women), severe vomiting and diarrhea, visual disturbances, irregular pulse, decreased blood pressure, and difficulty breathing. Victims normally recover within 24 hours, but severe cases may result in death due to respiratory failure. Atropine is the specific antidote. 3) Atropine is NOT indicated for poisoning due to muscimol, ibotenic acid, and other isoxazole derivatives. These produce symptoms in 30 minutes to 2 hours, and last for several hours. Nausea and vomiting are quite common, but the principal effect is on the CNS: confusion, visual distortion, a feeling of greater strength, delusions, and convulsions. Drowsiness is common, and often the victim falls asleep and cannot be aroused. Treatment is supportive, and recovery is spontaneous.

4)Poisoning from amanitin is extremely serious. Fatality from the amatoxins is 50%. It is so dangerous because symptoms are delayed 6 to 24 hours after ingestion, by which time the toxins are completely absorbed and bound. RNA syntheses is inhibited within *every* individual cell. The first stage is a latency period of 6 to 24 hors during which toxins are actively destroying the victim's liver and kidneys. The second stage of about 24 hours is characterized by violent vomiting, bloody diarrhea and severe abdominal cramps. The third period of 24 hours is apparent recovery, during which time the patient is usually discharged. The fourth stage is a relapse due to liver and kidney failure, leading to subsequent relapses or death. Treatment is supportive.

5) Gyromitrin may produce only headache and gastrointestinal symptoms, or proceed to liver, kidney, and RBC damage possibly resulting in death. A hydrolysis product is used by NASA as a rocket fuel. 6) Poisoning from Orellanine is extremely serious, since it is delayed as long from 36 hours to three weeks. Symptoms include nausea, vomiting, lethargy, anorexia, frequent urination, burning thirst, headache, sensations of coldness and shivering (generally without fever), progressive kidney failure. 7) Psilocybin has been used in primitive religions and medicine for its hallucinogenic effects, and rediscovered in the 1930's. Onset of symptoms occurs within an hour and may last 4-6 hours. Effects are primarily psychological and perceptual, including heightened color perception, religious ecstasy or anxiety, and sometimes hallucinations or delusions

mediated through the serotonergic systems. 8) Paxiluss involutus is a common cause of gastrointestinal symptoms in Eastern Europe, but may also produce a chronic hemolytic anemia.

Shaw has recently reported small molecule mycotoxins in the urine of a large series of autistic children. These metabolites from yeast, as well as products from Clostridia, came from intestinal contents. Some early cases even returned to normal after antifungal medication and/or Lactobacillus acidophilus was used. Their lowered fungal resistance was due to repeated courses of antibiotics for multiple ear infections. Tartaric acid, which is highly toxic and not produced in humans, was found in high levels in some children. It blocks the Krebs cycle at the malic acid site with effects of decreased energy and impaired gluconeogenesis. Arabinose, a pentose, was also identified and blocks receptor sites for B-6, lipoic acid, and biotin. Other effects of arabinose include pentosidine crosslinking, which decreases solubility and causes neurofibrillary tangles, decreases enzyme activity, decreases flexibility of collagen and muscles, and stimulates autoimmune disease.

In all likelihood, the mycotoxins are also responsible for chemical sensitivity. In 1977 Truss hypothesized that chemical sensitivity was caused by chronic infection with Candida due to the accumulation of the metabolite acetaldehyde. This chemical diffuses body wide, forming a Schiff base with any amine (displacing P5P) or any sulfhydryl group, also decreasing NAD. Randolph also felt that symptoms of chemical sensitivity were aggravated by chronic Candidiasis in women.

Cases

#1 AK, 7.5 lbs. at birth, weighed 12 at one year and was diagnosed FTT (failure to thrive). A progressive downhill course started at 8 yrs. And she was incapacitated by seizure-like rages at 12 yrs. Several physicians found nothing wrong and sent her for psychiatric evaluation. She was extensively studied and then treated with vaccines, Nystatin, and nutrients. Finally, the addition of Tricophyton and Epidermophyton at a dose based on DST produced a dramatic improvement, including a 10x-40x elevation of platelet neurotransmitter levels to normal. A dose based on the ISR produced the seizure/rages several times. We were never able to draw blood during the attacks because of her thrashing about. However, her mother used a glucometer during one episode to verify blood sugars at 40, descending into the thirties and twenties. (Shaw has reported severe hypoglycemia in an autistic child reversed with Nystatin.) We speculate that the proper dose of Tricophyton and Epidermophyton based on the DSR inhibited yeast activity and production of mycotoxins, which then permitted enzyme systems to return to normal function.

#2 WJ, 57 y/o female, had chemical sensitivities, severe allergies, and intestinal problems, which kept her captive to her environmental trailer for over 11 years. Suddenly she developed hemorrhagic areas on her skin body wide. Previously hospitalized for mania in 1981, she again developed mania and was hospitalized on the Psychiatry unit. Lithium, Elavil and tranquilizers were started along with Nystatin. Within 5 days, her skin lesions were fading as her mental status improved. MET vaccines were added later. She has remained normal for several years and is off medication, but continues her MET vaccine.

Since yeasts are part of the normal flora of the intestinal tract, it is imperative that they be considered in any case of food allergy. Food allergic reactions impair digestion and provide more opportunity for fermentation by yeasts and bacterial flora. This increases dramatically the toxicity of the intestinal contents. It is my opinion that food allergies and abnormal flora with yeast overgrowth always coexist. This suggests that a vigorous anti-yeast program should be used with every food allergic patient. The testing and use of the MET vaccine has been discussed previously.

Abstract Information & Notes

Eugene A. Shinn Date of talk: Friday, June 8, 4:00 p.m.
U.S. Geological Survey Phone: 727/803-8747 ext.3030
600 4th Street South Fax: 727/803-2032
St. Petersburg, FL 33701 E-mail: eshinn@usgs.gov

Medical School Attended: University of Miami, Florida - honorary Ph.D., University of South Florida, 1998
Residency: N/A
Board Certifications: N/A
Current Faculty Appointments: Adjunct professor University of South Florida and University of Miami
Current Job Description: Research Geologist
Other Information: Through geology/earth surface processes, I developed a theory that pathogens transported in African dust to the Caribbean caused the ongoing demise of coral reefs - this led to human health effects, especially asthma in the Caribbean.

Disclosure Statement: None

SPEECH TITLE: **"Transoceanic Soil Dust Transport and Medical Implications"**
The information below has been provided by the speaker.

1.) Goals and objectives: To make medical profession aware of a potentially increasing threat to human health

2.) Outline of talk/abstract: see attachment

3.) Conclusion of what is to be learned: That there is a threat - see attached abstract

4.) References:

Transoceanic Soil Dust Transport And Medical Implications
E.A. Shinn, Douglas Seba, Dale Griffin, Christina Kellogg, Charles W. Holmes

Increasing transoceanic dust flux may herald a potential large-scale threat to public health. Dust transported from Africa causes distress to chemically sensitive and medically compromised individuals and may affect others in the United States and Caribbean as transoceanic dust flux increases. Indigenous dust in the western US is known to transport the valley fever pathogen *Coccidioides immitis* but effects of African dust, which transports bacteria, viruses, and spores of fungi including numerous species of *Aspergillus,* have not been investigated until recently.

Estimates of annual African dust flux to the Amazon basin, Caribbean, and southeast US ranges into the hundreds of millions of tons. Flux of African dust to the Caribbean and US has increased dramatically since 1970 because of the ongoing drought in North Africa. The drought is a result of fluctuations in the North Atlantic Oscillation (NAO). Long-term dust monitoring in Barbados and Miami shows a correlation with NAO. The incidence of asthma on Barbados and nearby Trinidad, documented by the Caribbean Allergy and Respiratory Association (CARA), is among the highest in the world and has increased 17-fold since 1973. Mercury and aluminum, two components in African dust, have been detected at elevated levels in human blood on the island

of Puerto Rico. Arsenic has recently been implicated in endocrine disruption and has been linked to lung cancer.

Because soil dusts can serve as carriers for 1) pesticides, 2) heavy metals (mercury and arsenic), 3) naturally occurring radioactive isotopes (Be-7 and Pb-210), 4) viable bacteria, fungi, viruses, 5) organic debris, and 6) insects, further evaluation, monitoring and prediction of transatlantic soil dust events may be warranted.

Abstract Information & Notes
Thomas E. Croley, Ph.D. Date of talk: Friday, June 8, 2001 4:30 p.m.

Ideal Health Clinic of Allen Phone: 972/727-2800
204 N. Greenville Ave., Suite 145 Fax: n/a
Allen, TX 75002

Medical School Attended: Ph.D. in Human Anatomy from Baylor College of Dentistry
Residency: n/a
Board Certifications: n/a
Current Faculty Appointments: n/a
Current Job Description: Cold laser therapy within the Ideal Health Clinic of Allen (Texas)

Disclosure Statement: None

SPEECH TITLE: **"Balancing the Energy Flow of the Autonomic Nervous System in the Environmentally Challenged Patient"**

The information below has been provided by the speaker.

1.) Goals and objectives: Understand the functional adjustments being attained.

2.) Outline of talk/abstract: (1) Identify summative measurement pts. for sympathetic treatment. (2) Present relation of meridian energy flow and chakral energy. (3) Identify patient's ability to cope with autigenic presentation better following ANS balancing.

3.) Conclusion of what is to be learned: How this modality can be used with a given protocol to accomplish the desired goal of a balance of the components of the ANS.

4.) References: (See abstract)

BALANCING THE ENERGY FLOW OF THE AUTONOMIC NERVOUS SYSTEM IN THE ENVIRONMENTALLY-CHALLENGED PATIENT

Thomas E. Croley, Ph.D.
Ideal Health Clinic of Allen
204 N. Greenville Ave., Suite 145
Allen, TX 75002
972/727-2800

The environmentally challenged patients present some common pathologies within their bodily functions. One of the most common pathologies is an imbalance of the two components of the Autonomic Nervous System (ANS). In these patients, the "protective" sympathetic system dominates the parasympathetic system and is overly stimulated. Cold Laser Therapy (CLT) has been successfully used, to date, in bringing back a normal balance of these two components resulting in a normal functioning of the ANS helping these patients as they encounter the environmental challenges.[1] Dr. Reinhold Voll[2] has demonstrated the gall bladder 20 acupoint to be the Summative Measurement Point (SMP) for the sympathetic nervous system (SNS) which translates to be the most active acupoint representing the total activity of the SNS. Therefore, if the SNS is overly active in these environmental challenged patients, gall bladder 20 represents the site where one should depress the activity (in this case, using laser acupuncture). Additional sites of sympathetic energy accumulation when over stimulated are urinary bladder 14 and 25.

Following this sympathetic point nerve depression, the Vagus nerve should be stimulated using Kidney-19, 20 and 21 acupoints in order to stimulate the Parasympathetic Nervous System (PSP).[3]

Following treatment of these two components of the ANS, balancing of the seven chakral energies helps to restore proper energy to the functioning ANS.[4]

In a large majority of the patients treated in this manner, a reestablishment of a balance between the SNS and PNS components has been exhibited by the bodily functions controlled by the ANS, especially the parasympathetic which has been previously dominated by the sympathetic. For example, patients having difficulty sleeping at night have returned to more normal sleep patterns. Digestive problems in some patients have normalized. Blood pressure measurements taken frequently on these patients in the clinic have shown a return to normal levels in nearly all of those treated with the ANS balance technique. Urinary inconsistencies have also been adjusted in some of the patients.

Reestablishing the energy balance to the ANS components and the chakras assures a proper flow of energy along the meridians of the posterior body (sympathetic, Yang) to the anterior side of the body (parasympathetic, Yin). In accomplishing this normal flow of energy (Qi), the proper energy flow is reestablished in the head/face meridians. This has cleared double vision and/or tinnitus in many of the patients exhibiting these problems. Likewise, "brain clouding" exhibited in many of these patients is cleared.

Balancing of the ANS along with the other treatment techniques which work toward removing the chemical incident and/or immunizing the patient to particular antigens has allowed these patients to reach their goal of returning to normal lifestyle. Our protocols of treatment are based on sound principles of anatomical and physiological activities within the human body. For example, if a patient is challenged by aromatic chemicals such as formaldehyde, phenols or petroleum organics, the olfactory nerve is overly stimulated which results in an abnormal stimulation of the entorhinal cortex and the limbic system. The limbic system includes the hippocampus, the amygdaloid nuclear complex, the uncus (rostral part of the parahippocampal gyrus), the anterior part of the cingulate gyrus, the mammillary bodies, portions of the anterior hypothalamus and adjacent parolfactory area, the anterior nuclear mass and the doreomedial nucleus of the thalamus, septum pellucidum, habenular nuclear complex, and orbital gyri.[5,6,7,8,9]

The cold laser stimulation of peripheral nerves results in a stimulation of the cytochrome oxidase enzymes within mitochondria which results in an increased ATP production.[10] An increased availability of ATP to a system that is highly energy dependent, increases axoplasmic flow rates

within the neural circulation leading to changes both in proximal and distal nerves to the incident irradiation.[11] Therefore, the cold laser (low level laser) stimulation is capable of interfering with the over stimulation of the limbic system by reducing the excess stimulation of the sympathetic nervous system via the hypothalamus and the amygdala establishing the normal physiological stimulus and feedback to the limbic system. Experimental evidence has shown deficiencies in neuronal impulses from the hippocampus resulting in short-term memory losses (brain clouding) and an imbalance of the sympathetic nervous system. As well, several physiological changes occur in the normal, rhythmic stages of sleep. As a result, these changes exert abnormal effects on the body temperature and the immune system. Many of these effects are a result of an interruption of the normal neural stimulation to the components of the limbic system from the reticular activating system and the locus ceuleus of the brain stem.

Use of the cold laser on the acupoints of the components of the autonomic nervous system in order to balance the energy flow within this body system has proven to be an effective treatment in concert with the other treatment modalities of the environmentally-challenged patient. Other protocols using cold laser therapy are currently being explored.

References

1. T.E. Croley, 18th Annual International Symposium on Man and His Environment, June 8-11, 2000, Dallas, Texas.
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3. Julian N. Kenyon, Modern Techniques of Acupuncture, Vol. 1, p. 131. Thorsons Publishers, Inc., Wellingborough, Northhamptonshire, 1983.
4. Richard Gerber, Vibrational Medicine Bear & Company, Santa Fe, New Mexico, 1988.
5. A.C. Guyton, Textbook of Medical Physiology, 6th Ed., W.B. Saunders & Co., Philadelphia, 1981.
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10. Tiina Karu, The Science of Low-Power Laser Therapy, Gordon and Breach Science Publishers, Chp. 5, Responses of Neurons and Lymphocytes to Direct Irradiation, Russia, 1998.
11. Philip Gabel, Axoplasmic Flow May Cause LLLT's Latent and Systemic Effects Presented at the 2nd Congress of World Association for Laser Therapy, Sept. 2-5, 1998, Kansas City, Missouri.

Abstract Information & Notes

J. Howard Garrett Date of Talk: Friday, June 8, 5:00 p.m.
The Natural Way/WBAP Radio Phone: n/a
P.O. Box 140650 Fax: 214/365-0608
Dallas, TX 75214 E-mail: howard@dirtdoctor.com

School Attended: Texas Tech University, received his landscape architecture degree in 1969
Current Job Description: Landscape architect, columnist for Dallas Morning News, gardening talk show host on WBAP-820

Other Information: Author, of nine books on gardening, landscaping & pest control. Consultant, on farming, ranching, & landscaping.

Disclosure Statement: Financial interest in Garden-ville Inc., Muenster Milling, Alliance Milling

SPEECH TITLE: **"BOP The Basic Organic Program"**
The information below has been provided by the speaker.

1.) Goals and objectives: Teaching how to plant the landscape to consist totally of medicinal and culinary plants.

2.) Outline of talk/abstract: Design, bed preparation, trees, shrubs, ground covers, vines, perennials, annuals

3.) Conclusion of what is to be learned: The entire landscape can be made of useful plants and grown with organic techniques.

4.) References: Dirt Doctor's Dirt Newsletter, Texas Organic Vegetable Gardening Book, Texas Bug Book, The Organic Manual

BASIC ORGANIC PROGRAM (the BOP)

J. Howard Garrett

SOIL TESTING - Send soil samples to Texas Plant and Soil Lab in Edinburg, TX, 956-383-0739 for organic recommendations. Another way to test the soil is to dig a cubic foot of soil and sift it back into the hole. If you don't see about 10 earthworms, you need to do more of what's listed below.

PLANTING - Prepare new planting beds by scraping away existing grass and weeds, adding a 4-6" layer of compost, lava sand at 40-80 lbs., organic fertilizer at 20 lbs. horticultural cornmeal 10-20 lbs./1,000 sq. ft. and tilling to a depth of 3" into the native soil. Excavation and additional ingredients such as concrete sand, topsoil and pine bark are unnecessary and can cause problems. More compost is needed for shrubs and flowers than for groundcover. Add Texas greensand to black and white soils and high-calcium lime to acid soils. Soft rock phosphate is an effective amendment for all soils.

FERTILIZING - Apply an organic fertilizer 2-3 times per year. During the growing season, spray turf, trees and shrub foliage, trunks, limbs and soil at least monthly with Garrett Juice. Add lava sand annually at 40-80 lbs./1,000 sq. ft.

MULCHING - Mulch all shrubs, trees and ground cover with 3-5" of, shredded tree trimmings or shredded hardwood bark to protect the soil, inhibit weed germination, decrease watering needs and mediate soil temperature. Mulch vegetable gardens with 8" of alfalfa hay, rough-textured compost or shredded native tree trimmings. Avoid Bermuda hay because of the possibility of broadleaf herbicide contamination. Shredded native cedar is the best of all mulches.

WATERING - Adjust schedule seasonally to allow for deep, infrequent watering in order to maintain an even moisture level. Start by applying about 1" of water per week in the summer and adjust from there. Water needs will vary from site to site and from season to season. Add 1 tablespoon natural vinegar per gallon when watering pots, unless water is acid.

MOWING - Mow weekly, leaving the clippings on the lawn to return nutrients and organic matter to the soil. General mowing height should be 2-1/2" or taller. Put occasional excess clippings in compost pile. **Do not ever bag clippings**. **Do not let clippings ever leave the site.** Mulching mowers are best if the budget allows. Do not use line trimmers around trees.

WEEDING - Hand pull large weeds and work on soil health for overall control. Mulch all bare soil in beds. AVOID SYNTHETIC HERBICIDES, especially pre-emergents, broad-leaf treatments and soil sterilants. These are unnecessary toxic pollutants. Spray broadleaf weeds as a last resort with full strength vinegar, and citrus mix or remove mechanically. Commercial organic herbicides are now on the market.

PRUNING - Remove dead, diseased and conflicting limbs. Do not over prune. Do not make flush cuts. Leave the branch collars intact. Do not paint cuts except on red oaks and live oaks in oak-wilt areas when spring pruning can't be avoided. Remember that pruning cuts hurt trees. Pruning is done for your benefit, not for the benefit of the trees.

COMPOST MAKING - Compost, Nature's own living fertilizer, can be made at home or purchased ready-to-use. A compost pile can be started any time of the year in sun or shade. Anything once living can go in the compost - grass clippings, tree trimmings, food scraps, bark, sawdust, rice hulls, weeds, nut hulls and animal manure. Mix the ingredients together and simply pile the material on the ground. The best mixture is 80% vegetative matter and 20% animal waste, although any mix will compost. Since oxygen is a critical component, the ingredients should be a mix of coarse and fine-textured material to promote air circulation through the pile. Turn the pile once a month if possible, more often speeds up the process but releases nitrogen to the air. Another critical component is water. A compost pile should be roughly the moisture of a squeezed-out sponge to help the living organisms thrive and work their magic. Compost is ready to use as a soil amendment when the ingredients are no longer identifiable. The color will be dark brown, the texture soft and crumbly and it will smell like the forest floor. Rough, unfinished compost can be used as a top-dressing mulch around all paintings.

MANURE COMPOST TEA - Manure compost tea is an effective foliar spray because of many mineral nutrients and naturally occurring microorganisms. Fill any container half full of compost and finish filling with water. Let the mix sit for 10-14 days and then dilute and spray on the foliage of any and all plants. How to dilute the dark compost tea before using depends on the compost used. A rule of thumb is to dilute the leachate down to one part compost liquid to 4 to 10 parts water. The ready-to-use spray should look like iced tea. Be sure to strain the solids out with old pantyhose, cheese cloth or floating row cover material. Full strength tea makes an excellent fire ant mound drench when mixed with 2 oz. molasses and 2 oz. citrus oil per gallon. Add vinegar, molasses and seaweed to make Garrett Juice.

CONTROLLING INSECTS - Aphids, spider mites, whiteflies & lacebugs: release ladybugs and green lacewings regularly until natural populations exist. Garrett Juice and/or garlic-pepper tea (recipes below) are effective controls. Use strong water blasts for heavy infestations. **Caterpillars and bagworms:** release trichogramma wasps. Spray *Bacillus thuringiensis* (Bt) as a last resort. **Fire ants:** Drench mounds with Garrett Juice plus citrus oil and release beneficial nematodes. **Grasshoppers:** Eliminate bare soil, apply beneficial nematodes, and then dust or spray one or more of the following: self-rising flour, natural diatomaceous earth, fire ant control formula. Encourage biodiversity and feed the birds. **Grubworms:** beneficial nematodes and general soil health is the primary control. **Mosquitoes:** *Bacillus thuringiensis* 'Israelensis' for larvae in standing water. Spray citrus oil or garlic-pepper tea for adults. Lavender, vanilla,

citronella and eucalyptus also repel mosquito adults. **Slugs, snails, fleas, ticks, chinch bugs, roaches, crickets:** spray or dust diatomaceous earth products and crushed red pepper. Citrus oil also kills these pests. For more details on pest control, check out the new *Texas Bug Book.*

CONTROLLING DISEASES - Black spot, brown patch, powdery mildew and other fungal problems: best control is prevention through soil improvement, avoidance of high-nitrogen fertilizers and proper watering. Spray Garrett Juice plus garlic and/or neem. Baking soda or potassium bicarbonate can also be added. Treat soil with horticultural cornmeal at about 20 lbs./1,000 sq. ft. Alfalfa meal and mixes containing alfalfa are also good disease fighters.

GARLIC-PEPPER TEA INSECT REPELLENT - In a blender with water, liquefy 2 bulbs of garlic and 2 cayenne or habanero peppers. Strain away the solids. Pour the garlic-pepper juice into a 1 gallon container. Fill the remaining volume with water to make one gallon of concentrate. Shake well before using and add 1/4 cup of the concentrate to each gallon of water in the sprayer. To make garlic tea, simply omit the pepper and add another bulb of garlic. For additional power add 1 tablespoon of seaweed and molasses to each gallon. Always use plastic containers with loose fitting lids for storage.

GARRETT JUICE (foliar spray and soil drench) - Mix the following per gallon of water: 1 cup of compost tea or liquid humate, 1 ounce liquid seaweed, 1 ounce blackstrap molasses, 1 ounce apple cider vinegar. **To make a mild insect control product, add 1 oz. of citrus oil per gallon of spray. To make the fire ant killer, add 2 oz. of citrus oil per gallon.** When spraying the foliage of plants, don't use over 2 oz. of citrus oil per gallon of spray. This mixture also works as a soil detox.

DIRT DOCTOR'S POTTING SOIL - 5 parts compost, 4 parts lava sand, 3 parts peat moss, 2 parts cedar flakes, 1 part soft rock phosphate, 1 part earthworm castings, ½ wheat bran/cornmeal soil amendment, ¼ part organic fertilizer, ¼ part sul-po-mag, ¼ part Texas greensand. This is a very powerful potting soil and needs no additional fertilizer. It is also too strong to use for most interior house plants.

SATURDAY, JUNE 9, 2001 FACULTY

Iris R. Bell, M.D.
Program in Integrative Medicine
University of Arizona
College of Medicine
Tucson, AZ
520/626-3512

Clement E. Furlong, Ph.D.
Department of Genetics
University of Washington
Seattle, WA
206/543-1193

Satoshi Ishikawa, M.D.
Clinical Environmental Health Center, Kitasato Hospital
The Kitasato Institute
Machida-shi, Tokyo, Japan
81/427-95-5784

Rima E. Laibow, M.D.
Alexandria Institute of Natural and Integrative Medicine
Croton on Hudson, NY
914/827-9557

Allan D. Lieberman, M.D.
Center for Occupational & Environmental Medicine North Charleston, SC
843/572-1600

William J. Meggs, M.D., Ph.D.
E. Carolina University School of Medicine
Dept. of Emergency Medicine
Greenville, NC
252/816-2954

Jean A. Monro, M.D.
Breakspear Hospital
Hemel Hempstead, Herts, England
011/44-1442-261333

Ron Overberg, Ph.D. C.C.N.
Environmental Health Center-Dallas
Dallas, TX
214/368-4132

Kalpanna D. Patel, M.D.
Northwest Center for Allergy & Environmental Medicine
Buffalo, NY
716/837-1320

William J. Rea, M.D.
Environmental Health Center - Dallas
Dallas, TX
214/3684132

Sherry A. Rogers, M.D.
Northeast Center for Environmental Medicine
Syracuse, NY
315/488-2856

Theodore R. Simon, M.D.
Functional Imaging of Texas, P.A.
Dallas, TX
972/566-4710

NINETEENTH ANNUAL INTERNATIONAL SYMPOSIUM ON MAN AND HIS ENVIRONMENT

SCHEDULE FOR SATURDAY, JUNE 9, 2001

8:15 ANNOUNCEMENTS/MODERATOR: Sherry A. Rogers, M.D.

8:30 Rima E. Laibow, M.D. "Neuronal Repair of Neurotoxicity"
8:50 Q & A

9:00 Allan D. Lieberman, M.D. "CO Poisoning, The Premier Neurotoxicant"
9:20 Q & A

9:30 William J. Rea, M.D. "Neurotoxicity Peripheral"
9:50 Q & A

10:00 BREAK WITH EXHIBITORS/MODERATOR: George Miller, M.D.

10:30 Kalpanna D. Patel, M.D. "Neurotoxicity, Aspects of Toxic Heavy Metals Solvents and Treatment"
10:50 Q & A
11:00 Iris R. Bell, MD "Psychophysiological Studies in Gulf War Veterans"
11:20 Q & A

11:30 Jean A. Monro, M.D."Depleted Uranium and Its Effects"
11:50 Q & A

12:00n OPEN LUNCH

MODERATOR: Kalpanna D. Patel, M.D.
1:30 Ron Overberg, Ph.D. C.C.N. "Nutritional Findings of Neurotoxicity"
1:50 Q & A

2:00 Sherry A. Rogers, M.D. "Scientific Basis for Reversing Recalcitrant Neurotoxicity and Other Disease Through Detoxification"
2:20 Q & A

2:30 Theodore R. Simon, M.D. "Cancer and Nuclear Medicine"
2:50 Q & A

3:00 BREAK WITH EXHIBITORS/MODERATOR: Adriene Buffalo, M.D.

3:30 William J. Meggs, M.D. "Depression Anxiety Associated with Allergies and Chemical Sensitivity"
3:50 Q & A

4:00 Clement E. Furlong, Ph.D."Human Paraoxonase Polymorphisms: Role of the Metabolism of Lipids and Drugs"

4:20 Q & A

4:30 Satoshi Ishikawa, M.D. "Neurological Manifestations Due to Low Dosage Exposure of Organophosphorus Compounds"
4:50 Q & A

5:00 CASE STUDIES & PANEL/MODERATOR: Richard Jaeckle, M.D.
1.) Case title currently not available.
2.) Case title currently not available.

6:00 RECEPTION

SATURDAY, JUNE 9, 2001

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

Rima E. Laibow, M.D. Date of talk: Saturday, June 9, 8:30 a.m.

Alexandria Institute of Natural and Integrative Medicine Phone: 914/827-9557
10 Old Post Road South Fax: 914/827-3995
Croton on Hudson, NY 10520-2350 E-mail: laibow@juno.com

Medical School Attended: Albert Einstein College of Medicine
Residency: Lincoln Hospital and St. Luke's Hospital
Board Certifications: 1) Diplomate American Board of Forensic Examiners, 2) Diplomate American Board of Traumatic Stress Studies, 3) Diplomate Neurotherapy Certification Board.
Current Faculty Appointments: N/A
Current Job Description: Medical Director of the Alexandria Institute of Natural and Integrative Medicine
Other Information: 1) Senior Medical Editor Alternative Medicine: The Definitive Guide
2) Author: The Medical Applicatiions of NeuroBioFeedBack in Evans and Aberbanel, Introduction to Quantitatve EEG and NeuroBioFeedBack, Academic Press, 1999
3) President: NeuroTherapy Certification Board
4) Past President: Quantitative EEG Technicians Certification Board
5) Editorial Board, Journal of Neurotherapy.

Disclosure Statement: None
SPEECH TITLE: **"Neuronal Repair of Neurotoxicity"**

The information below has been provided by the speaker.

1.) Goals and objectives: The practioner will be able to assess the appropriateness of several modes of treatment for patients experiencing Neurotoxicity including neuronally stimulated repair through NeuroBioFeedBack Supplemented with nutrition, detoxification and repair.

2.) Outline of talk/abstract: Neuronal dysfunction and pathology is very difficult to correct in the face of heavy metal poisoning. NeuroBioFeedBack offers the brain the opportunity to reregulate its direct and indirect functional control mechanisms. This modality and the nutritional/detoxification support which makes repair more profound will be discussed in detail.

3.) Conclusion of what is to be learned: Practioners will be better able to determine appropriate treatment(s) for patients with neurotoxicity by including novel modalities in their repetoire.

4.) References: (References 1, 2, & 3 from speech #1) plus -Laibow, RE; Stubblebing, AN; Sandground, H; Bonaly, A and Bounais, H; Neuropsycho-toxicity and QEEG: NeuroBioFeedBack in Diagnosis and rehabilitation" in J. Chem Technol, Environ. Toxicol. And Occup. Med., Vol 5, No 4, 1996 pp 325-328.

Notes:
Abstract Information & Notes

Allan D. Lieberman, M.D. Date of talk: Saturday, June 9, 9:00 a.m.

Center for Occupational & Environmental Medicine, P.A. Phone: 843/572-1600
7510 Northforest Dr. Fax: 843/572-1795
North Charleston, SC 29420-4297 E-mail: allanl@coem.com

Medical School Attended: Chicago Medical School
Residency: Children's Memorial Hosp. - Chicago
Board Certifications: Am. Bo. Env. Med.
Current Faculty Appointments: Brown Univ. - Dept. of Biochemistry, Asst. Professor
Current Job Description: Medical Director Center for Occupational Environmental Medicine, Charleston S.C.

Disclosure Statement: None

SPEECH TITLE: **"CO Poisoning, The Premier Neurotoxicant"**
The information below has been provided by the speaker.

1.) Goals and objectives: To make aware of the 3 most common causes of Neurotoxicity.

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned: Clinical manifestations and sequelae of 3 of the most common causes of Neurotoxicity.

4.) References:

CO POISONING
The Premier Neurotoxicant

By Allan D. Lieberman, M.D.

Goal: Increase awareness that CO poisoning is the single greatest cause of poisoning in America.

Objectives:
1. Recognize the chemical patterns of injury
2. Recognize the underlying tissue pathology
3. To not underestimate the impairments and disability that can come from CO poisoning

In the course of practicing occupational and environmental medicine, physicians see a host of workers and patients who are exposed to neurotoxic substances.

My experience over 24 years of practice reveals a failure of most physicians to recognize neurotoxicity and to deny truly impaired and disabled workers their right to fair compensation for a work related injury.

This presentation recognizes three of the most common causes of neurotoxic injury - solvents, pesticides, and carbon monoxide. Many of the other speakers will present papers on solvents.

An appreciation of the extensive pathological changes seen at autopsy in CO poisoned people explains clearly the clinical features which follow CO poisoning.

In acute CO poisoning with early death, all the viscera, muscles, and the brain show a pink color, marked congestion, and petecheal or massive hemorrhages. In the brain of patients dying after a delay of weeks, the changes typical of anoxia or ischemia are seen and consist of focal or laminar necrosis of the second and third cortical layers and often of the superficial white matter. Lesions of the Purkinje cells of the cerebellum and Ammon's horn are common and consist of ischemic, homogenizing degeneration with glial proliferation and loss of nerve cells. Autopsy studies of those who survive and die years later for other reasons show varying degrees of total, laminar or disseminated focal atrophy. Occasional ischemic necrosis is found in other organs including the heart, skeletal muscles, and kidneys.

Localized damage or hemorrahage into the anterior part of globus pallidus has been considered a characteristic of CO poisoning and attributed to a primary vascular lesion rather than to an effect produced by anoxia. Demyelination of the white matter is more frequently found in those surviving the early stages and is then accompanied by diffuse gliosis and cereberal atropy.[1]

The acute and chronic effects of CO can mimic virtually any neurologic or psychiatric illness with symptoms resembling:
1. MS
2. Parkinsonism
3. Korsakoffs Amnestic Syndrome
4. Bipolar Disorder
5. Schizophrenia and
6. Hysterical conversion reaction
have been reported.

Insults to the basal ganglia cause:
1. Tremor
2. Decreases in motor speed
3. Slowed reaction time
4. Poor manual dexterity
5. Decreased eye hand coordination, and
6. Poor sequencing of complex motor movements.[2]

Characteristic of CO poisoning is an induced delayed neuropsychiatric syndrome which consists of :
1. A pseudorecovery period (of up to several weeks)
2. Followed by an abrupt onset of neurologic and psychiatric deterioration (reported to range from 2-30%)

3. Manifestations include gross neurologic impairment as:
- Parkinsonism
- Apraxia
- Intellectual deterioration
- Memory impairment
- Personality changes

4. Long term sequelae of CO poisoning is:
- Depression
- Anxiety
- Agitation

Case Reports:

Conclusion

COHb level correlates poorly, if at all, with loss of consciousness, eventual neurological defect and mortality. Duration of exposure usually provides a better correlation. Longer exposures to low levels may have more dangerous consequences than higher-level, shorter exposures.

If exposed to CO seek immediate medical attention if you experience:
1. Severe headache
2. Dizziness
3. Nausea and vomiting

References

1. Garland H, Pierce J. Neurological complications of CO poisoning. Quarterly J Med 1967;144:445-455.
2. Sullivan and Krieger. Hazardous materials toxicology. page 1162.
3. Bogusz et al. A comparison of two types of acute CO poisoning. Arch Toxicol 1975; 33:141-149.
4. Sokal & Kralkowska. CO poisoning. Arch Toxicol 1985;57;196-199.
5. Lasater SR. CO posioning. Can Med Asso J 1986;134:991-92.
6. Penney DG, White SR. The neural and behavioral effects of carbon monoxide. In Jemsen KF, ed. The vulnerable brain and environmental risks, volume 3: toxins in air and water. New York: Plenum Press, 1994.

Abstract Information & Notes

William J. Rea, M.D. Date of talk: Saturday, June 9, 9:30 a.m.

Environmental Health Center - Dallas Phone: 214/368-4132
8345 Walnut Hill Lane, Suite 220 Fax: 214/691-8432
Dallas, TX 75231 E-mail: wjr@ehcd.com

Medical School Attended: Ohio State University College of Medicine
Residency: University of Texas SW Medical School; Parkland Memorial Hospital; Baylor Medical Center, Veteran's Hospital; Children's Medical Center
Board Certifications: American Board of Surgery; American Board of Thoracic Surgery; American Board of Environmental Medicine
Current Faculty Appointments: N/A

Current Job Description: M.D./President - Environmental Health Center - Dallas

Disclosure Statement: None

SPEECH TITLE: **"Neurotoxicity Peripheral"**

The information below has been provided by the speaker.

1.) Goals and objectives: For the clinician to understand that one of the causes of peripheral neuropathy is neurotoxicity.

2.) Outline of talk/abstract: History, treatment and laboratory tests and how to treat the problem.

3.) Conclusion of what is to be learned: Diagnosis and treatment.

4.) References: Chemical Sensitivity, Volumes I, II, III, IV

NEUROTOXICITY - PERIPHERAL

William J. Rea, M.D., F.A.C.S, F.A.A.E.M.
Yaqin Pan, M.D.

Abstract: Thirty (30) patients (M-6; F-24; ages 29-74 average age of 45.9 years) who had the complaints of numbness and tingling, loss of sensation or motion in a peripheral nerve or an increase in sensation were studied. Sensory nerve condition abnormality was present in all 30 patients. Associated symptoms included depression with less intensity headaches, short-term memory loss than CNS reactions in 80% of the patients. These symptoms were secondary to the peripheral nerve symptoms and signs. Chronic fatigue, fibromyalgia and bone pain was present in 57% of the patients. Respiratory symptoms included asthma and other types of bronchospasm in 40. ENT complaints of tinnitus, hearing loss and laryngeal edema were also present in 40% of the patients. Cardiovascular symptoms included anaphylaxis, mitral valve disorder and vasculitis in 27% of the patients, G.I. complaints of irritable bowel syndrome and malabsorption were seen in 17% of the patients.

Outline: Chemical sensitivity was seen in all the patients, food sensitivity in 33% and biological inhalant sensitivity in 23% with EMF sensitivity seen in 3%. Immune deregulation in the form of abnormal T-cells and subsets were seen in 37% of the patients.

Fifty (50) percent of the patients had CMI tests 3 or below indicating poor T-cell function. 87.5% had abnormal T-cell numbers with 43% having lower T_4, 25% low T_8, and 37% low B cells. T_4 was increased 12.5%, T_8 18.5% of the time. Eight (8) patients had triple camera brain SPECT and 8 were positive for toxicity. 22 patients had pupillography for measurement of the autonomic nervous system and 100% of these were abnormal. 56 neurometer tests for peripheral, median and ulnar digital, superficial peroneal, trigeminal, sural, nerve damage was performed in all 30 patients with 40 being positive and 16 negative. All patients had at least one objective abnormality. Eight (8) patients had toluene in their blood, 7 xylene and 1 benzene components. Eight (8) chlorinated compounds and 4 styrene, 9 - 2-methylpentane, 13 3-methylpentane and 10 n-Hexane. Interestingly, 18 patients had DDT and DDE and 7 had chlordane compounds, 2

hexachlorobenzene, 1 B-BHC and 1 Mirex. Intradermal skin testing show sensitivity to formaldehyde 70%, orrisroot 78%, cigarette smoke 70%, unleaded gas-diesel fuel 65%, new print 61% and petroleum derived ethanol 43%, Cl 35%, propane gas 26%, cologne 61%. 100% were sensitive to foods with 95% to molds, algae 68%, dust 73%, trees, weeds, grass 68%, terpenes 59%, virus 41%, bacteria 36%. Intradermal metal testing was performed on a subset of 8 who had high exposures to metal with sensitivity to nickel, zinc and titanium at 62%, tin 50%, copper 37%, aluminum and lead 25%, gold and stainless steel 12.5%.

Conclusion: 25 patients received treatment of massive avoidance of pollutants in air, food, water, injection therapy for primary and secondary offenders, oral and intravenous nutrient therapy, oxygen, heat depuration (sauna) and physical therapy and autogenous lymphocytic factor. 21 of 25 patients improved significantly for an effective rate of 84%.

Goals:
1. To understand the demographics of toxic peripheral neuropathy.
2. To be able to apply this knowledge in the diagnosis of peripheral neuropathy.
3. Allow the clinician to understand that successful treatment is available.

Notes:
Abstract Information & Notes

Kalpanna D. Patel, M.D. Date of talk: Saturday June 9, 10:30 a m

Northwest Center for Allergy & Environmental Medicine Phone: 716/837-1320
65 Wehrle Dr. Fax: 716/833-2244
Buffalo, NY 14225 E-mail: aehcwhy@wny

Medical School Attended: BJ Medical School
Residency: Bexar County Hospital
Board Certifications: American Board of Pediatrics Environmental Medicine
Current Faculty Appointments: Asst. Prof. Pediatrics, Suny Buffalo
Current Job Description: President American Board of Env. Med, President Allergy and Env. Health Center

Disclosure Statement: None

SPEECH TITLE: **"Neurotoxicity, Aspects of Toxic Heavy Metals Solvents and Treatment"**

The information below has been provided by the speaker.

1.) Goals and objectives:
1. To demonstrate the role of solvents and toxic heavy metals in Toxic Brain Syndrome, as the brain is the most sensitive organ for injury from chemicals.
2. To review major symptoms of toxic chemical exposure and toxic heavy metal to the brain, like memory loss, lack of concentration, headache, living in brain fog, inability to recall, feeling lost in familiar places, etc.
3. To demonstrate the efficacy of Environmental Medicine approach in reversing symptoms and obtain near optimal health.

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:
1. Many chemicals impair brain function.
2. Toxic heavy metals also impair brain function. When chemicals and toxic heavy metals are combined, they potentiate the toxic effect on the brain and central nervous system. The toxic effects are cumulative, aggressive and progressive. The loss of brain function resembles accelerated aging.
3. Comprehensive environmental evaluation is one of the most important tools to use in toxic brain patients.
4. Comprehensive treatment program can reverse the symptoms of neurotoxicity. Comprehensive treatment program includes avoidance, glass bottled water, less chemically contaminated food and minerals to augment detoxification, antigen injection treatment, intravenous treatment with antioxidants and vitamins, chelation to eliminate toxic heavy metals, heat depuration, and physical therapy to reduce total toxic load of chemicals and toxic heavy metals.

4.) References: Chemical Sensitivity Volume 1-4, William Rea, MD
Chemical Brain Injury, Kaye Kilburn

Abstract Information & Notes

Iris R. Bell, M.D. Date of talk: Saturday, June 9, 11:00 a.m.

Program in Integrative Medicine Phone: 520/626-3512
University of Arizona Fax: 520/626-3518
College of Medicine E-mail: ibell@u.arizona.edu
P.O. Box 245153
Tucson, AZ 85724

Medical School Attended: Stanford
Residency: U. of California - San Francisco
Board Certifications: Psychiatry with added qualification in Geriatric Psychiatry
Current Faculty Appointments: Associate Professor of Psychiatry
Current Job Description: Director of Research, Program in Integrative Medicine, University of Arizona College of Medicine

Disclosure Statement: None

SPEECH TITLE: **"Psychophysiological Studies in Gulf War Veterans"**

The information below has been provided by the speaker.

1.) Goals and objectives: To review sensitization model for Gulf War Syndrome and present relevant data

2.) Outline of talk/abstract: Summarize descriptive evidence for chemical exposure and chemical sensitivity in Gulf War veterans and give overview of data analysis in process testing for sensitization to JP-8 jet fuel exposures in Gulf Veterans.

3.) Conclusion of what is to be learned: A subset of ill Gulf Veterans may be vulnerable to acquiring new chemical sensitivities manifesting in changes in test period variability (autonomic dysfunction)

4.) References: No papers or lab data to date.
Other - Bell IR et al. J Chronic Fatigue Syndrome 1997;3:15-42.
Bell IR et al. Military Medicine 1998; 163:725-732.

Psychophysiological Studies in Gulf War Veterans
Iris R. Bell, MD PhD

Previous studies have shown that a large percentage of Gulf War veterans endorse multiple symptoms in multiple systems, e.g., fatigue, skin rashes, headaches, musculoskeletal pain, and memory loss, at increased rates. Ill Gulf veterans also report acquired low level chemical odor intolerance (CI) at higher rates than do healthy Gulf or Era veterans (Bell et al. 1998). Decreased heart rate variability is a nonspecific biomarker of reduced adaptability across various disorders, including fibromyalgia (which occurs at increased rates in ill Gulf veterans), multiple sclerosis, lupus, cardiac disease, and posttraumatic stress disorder. The sensitization model for CI (Bell et al 1999) suggests that ill Gulf veterans, especially those with CI, should show progressive amplification of adverse effects on heart rate variability of repeated low level chemical exposures to a salient stimulus (JP-8 jet fuel) as well as cross-sensitization to subsequent perfume exposures in the laboratory. The present study compared 4 groups of U.S. military veterans (mean age 40 SD 8 yrs, 85% male): ill Gulf veterans with CI, ill Gulf veterans without CI, healthy Gulf veterans, healthy Era veterans (served in military at same time but never deployed to Gulf region). The design was a parallel groups, randomized, double-blind assignment to either 3 sessions of repeated subolfactory threshold levels of JP-8 jet fuel or 3 sessions of repeated clean air (sham) exposures.

All groups and subgroups received detectable perfume exposures in session 4. Sessions were spaced 1 week apart. Subjects also underwent cognitive testing with the computerized visual divided attention test (DAT), which has shown replicable performance differences between civilians with and without CI. At Session 1 baseline, groups did not differ for resting heart rate, blood pressure, mean interbeat interval, standard deviation of the interbeat interval (SD IBI = heart rate variability), or eyeblink rate. The ill Gulf veterans with CI exhibited significantly slower central reaction times than did healthy Era veterans on the DAT at baseline. The patterns of change for SD IBI over sessions were complex and different between groups as a function of JP-8 versus sham exposures. The ill Gulf veterans with CI showed the predicted sensitization pattern to JP-8, with the lowest SD IBIs (poorest heart rate variability) of all groups. Statistical analyses using general linear and random effects regression models indicated that the two ill Gulf groups combined differed significantly from healthy Gulf veterans in their SD IBI patterns as a function of JP-8 exposure vs sham over sessions, even after controlling for age, ethnicity, sex, and psychological disturbance on the MMPI subscales.

We then evaluated the divergence between observed and expected values of SD IBI for perfume exposure as a function of prior JP-8 versus clean air (sham) exposures in the laboratory. Values close to expected would reflect a slope similar to that seen over the first 3 sessions. Notably, with perfume, the groups except the ill Gulf veterans without CI and healthy Era veterans diverged significantly from both their prior JP-8 exposure and their sham response patterns. Only healthy Era veterans showed statistically comparable changes in their perfume responses regardless of

having received JP-8 or clean air sham in the prior three sessions. Notably, the direction of response in the ill Gulf veterans without CI and the healthy Gulf veterans was opposite for their respective JP-8 and sham-exposed subgroups, consistent with exposure-dependent and intensity-dependent oscillatory effects observed in animal studies by Antelman and Caggiula (1996; 1998). Taken together, the data suggest that ill Gulf veterans with CI have impaired divided attention task performance and that ill Gulf veterans with and without CI differ from healthy Gulf veterans in their pattern of change in heart rate variability over time as a function of repeated JP-8 jet fuel exposures vs sham (clean air). Prior exposures to JP-8 vs clean air influence the nature of the subsequent response to perfume in all Gulf veterans, ill or healthy, with and without CI, though the direction of the response varies between groups.

References

1. Antelman SM, Caggiula AR. Oscillation follows drug sensitization: implications. Crit Rev Neurobiol 1996; 10(1):101-117.
2. Bell IR, Baldwin CM, Fernandez M, Schwartz GER. Neural sensitization model for multiple chemical sensitivity: overview of theory and empirical evidence. Toxicol Industr Health 1999; 15:295-304.
3. Bell IR, , Warg-Damiani L, Baldwin CM, Walsh M, Schwartz GE. Self-reported chemical sensitivity and wartime chemical exposures in Gulf War veterans with and without decreased global health ratings. Military Medicine 1998; 163:725-732.
4. Caggiula AR, Antelman SM, Kucinski BJ, Fowler H, Edwards DJ, Austin MC, Gershon S, Stiller R. Oscillatory sensitization model of repeated drug exposure: cocaine's effects on shock-induced hypoalgesia. Prog NeuroPsychopharmacol Biol Psychiatry 1998; 22:511-521.
5. Iowa Persian Gulf Study Group. Self-reported illness and health status among Gulf War veterans. A population-based study. JAMA 1997; 277:238-245.

Notes:
Abstract Information & Notes

Jean A. Monro, M.D. Date of talk: Saturday, June 9, 11:30 am

Breakspear Hospital Phone: 011/44-1442-261333
Lord Alexander House, Waterhouse Street Fax: 011/44-1442-266388
Hemel Hempstead, Herts HP1 1DL, England E-mail: jmonro@breakspear.org

Medical School Attended: London Hospital Medical School, England
Residency: London Hospital
Board Certifications: MB, BS, MRCS, LRCP, FAAEM, DIBEM, MACOEM
Current Faculty Appointments: n/a
Current Job Description: Medical Director of Breakspear Hospital, England

Disclosure Statement: None

SPEECH TITLE: **"Depleted Uranium and Its Effects"**

The information below has been provided by the speaker.

1.) Goals and objectives: To illustrate that depleted uranium can have chemical toxic effects. Abnormal ion channel function is known to occur with heavy metals. Ion channel function can be

shown to occur in chronic fatigue syndrome, the main feature of many patients who have been exposed to depleted uranium. Other ion channelopathies occur with orgfanophosphates and syngergistic effects are significantly over-looked.

2.) Outline of talk/abstract: An account of toxicity of depleted uranium will be illustrated, and also a discussion on channelopathies.

3.) Conclusion of what is to be learned: Chemical sensitivity could well be related to chronic fatigue syndrome of an individual by channelopathies from many sources.

4.) References:

1. Priest ND, School of Health, Biological and Environmental Sciences, Middlesex University, London. Toxicity of depleted uranium. The Lancet;Vol 357:January 27, 2001.

2. Chaudhuri A. The symptoms of chronic fatigue syndrome are related to abnormal ion channel function. Med Hypotheses 200 Jan;54(1):59-63.

3. Brinkmeier H. An endogenous pentapeptide acting as a socium channel blocker in inflammatory autoimmune disorders of the central nervous system. Nat Med 200 Jul;6(7):808-11.

Notes:
Abstract Information & Notes

Ron Overberg, Ph.D. C.C.N. Date of talk: Saturday, June 9, 1:30 p.m

Environmental Health Center-Dallas Phone: 214/368-4132
8345 Walnut Hill, Ste. 220 Fax: 214/691-8432
Dallas, TX 75231 E-mail: drron@nutriwellness.com

Medical School Attended: University of Texas at Dallas - Ph.D. 1985
Residency: International and American Association Of Clinical Nutritionists
Board Certifications: (IAACN) - Board Certified Clinical Nutritionist by C.N. CB
Current Faculty Appointments: Assistant Professor at Parner Chiropractic College
Current Job Description: Nutrition and Diet Counselor, Educator and Motivator
Other Information: Visit nutriwellness.com

Disclosure Statement: None

SPEECH TITLE: **"Nutritional Findings of Neurotoxicity"**

The information below has been provided by the speaker.

1.) Goals and objectives: To raise the awareness of the extent of fat-soluble vitamins and anti-oxidants in environmentally sensitive patients.

2.) Outline of talk/abstract: An analysis of thirty environmentally sensitive patients in respect to blood levels of : Coenzyme Q10, alpha-tocopherol, gamma-tocopherol, lycopene, beta-carotene, alpha-carotene, vitamin A and vitamin C. Each patient's values are compared to those of

individuals of the same sex and age and common deficiency trends and their treatment is discussed.

3.) Conclusion of what is to be learned: Environmentally sensitive patients have definite fat-soluble vitamin and anti-oxidant deficiencies which need to be identified and treated as part of their overall recovery program.

4.) References:

Nutritional Findings of Neurotoxicity

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

Objectives: To show anti-oxidant deficiencies in environmentally sensitive patients with neurological symptoms.

Abstract: Thirty environmentally sensitive patients were evaluated for blood levels of: Coenzyme Q10, alpha-tocopherol, gamma-tocopherol, lycopene, beta-carotene, alpha-carotene, vitamin A and vitamin C. Each patient's values are compared to those of individuals of the same sex and age and common deficiency trends and their treatment is discussed.

Conclusion: Enviromentally sensitive patients with neurotoxicity have definite fat-soluble antioxidant deficiencies which need to be identified and treated as part of their overall recovery program.

****
Study

Thirty environmentally sensitive patients were evaluated for blood levels of: Coenzyme Q10, alpha-tocopherol, gamma-tocopherol, lycopene, beta-carotene, alpha-carotene, vitamin A and vitamin C. Each patient's values are compared to those of individuals of the same sex and age and common deficiency trends and their treatment is discussed.

****
Antioxidant Lab Analysis

30 patients total
23 females
7 males

Ages 14 - 69
Average age 50 years
****
Diagnosis # %

Neurological Symptoms (TBS, memory loss, difficulty concentrating, 20 67
neurotoxicity, neuropathy, H/A, Vertigo)

M.S. (Fatigue, Fibromyalgia) 18 60

Respiratory (ARS, Bronchitis, Asthma, SOB, Chronic cough) 19 63

C.V. (Arrhytmia, vasculitis, chest pain, angioedema, hypertension, 16 53
hypotension)

G.I. (IBS, Malabsorption) 10 33

Immune Dysregulation 7 23

Endocrine Dysregulation (PMS, Hormone Imbalance) 4 13

Dermatitis 2 7

Candiditis 2 7

Lipid-Soluble Antioxidants	NORMAL		HIGH		LOW	
	#	**%**	**#**	**%**	**#**	**%**
Coenzyme Q10	1	3	8	27	21	70
alpha-Tocopherol	1	3	8	27	21	70
gamma-Tocopherol	0	0	0	0	30	100
Lycopene	2	7	1	3	27	90
beta-Carotene	0	0	18	60	12	40
alpha-Carotene	0	0	7	23	23	77
Vitamin A (Retinol)	0	0	10	33	33	67
Water-Soluble Antioxidants						
Vitamin C (ascorbate)	0	0	23	77	7	23

****

Sources of Antioxidants

Coenzyme Q10: seafood and organ meats
alpha-Tocopherol: soybean, rice bran oil and palm oil
gamma-Tocopherol: soybean, rice bran oil, corn oil and palm oil
Lycopene: tomato, guava, watermelon, pink grapefruit and apricot, red palm oil (not in strawberries, red bell peppers, D. Salina)
beta-Carotene: carrot, tomato, mango, sweet potato, spinach, cantaloupe, apricots, squash, strawberries, bell pepper, broccoli, D. Salina, palm oil
alpha-Carotene: carrot, pumpkin, D. Salina, palm oil
Vitamin A: Cod liver oil, fish liver oil, liver
Vitamin C: Corn, sago palm, potato, beet, tapioca

Of interest
Lutein, zeaxanthin spinach, collard greens, beet greens, watercress, collard greens, mustard greens, swiss chard, pumpkin, marigolds
Cryptoxanthin: papayas, peaches, tangerines, oranges
Tocotrienols: palm oil, rice bran oil and cereal brans

Note: Only beta-carotene, alpha -carotene and cryptoxanthin are converted in vitamin A

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Conclusion:

Enviromentally sensitive patients with neurotoxicity have definite fat-soluble antioxidant deficiencies which need to be identified and treated as part of their overall recovery program.

Abstract Information & Notes

Sherry A. Rogers, M.D. Date of talk: Saturday, June 9, 2:00 p.m
Northeast Center for Environmental Medicine Phone: 315/488-2856
2800 W. Genesee St. Fax: 315/488-7518
Syracuse, NY 13219 E-mail: n/a

Medical School Attended: State University of New York
Residency: Health Sciences Center at Syracuse
Board Certifications: Environmental Medicine & Family Practice
Current Faculty Appointments: n/a
Current Job Description: Medical Director Northeast Center for Environmental Medicine, Also author of over a dozen books, a monthly referenced newsletter for 12 years, latest books (2001) on detoxification, "Pain-free in 6 weeks" and "Detoxify or Die"

Disclosure Statement: None

SPEECH TITLE: **"Scientific Basis for Reversing Recalcitrant Neurotoxicity and Other Disease Through Detoxification"**

The information below has been provided by the speaker.

1.) Goals and objectives: To demonstrate that undetected nutrient deficiencies and ubiquitous environmental xenobiotics bioaccumulate in man, mimicking any symptom and disease, most of which reportedly have no discernible cause. When corrected, the most recalcitrant and baffling symptoms can disappear, depending on the initial total load of the individual.

2.) Outline of talk/abstract: Nutrient deficiencies, dysfunctional detoxification pathways, heavy metal, pesticide, and other xenobiotic toxicities have a synergistic effect in creating disease. First a clinician must make the paradigm shift, replacing the notion that a condition has no known cause, and that drugs are the only option. Next understanding how to identify and correct nutrient deficiencies, revitalize the detoxification pathways and identify and then depurate the bioaccumulated xenobiotics, allows him to orchestrate wellness.

3.) Conclusion of what is to be learned: Regardless of the futility of the diagnostic label or how recalcitrant a condition appears, there is often a solution if detoxification via the environmental medicine approach is used.

4.) References:

Rea WJ, *Chemical Sensitivity, Volumes I-IV,* CRC Press, Boca Raton, 1992-8

Manson MM, Ball HWL, Neal GE, et al., Mechanism of action of dietary chemo-protective agents in rat liver: induction of phase I and II drug metabolizing enzymes and aflatoxin B1 metabolism *Carcinogenesis* 18;9:1729-1738, 1997

U.S. Department of Health & Human Services, Agency for Toxic Substances and Disease Registry, *Case studies in environmental medicine, Vol.10, Cadmium Toxicity,* 1-19, June 1990

U.S. EPA, *National Human Adipose Tissue Survey, Vols. I-V,* EPA-560/5-84-003 through 87-035, 1984-1987

Lieberman, Reactive intestinal dysfunction syndrome (RIDS) caused by chemical exposures, *Arch Environ Health,* 53;5:354, 1998

U.S. environmental Protection Agency, *Chemicals identified in human biological media, a data base.* EPA-560/5-84-003, 1984.

Rea, W., et al, *J Nutr Environ Med,* 7;2:141, 1996

Roland N, et al, Interaction between the intestinal flora and xenobiotic metabolizing enzymes and their health consequences, *World Rev Diet* 74:123, 1993

Bruce, A diet high in whole and unrefined foods favorably alters lipids, antioxidant defenses, and colon function, *J Am Coll Nutr,* 19;1:61, 2000

Abstract Information & Notes

Theodore R. Simon, M.D. Date of talk: Saturday, June 9, 2:30 p.m.
Functional Imaging of Texas, P.A. Phone: 972/566-4710
4429 Southern Ave. Fax: 972/566-4762
Dallas, TX 75202 E-mail: ted@aya.yale.edu

Medical School Attended: Yale
Residency: University of California at San Francisco; Yale
Board Certifications: American Board of Nuclear Medicine
Current Faculty Appointments: withheld
Current Job Description: Nuclear Medicine Physician

Disclosure Statement: None

SPEECH TITLE: **Cancer and Nuclear Medicine**

The information below has been provided by the speaker.
Single Photon Emission Computed Tomography (SPECT) continues to develop new ways to diagnose, stage, palliate, and treat cancer even as experience with Positron Emission Tomography (PET) becomes increasingly available. Recent developments will be reviewed emphasizing expanded opportunities to manage prostate cancer.

1.) Goals and Objectives: This presentation will address both the lay and professional health care provider with an overview of options for identifying and managing cancer using nuclear medicine techniques.

2.) Outline: Clinical case examples will be presented as a means of illustrating diagnostic, palliative, and therapeutic algorithms.

3.) Conclusions: Both imaging and non-imaging developments are enhancing reasonable expectations for the person diagnosed with an increasingly broad range of cancers. Both the quality of life as well as longevity has dramatically improved as a result of new opportunities.

4.) References:
Notes:

Abstract Information & Notes

William J. Meggs, M.D., Ph.D. Date of talk: Saturday, June 9, 3:30 pm

E. Carolina University School of Medicine Phone: 252/816-2954
Dept. of Emergency Medicine Fax: 252/816-3589
Brody Bldg., Room 4W54 E-mail: meggs@brody.med.ecu.edu
Greenville, NC 27858

Medical School Attended: University of Miami
Residency: Internal Medicine - University of Rochester; Allergy & Immunology - N.I.H.; Medial Toxicology - New York University
Board Certifications: Internal Medicine, Emerg. Med, Medical Toxicology, Allergy & Immunology
Current Faculty Appointments: Brody Medical School at East Carolina Univ.
Current Job Description: Chief, Division of Toxicology; Professor & Vice Chair for Clinical Affairs, Dept. of Emergency Medicine

Disclosure Statement: None

SPEECH TITLE: **"Depression and Anxiety Associated with Allergies and Chemical Sensitivity"**

The information below has been provided by the speaker.

1.) Goals and objectives: See slides

2.) Outline of talk/abstract: See slides

3.) Conclusion of what is to be learned: See slides

4.) References: See slides

Notes:

Abstract Information & Notes

Clement E. Furlong, Ph.D. Date of talk: Saturday, June 9, 4:00 p.m.

Department of Genetics Phone: 206/543-1193
University of Washington Fax: 206/543-0754
Box 357360 E-mail: clem@u.washington.edu
Seattle, WA 98195-7360

Medical School Attended: Ph.D. 1968, Biochemistry, University of California, Davis
Residency: n/a
Board Certifications: n/a
Current Faculty Appointments: Departments of Genetics and Medicine, Division of Medical Genetics
Current Job Description: Research in human biochemical genetics
Other Information:

Disclosure Statement: Bager Corporation (research support)

SPEECH TITLE: **"Human Paraoxonase Polymorphisms: Role of the metabolism of Lipids and Drugs"**

The information below has been provided by the speaker.

1.) Goals and objectives: The goals are to explain the effects on lipid and drug metabolism of several polymorphisms in the coding and promoter regions of the human paraoxonase (*PON1*) gene on human chromosome 7. PON1 is found tightly associated with high density lipoprotein (HDL) particles. PON1 reduces oxidized lipid content of both LDL and HDL. PON1 also metabolizes a number of drugs. Biochemical, genetic, animal model and epidemiological studies will be described.

2.) Outline of talk/abstract:

PON1 Is An HDL-associated Enzyme
PON1 Protects HDL And LDL from Oxidation
PON1 Metabolizes Oxidized Lipids
Most of the Epidemiological Studies Looking at PON1 and Cardiovascular Disease Have Only Examined PON1 Genotype(s)
PON1 Status (Genotype Plus Phenotype) is a Better Predictor of Vascular Disease Than Genotype Alone.
PON1 Metabolizes Number of Drugs, Activating Some And Inactivating Others
PON1 Status Will Be Important to Consider In Human Pharmacokinetics

3.) Conclusion of what is to be learned: The major point to be learned from lecture #2 is that both the levels of expression of PON1 as well as the isoform expressed can be important in determining risk for cardiovascular disease. The importance of using the determination of PON1 status (genotype plus phenotype) vs. genotyping alone for epidemiological studies on the association of PON1 genetics with specific diseases will be discussed. The role of PON1 in the metabolism of several drugs will also be discussed as well as the pharmacokinetic implications of genetic variability in PON1.

4.) References:

1. Aviram M (1999) Does paraoxonase play a role in susceptibility to cardiovascular disease? Mol Med Today 5:381-6
2. Aviram M, Rosenblat M, Bisgaier CL, Newton RS, Primo-Parmo SL, La Du BN (1998) Paraoxonase inhibits high-density lipoprotein oxidation and preserves its functions. A possible peroxidative role for paraoxonase. J Clin Invest 101:1581-90
3. Biggadike K, Angell RM, Burgess CM, Farrell RM, Hancock AP, Harker AJ, Irving WR, et al (2000) Selective plasma hydrolysis of glucocorticoid gamma-lactones and cyclic carbonates by the enzyme paraoxonase: an ideal plasma inactivation mechanism. J Med Chem 43:19-21
4. Billecke S, Draganov D, Counsell R, Stetson P, Watson C, Hsu C, Du BN (2000) Human serum paraoxonase (pon1) isozymes Q and R hydrolyze lactones and cyclic carbonate esters [In Process Citation]. Drug Metab Dispos 28:1335-42
5. Jarvik GP, Rozek LS, Brophy VH, Hatsukami TS, Richter RJ, Schellenberg GD, Furlong CE (2000) Paraoxonase (PON1) phenotype is a better predictor of vascular disease than Is PON1(192) or PON1(55) genotype [In Process Citation]. Arterioscler Thromb Vasc Biol 20:2441-7
6. Katsuhiko T, A. Nakamura, S. Watanabe, Y. Okuyama and A. Morino (1998) Paraoxonase has a major fole in the hydrolysis of prulifloxacin (MN441), a prodrug of a new antibacterial agent. Drug Metab Dispos 26:355-359
7. Lusis AJ (2000) Atherosclerosis. Nature 407:233-241
8. Mackness B, Durrington PN, Mackness MI (1998a) Human serum paraoxonase. Gen Pharmacol 31:329-36
9. Mackness MI, Arrol S, Abbott CA, Durrington PN (1993) Is paraoxonase related to atherosclerosis. Chem Biol Interact 87:161-71
10. Mackness MI, Arrol S, Durrington PN (1991) Paraoxonase prevents accumulation of lipoperoxides in low-density lipoprotein [published erratum appears in FEBS Lett 1991 Nov 4;292(1- 2):307]. FEBS Lett 286:152-4
11. Mackness MI, Mackness B, Durrington PN, Fogelman AM, Berliner J, Lusis AJ, Navab M, et al (1998b) Paraoxonase and coronary heart disease. Curr Opin Lipidol 9:319-24
12. Richter RJ, Furlong CE (1999) Determination of paraoxonase (PON1) status requires more than genotyping. Pharmacogenetics 9:745-753
13. Shih DM, Gu L, Xia YR, Navab M, Li WF, Hama S, Castellani LW, et al (1998) Mice lacking serum paraoxonase are susceptible to organophosphate toxicity and atherosclerosis. Nature 394:284-7

Abstract Information & Notes

Speakers Name: **Satoshi Ishikawa, M.D.,** Date of talk: Saturday, June 9, 4:30 pm
Mikio Miyata, M.D., and Ko Sakabe, M.D.

Satoshi Ishikawa, M.D.

Clinical Environmental Health Center, Kitasato Hospital Phone: 81/427-95-5784
The Kitasato Institute Fax: 81/427-99-2287
4-5-19 Minami Tusukushino E-mail: ishikawa@kitasato-u.ac.jp
Machida-shi, Tokyo 194-0002 Japan

Medical School Attended: Tohoku University
Residency: Tokyo University

Board Certifications: MD, Ph.D., Fellow of AAEHF
Current Faculty Appointments: Professor of Environmental Medicine. Neuro-Ophthalmology
Current Job Description: Director of Clinical Environmental Health Center, Kitasato Hospital, Ketasato Institute.
Other Information: 1994 - 1998 Dean of Kitasato University, School Of Medicine

Mikio Miyata, M.D.
Current Job Description: Chief of Clinical Environmental Health Center, Kitasato Hospital, Kitasato Institute.

Ko Sakabe, M.D.
Current Job Description: Vice Director of Clinical Environmental Health Center, Kitasato Hospital, Kitasato Institute.

Disclosure Statement: None

SPEECH TITLE: **"Neurological Manifestations Due to Low Dosage Exposure of Organophosphorus Compounds"**

The information below has been provided by the speaker.

1.) Goals and objectives: Analyses of clinical manifestations in patients with chronic low dosage exposure to organophophorus (OP) compounds

2.) Outline of talk/abstract: Pupil and eye movement especially ocular smooth pursuit movement are the most easy, and less expensive method to detect neuro-toxicity of the patients with organophosphorus compounds. One of the major compounds is fenitrothion as a pesticide and chlorphyrifos in ant-termite in Japan.

3.) Conclusion of what is to be learned: Improvement after the use of antidotes in patients with OP will be demonstrated.

4.) References:
Shirakawa S, Rea W, Ishikawa S, et al.: Evaluation of the autonomic nervous system response by pupillographical study in the chemically sensitive patients. Environmental Medicine 8:121-127, 1991.

SUNDAY, JUNE 10, 2001 FACULTY

Pierre L. Auger, M.D.
Clinique De Santé Au Travail et De Santé Environnementale
Montreal, Quebec, Canada
514/849-5201

Professor Michel Bounias
University of Avignon, France
BioMathematic's and Toxicology Unit,
Chemin du Petit Bosquet
Saint-Christol D'Albion F-84390, France
011 33 490 750 888

Bertie Griffiths, Ph.D.
Environmental Health Center-Dallas
Dallas, TX 75231
214/368-4132

Kaye H. Kilburn, M. D.
University of Southern California Medical Center
Keck School of Medicine
Los Angeles, CA 90033
323/442-1830

Eva Millqvist, M.D.
Allergy Centre, Department of Respiratory Medicine
Sahlgrenska University Hospital
Gothenburg, Sweden
011/46-31-3423635

Ko Sakabe, M.D.
Clinical Environmental Health Center,
Kitasato Hospital, Kitasato Institute.
Machida-shi, Tokyo, Japan
81/427-95-5784

NINETEENTH ANNUAL INTERNATIONAL SYMPOSIUM ON MAN AND HIS ENVIRONMENT

SCHEDULE FOR SUNDAY, JUNE 10, 2001

8:15 ANNOUNCEMENTS/MODERATOR: Satoshi Ishikawa, M.D.

8:30 Eva Millqvist, M.D. "A Capsaicin Cough Test in Patients with Multiple Chemical Sensitivity"
8:50 Q & A

9:00 Kaye H. Kilburn, M. D. "Performance Testing for Neurotoxicity"
9:20 Q & A

9:30 Professor Michel Bounias, Ph. D.,"NeuroBioFeedback, Mathematics and Clinical Neurotoxicology"
9:50 Q & A

10:00 BREAK WITH EXHIBITORS

10:30 Pierre L. Auger, M.D. "Health Effects of Indoor Fungi Environmental Evaluation, Remediation"
10:50 Q & A
11:00 Ko Sakabe, M.D. "Challenge Test of Toluene and Formaldehyde in Patients "
11:20 Q & A

11:30 Bertie Griffiths, Ph.D. Title Currently Unavailable
11:50 Q & A

12 Noon SUMMARY AND CLOSE: Ronald Finn, Ph.D.

SUNDAY, JUNE 10, 2001

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

Eva Millqvist, M.D. Date of talk: Sunday, June 10, 8:30 a.m.

Allergy Centre, Department of Respiratory Medicine Phone: 011/46-31-3423635
Sahlgrenska University Hospital Fax: 011/46-31-417824
Gothenburg, S-413 45, Sweden E-mail: eva.millqvist@medfak.gu.se

Medical School Attended: Karolinska Institute, Stockholm, Sweden
Residency: Gothenburg
Board Certifications: M.D., Ph.D.
Current Faculty Appointments: N/A
Current Job Description: Research in Asthma and Asthma like Conditions. Working with patients sensitive to strong scents and chemicals.
Other Information: Working with developing a new method to test chemical sensitivity.

Disclosure Statement: None

SPEECH TITLE: **"A Capsaicin Cough Test in Patients with Multiple Chemical Sensitivity"**

The information below has been provided by the speaker.

1.) Goals and objectives: To find an objective method in diagnostics of Multiple chemical sensitivity (MCS), also known as "idiopathic environmental intolerance (IEI)"

2.) Outline of talk/abstract: Twelve patients, with respiratory symptoms and fulfilling Cullen's definition of MCS, were provoked with inhaled capsaicin in increasing concentrations. Capsaicin,

the pungent ingredient in red pepper, is known to stimulate coughing via the unmyelinated slow C-fibres of the sensory nervous system.

3.) Conclusion of what is to be learned: Patients with airway symptoms fulfilling Cullen's definition of MCS showed a sensory hyperactivity according to a standardized cough capsaicin provocation. A capsaicin test may be an objective way to diagnose MCS in patients with respiratory symptoms.

4.) References:

1. Millqvist E, Löwhagen O. Placebo-controlled challenges with perfume in patients with asthma-like symptom. Allergy 1996;51:434-9.
2. Millqvist E, Bende M, Löwhagen O. Sensory hyperreactivity - a possible mechanism underlying cough and asthma-like symptom. Allergy 1998;53:1208-12.
3. Millqvist E, Bengtsson U, Löwhagen O. Provocations with perfume in the eyes induce airway symptoms in patients with sensory hyperreactivity. Allergy 1999;54(5):495-9.

Abstract Information & Notes

Kaye H. Kilburn, M. D. Date of talk: Sunday, June 10, 9:00 a.m.

University of Southern California Medical Center Phone: 323/442-1830
Keck School of Medicine Fax: 323/442-1833
2025 Zonal Ave., CSC-201 E-mail: kilburn@usc.edu
Los Angeles, CA 90033

Medical School Attended: University of Utah College of Medicine
Residency: University of Utah Hospitals
Board Certifications: California, Louisiana, North Carolina, Missouri, Wyoming, New York
Current Faculty Appointments: Professor of Medicine, University of Southern California Keck School of Medicine
Current Job Description: Director of Environmental Sciences Lab, Ralph Edgington Professor of Medicine, University of Southern California - Keck School of Medicine
Other Information: Editor-in-Chief, Archives of Environmental Health and President & Director, Neuro-Test, Inc.

Disclosure Statement: None
SPEECH TITLE: **"Performance Testing for Neurotoxicity"**
The information below has been provided by the speaker.

1.) Goals and objectives: Understand what tests are useful to detect effects of chemicals, how they are performed and how data is calculated and interpreted.

2.) Outline of talk/abstract: Abstract provided

3.) Conclusion of what is to be learned: Sway speed tests balance, simple & choice visual reaction times measure processing speed, color discrimination and visual fields scan vital functions for effects of chemicals. Comparison of groups is improved by adjusting measurements for age, sex, education and other factors and individual comparisons have better discrimination to develop profiles of effects of many chemicals.

4.) References: Kilburn KH, Thornton JC & Hanscom BE: A field method for blink reflex latency (BRL R-1) and prediction equations for adults and children. Electromyography Clin Neurophysiol, 38:25-31, 1998.
Kilburn KH, Thornton JC & Hanscom BE: Population based prediction equations for neurobehavioral tests. Arch Environ Health, 53:257-263, 1998
Kilburn KH: Chemical Brain Injury, New York, John Wiley & Sons, Inc. 1998.

PERFORMANCE TESTING FOR NEUROTOXICITY

Kaye H. Kilburn, M.D.
University of Southern California
Keck School of Medicine
2025 Zonal Avenue, CSC 201
Los Angeles, CA 90033
E-mail: kilburn@usc.edu

ABSTRACT

Biological effects on the brain are measured with function tests. For individual subjects measures of performance are more sensitive than the relatively crude clinical neurological examination, than anatomy, by imaging: CT and MRI, PET and SPEC and by histopathology. Changes in concentrations of substances in blood and serum are not helpful. Comparisons of functions to the predicted normals are useful.

Extending this strategy applies the same tests to populations, comparing mean values for groups by standard statistical methods. Attempting to detect individual differences by comparing them to a range of normal values for each test is impossibly insensitive. Sensitivity can be improved by predicting a value for the individual for each test, calculating the 95% confidence interval and considering as abnormal any value outside this interval that gives it a less than 5% possibility of occurring by chance.

Physiological measures to detect abnormal balance, reaction time, color and visual fields are most useful. They profile impaired brain function when coupled with verbal recall (memory), digit symbol substitution, problem solving, connecting numbers and letters to make trails and vocabulary contrasted with long-term memory, information, picture completion and similarities.

Kilburn, KH: Chemical Brain Injury. New York: John Wiley and Sons, 1998.
Feldman, R: Occupational and Environmental Neurotoxicology. Philadelphia: Lippincott-Raven,1999.

TABLE 1
FLIGHT ATTENDANTS TESTING

Exposed (33) Unexposed(202) p			
Age years	47.7 6.9	45.0 21.1	.76
Education Level years	14.3 1.9	12.9 2.3	.0001
Psychological-Mental			
Profile of Mood States Score	52.1 40.7	21.0 31.6	.0001
Symptom Frequencies	5.0 1.4	2.6 1.2	.0001
Percent Predicted			
Balance Eyes Closed	101.6 4.1	100.0 2.5	.003
Grip Strength Left	91.3 20.7	99.1 17.5	.023
Color score Right	82.7 47.0	102.6 51.1	.038
Left	70.9 36.6	102.6 51.1	.0008

TABLE 2
ABNORMAL TESTS IN FLIGHT ATTENDANTS

Abnormalities	**Patients**
>14	2
6-8	4
3-5	9
2	6
0-1	12

TABLE 3

PULMONARY FUNCTION TESTS OF EXPOSED SUBJECTS AS PERCENT PREDICTED COMPARED WITH UNEXPOSED SUBJECTS

	Exposed Mean±/Sd	**Unexposed Mean±Sd**	**p Value**
Pulmonary Function Tests			
FVC % of pred	105.9±8.8	101.6±15.1	.118
FEV_1 % of pred	101.1±9.0	93.6±15.8	.009R
FEF_{25-75} % of pred	94.6±9.0	88.1±35.0	.305
FEF_{75-85} % of pred	74.7±22.8	78.1±52.7	.715
FEV_1/FVC	77.0±4.6	72.8±9.5	.013R

R = exposed values better than controls

Abstract Information & Notes

Professor Michel Bounias Date of Talk: Sunday, June 10, 9:30 a.m.

University of Avignon, France Phone: 011 33 490 750 888
BioMathematic's and Toxicology Unit, Fax: same
Chemin du Petit Bosquet E-mail: n/a
Saint-Christol D'Albion F-84390, France

Medical School Attended: University of Lyon: INSA School of Engineers; Faculty of Sciences 3rd cycle
Residency: Same as above
Board Certifications: Docteur d'Etates Sciences/DEA (3rd cycle) in Biometrics/Engineer in Biochemistry
Current Faculty Appointments: Professor, Director of Research (Nat'l. Officer), Ministerial Expert
Current Job Description: 1.) Theoretical research: Mathematical foundations of existence of physical universe and biological systems, up to brain and conscious perception functions. 2.) Toxicology (from molecular to Planetary levels): Applications of Mathematical theory.
Other Information: About 400 scientific papers published, 10 books authored

1.) Elaboration of a new deontology for the management of Planet Earth, foundations of a chart of objective rights of all living organisms.
2.) Editorial Activity (CoEditor and chief advisor of Scientific Journals)
3.) Musicologist and Composer
4.) Symphonies, lieders for soprano, etc.)

Disclosure Statement: None

SPEECH TITLE: **"NeuroBioFeedback, Mathematics and Clinical Neurotoxicology"**
The information below has been provided by the speaker.

1.) Goals and objectives: Examination of various neurotoxicological situations and the ways how NeuroBioFeedback could be concerned.

2.) Outline of talk/abstract: Mental imaging constructed from both external and internal signals can be impaired by neurotoxic factors. Even a weak neuronal configuration change can alter the correspondence of a mental image with its associated homeostatic processes. A complex array of interactions connects molecular intoxication and physiological processes with brain activity and sensorial perceptions. A cascade of consequences involves ultralow dose-effects and synergistic damage.

3.) Conclusion of what is to be learned: No standard threshold can be expected in chemical toxicity, and causes may be hidden or undetectable. However, identification and removal of pollutant residues and stimulation of natural defenses should be integrated with treatments.

4.) References:

1. Wago, 1994, Med. Philos.13, 257.
2. Oesch et al., 2000, Toxicol. Pathol. 28, 382.
3. de Zeeuw, 1998, Toxicol. Lett. 102/3, 103.
4. Weiss, 1994, Neurotoxicology 11, 305.
5. Bauman et al., 1997, Therapie 52, 607.
6. Bounias, 1990, J. Enzyme Inhibition 3, 323. etc. (23 ref.)

NEUROBIOFEEDBACK, MATHEMATICS AND CLINICAL NEUROTOXICOLOGY

M. Bounias
Research Director and Professor, University of Avignon and INRA-DSPE,
BioMathematics & Toxicology Unit
Chemin du Petit Bosquet, F-84390
Saint-Christol d' Albion, France
(corresponding author: (33) 490 750 888)

RE. Laibow, MD and Maj. Gen. A. Stubblebine
The Alexandria Institute of Natural and Integrative Medicine
10, Old Post Rd Sth
Croton-on-Hudson, New York 10520

Summary: NBF is founded on anticipatory properties of mental imaging. Control loops associate

brain receptors as endpoints of molecular functioning to mental images which in turn drive back to homeostatic regulation. This mechanism is reflected in category theory. False mental images are associated with disease and neurotoxicology emerges from a complex array of both environmental and endogenous causes and process.

Introduction and preliminaries. NeuroBioFeedback (NBF) is founded on directed anticipatory mental imaging[10-11]. During this process, the brain fits the homeostatic regulation of the body to EEG-piloted instructions for restored mental images of molecular processes. The present work aims at examining the implications of these phenomena in neurotoxicology.
Mathematical background. Mental imaging is mathematically supported by fixed points standing for stable convergence points of sequences of neuronal chaining configurations[9,12]. In neurotoxic situations, a correct fixed point $f(a)=a$ is falsified into $f(a')=a'$. Parameter a' is either no longer representing a physiological reality or it stand for a wrong state. Thus, NBF targets to turn back a' into a"a. Interactions of molecular systems constitute mappings which in turn are translated in the brain into mappings of mental images. Theses processes denote mathematical morphisms. (Mor) which are able to account for recognition patterns[71]. Endogenous structures correspond to isomorphisms and automorphisms while exogenous to endogenous mappings involve homomorphisms and endomorphisms.

Immunological endpoints. Immunity differentiates between morphisms, and keeps relations between morphisms and biological components, which constitute "Categories". Neurotoxicity may thus impair these processes through confusion of categories.

Neurotoxicity Sites and Expression.
Endogenous receptors. Brain receptors are associated with both conscious mental processes:cognition, emotion, anxiety, fear, hunger, thirst, taste[5,16,20,22,38,40,41,49,51,61,67,69] and unconscious regulatory systems: DNA transcription, protein kinase induction, sensorial nerve transmission[14,18,19,55]. Reciprocally, CNS-directed control of body functions is impaired by stress and neurotoxicants[34].

Alterations in morphisms of objects and mental images. Receptors are involved in diseases[66] with EEG traces[42] reflecting feedback neuronal interactions[32]. Very complex interactions of environmental factors with neurotoxicity have been reported, for instance in Transmissible Spongiform Encephalopathies (TSE)[15,54,56,65]. Adding to cancer observations[54], this invalids using of standard threshold exposure[45].

Antinomies and neglect in neurotoxicity evaluation.
The body burden of pollutants and causality assessment. Reducing the exposure to toxicants is a major goal[52] and the NHEXAS has started a survey of human body pollution[35], while such data are generally lacking because of cost[25]. Ultra-low dose effects[72], indirect immune effects[9] and delayed disease emergence, and synergistic effects[53] can mask causality factors. However, by-products may denote chemical injury, like for autism[28] and recent receptor interaction functional changes have been found[59]. Individual susceptibility to neurotoxicants is emphasized by fluctuations and polymorphism in detoxification systems[27,37] and attention is called indirect (e.g. maternal) exposure[2,64], However, neurotoxicity remains underestimated[17] and biphasic responses[8] are poorly addressed.

Complexity and confusion is neurotoxicity risk evaluation. Real causality has been masked by claims about confounding factors, phobia[46] and overestimation of risk by the public[63]. However, drugs induce real neurotoxicity[30], psychostress produce damaging effects[3] while ultra low doses of chemicals produce neurobehavioral, troubles that just suggestion does not

produce[9,12,57,72]. Furthermore, some neurological targets respond to a toxicant while others do not[75], so that a negative test does not mean absence of toxicity. Reciprocally, an apparent remedy may be deleterious, like organophospates (OP) proposed as cholinergic hypofunctional agents against age-related cognitive impairment[56] while (OP) pesticides produce very dangerous effects[9,24].

Endogenous neuroprotection and neurotoxicants.
Endogenous neuroprotectants. CNS repair and protection from neurotoxicants is achieved by astrocytes[4] and microglia[43], by melatonine[31], Dopamine D2 receptors[14], GABA agonists[19] and L-arginine via cholinergic and NO receptors[33], Glutamate itself protects hippocampal cells against NMDA-induced death at physiological levels[1] though it becomes toxic at higher doses.

Endogenous neurotoxicants. Glu-degrading enzymes prevent Glu neurotoxicity[48]. But dopamine[72] and L-cysteine[39] can also be neurotoxic while melatonine could enhance the transfer of toxic manganese to the brain through glucocorticoid stimulation[60].

Conclusions. Targets and pathways of neurotoxicity are so numerous and complex (from behavioral to molecular and physiological aspects) that detection of causes and chemical management may be problematic. NBF is appropriate in an integrative treatment including residue elimination and enhancement of natural defenses.

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[1]Adamchik, Y., et al., 2000. Neuroscience (Oxford), 99(4), 731-736. [2]Andersen, H., et al., 2000 Toxicology, 144(1-3), 121-127. [3]Andrew, H., et al., 2000 Breast Cancer Res. Treat., 59(3), 199-209. [4]Aschner, M.., 2000. Neurotoxicology, 21(1-2), 175-180. [5]Ashton, C., et al., 2000 Psychopharmacology (Ber), 152(1), 87-92. [6]Bauman, P., et al., 1997. Therapie, 52, 607-608. [7]Benesova, O., et al., 1999. Gen. Physiol. Biophys., 18 (focus issue). 21-27. [8]Bounias, M.., 1990. J. Enzyme Inhibition, 3, 323-326. [9]Bounias, M., 2000. Treatise of General Toxicology (Springer-Verlag, Paris, Berlin). (See Section IV, Physiological Targets, 91-98, for review). [10]Bounias, M., 2000 CASYS'99 Int. Math. Conf. Liege, Belgium (D.M. Dubois ed.). Amer. Inst. Phys. CP517, 233-243. [11]Bounias, M., et al., 2001, CASYS2000 Int. Math. Conf. Liege, Belgium (D.M. Dubois ed.). Amer. Inst. Phys. Conf. Proc. in press, 15 pp. [12]Bounias, M., et al., Toxicol. Lett., 1995, 80, 19-24, [13]Bounias, M., et al., 1997, BioSystems 42, 191-205. [14]Bozzi, Y., et al., 2000 J. Neurosci., 20(22), 8643-8649. [15]Buzard, G., et al., 2000 J. Environ. Pathol. Toxicol, Oncol., 19(3), 179-199. [16]Caicedo, A., et al., 2000. J. Neurosci. 20(21), 7978-7985. [17]Chakrabarti, S., 2000 Pharmacol., Biochem. Behav., 66(3), 523-532. [18]Chen, J., et al., 2000. Neuropharmacology, 39(12), 2231-2243. [19]Chen Xu, W., et al., 2000 Brain Res., 874(1). 75-77. [20]Cleton, A., et al., 2000 130(5), 1037-1044. [21]Cobb, S., et al., 2000 Neuropharmacology, 39(11), 1933-1942. [22]Cordero, I., et al., 1998. Behav.. Neurosci., 112(4), 885-891. [23]Cox, C., et al., 2000. Proc. Natl. Acad. Sci. (USA), 97(17), 9724-9728. [24]Crinnion, W., 2000. Alternative Medicine, 5(5), 432-447, [25]de Zeeuw, R., 1998, Toxicol. Lett 102/103, 103-108. [26]Dube, M., et al., 2000 Peptides (NY), 21(6), 793-801. [27]Eaton, D., 2000 Neurotoxicology, 21(1-2), 101-111. [28]Edelson, S., et al., 1998. Toxicol. Indus. Health, 14(4), 553-563. [29]El-Missiry, M., et al., 2000 J. Biochem. Mol. Toxicol., 14(5), 238-243, [30]Fleckenstein, A., et al., 2000. Eur. J. Pharmacol., 406(1), 1-13. [31]Franceschini, D., et al., 1999 Adv. Exp. Med. Biol., 467, 207-215. [32]Freeman, W., 1975. Mass action in the nervous system. Academic Press, New York. [33]Giardino., L., et al., 2000 Neuroreport, 11(8), 1769-1772. [34]Hajnal, A., et al., 2000 Physiol. Behav,, 70(1-2), 95-103. [35]Hogue, C., 2000. Chem & Engin. News, 78(3), 28-29. [36]Hughes, J., et al., 2000. Psychopharmacology (Ber), 152(1), 119-121, [37]Ihara., H., et al., 1999 Ann. Clin. Biochem., 36(3), 347-352. [38]Izumi, T., 1998. Hokkaido Igaku Zasshi, 73(5), 463-473. [39]Janaky, R., et al.,

2000, Neurochem. Res., 25(9/10), 1397-1405. [40]Kajdaniuk, D., et al., 2000 Pathophysiology, 7(1), 47-51. [41]Kesslak, P., et al., 1998, Behav. Neurosci., 112(4), 1012-1019. [42]Kim, C., et al., 1996. Korean J. Pharmacol. 32(3), 293-300. [43]Kim, W-G, et al., 2000, J. Neurosci., 20(16), 6309-6316. [44]Kovalev, G., et al., 2000. Eksp. Klin. Farrmakol., 63(1), 3-6. [45]Kroes, R., et al., 2000 Food Chem. Toxicol., 38(2/3), 255-312. [46]Lees-Haley, P., 1995. Toxicol. Lett., 82/83, 197-202. [47]Lehotzky, K., 2000, Cent. Eur. J. Public Health, 8(suppl.), 55-56. [48]Matthews, C., et al., 2000. J. Neurochem., 75(3). 1045-1052. [49]Mirza, N., et al., 2000, Psychopharmacol. (Ber), 148(3), 243-250. [50]Moret, C., et al., 2000 Eur. J. Pharmacol., 404(1-2), 1-12. [51]Muller, M., et al., 2000, Endocrinology 141(11), 4262-4269. [52]N.I.E.H. (Anonymous from Res. Tr. Pk.), 2000 Mutat. Res. 462(2-3), 407-421. [53]Noraberg, Jens, et al., 2000, Neurotoxicology, 21(3), 409-418. [54]Oesch, F., et al., 2000, Toxicol. Pathol., 28(3), 382-387. [55]Okubo, Y., et al., 1998. J. Biol. Chem., 273(40), 25961-25966. [56]Omote, K., et al., 2000. Anesthesiology, 93(1), 173-178. [57]Osterberg, K., et al., 2000. Scand. J. Work, Environ. Health 26(3), 219-226. [58]Overstreet, D., 2000, Neurotoxicology 21(1-2), 75-81. [59]Petterson, K., et al., 2000. Oncogene, 19(43), 4970-4978. [60]Purdey, M., 2000, Medical Hypothesis, 54(2), 278-306. [61]Raber, J., et al., 2000, J. Neurosci., 20(5), 2064-2071. [62]Rameau, G., et al., 2000, Neuropharmacology, 39(12), 2255-2266. [63]Ray, D., 2000, Neurotoxicology, 21(1-2), 219-221. [64]Rice, J., et al., 2000, Toxicol. Pathol. 28(1), 202-214. [65]Sahoo., A., et al., 2000, 39(1), 7-12. [66]Schafer et al., 1983. J. Lab. Clin. Med., 102, 870-880. [67]Shibata, K., et al., 1999. Neuropeptides (Edinburgh), 33(6), 503-509. [68]Shin, H., et al., 1999, Korean J. Physiol. Pharmacol. 3(3), 237-244. [69]Tarazi, F., et al., 1999. Mol. Psychiatry, 4(6), 529-538. [70]Tilson, H., 2000, Toxicol. Pathol. 28(1), 149-156. [71]Wago, H., 1994, Med. Philos, 13(4), 257-263. [72]Wan, F-J, et al., 2000 Chinese J. Physiol. (Taipei), 43(2), 69-74. [73]Wang, Y-Q., et al., 2000 Neurosci. Lett. 290(3), 193-196. [74]Weiss, B., 1994. Neurotoxicology, 11,305-314. [75]Zajaczkowski, W., et al., 2000 Neurotoxic Res., 1(4), 299-310.

Abstract Information & Notes

Pierre Auger, M.D. Date of talk: Sunday, June 10, 10:30 a.m.

Clinique De Santé Au Travail et De Santé Environnementale Phone: 514/849-5201
3666 St-Urbain Fax: 514/731-0668
Montreal, Quebec H2X 2P4 Canada E-mail: pierrelauger@qc.aira.com

Medical School Attended: Laval University
Residency: Toronto-Quebec, Paris-London
Board Certifications: Occupational Medicine (Royal College)
Current Faculty Appointments: Adjoint Proffessor McGill
Current Job Description: Occupational Environmental Medicine Practice - Clinical Practice and Preventive Medicine.

Disclosure Statement: None

SPEECH TITLE: **Health Effects of Indoor Fungi Environmental Evaluation, Remediation**

The information below has been provided by the speaker.
Authors: Pierre L. Auger, MD, Louis Patry, MD, Marie-France Pinard PhD., Claude Mainville Ing.

1.) Goals and Objectives: Provide a summary of the health effects of molds in indoor air problems, a critical overview of sampling and analytical methods and of the basic principles of remediation.

2.) Outline of talk/abstract: Fungi are organisms that can produce spores, mycelia and organic compounds that can become harmful to humans. They are ubiquitous. The health effects can be divided in six categories: imitative and non-specific symptoms, allergic diseases, respiratory infections, alveolitis and Organic Dust Toxic Syndrome (ODTS), chronic bronchitis, and effects of mycotoxins. The exposure assessment should be done by medical and/or epidemiological investigation, visual and engineering inspections and finally if necessary by monitoring culturable or total spores in the air, settle dust, surface or bulk material or liquids sampling. Serum or urine sampling is possible but not usually available and difficult to interpret. As regard remediation, in all situations causes of water accumulation must be rectified. Four level of contamination have been devised: level 1 (less than 10 sq. ft. or 3 meter^2), level 2 (10-30 sq. ft. or 3-10 m^2), level 3 (30-100 sq. ft. or 10-30 m^2). Level 4 over 100 sq. ft. or 30 m^2). Short case reports will be presented.

3.) Conclusion of what is to be learned: Health effects of indoor fungi are multiple. Environmental measurements must be used with caution, as interpretation of results can be difficult. Proper meticulous remediation is the first important step in alleviating the effects of exposure to toxicogenic molds.

4.) References:

1. Husman T, Health Effects Indoor - Air Microorganisms Scandinavian J, Work Environ. Health 1996 22: 5-13
2. Husman T., Health Effect of Microbes Proceedings of Healthy Building 2000, Volume 3 p. 13-24
3. New York City, Department of Health 1999 Guidelines on assessment and remediation of fungi in indoor environments www.ci.nyc.ny.us./html/doh/html/epi/moldrpt1.html
4. Pasanen AL Evaluation of Indoor Fungal Exposure Proceedings of Healthy Building 2000, Volume 3 p. 25-38
5. Rylander R., Megevand F. Environmental Factors for Respiratory Infections. Arch Environ Health 2000, 55, 300-303
6. Shaughnessy R., Morey PR, Remedial of microbial contamination in Bioaerosols: assessment and control ed. ACGIH, 1999 chapter 15, pp 1-17

Abstract Information & Notes

Speakers Name: **Ko Sakabe, M.D.,** Date of talk: Sunday, June 10, 11:00 am
Satoshi Ishikawa, M.D., and Mikio Miyata, M.D.

Ko Sakabe, M.D.
Current Job Description: Vice Director of Clinical Environmental Health Center, Kitasato Hospital, Kitasato Institute.

Satoshi Ishikawa, MD.

Clinical Environmental Health Center, Kitasato Hospital Phone: 81/427-95-5784
The Kitasato Institute Fax: 81/427-99-2287
4-5-19 Minami Tusukushino E-mail: ishikawa@kitasato-u.ac.jp
Machida-shi, Tokyo 194-0002 Japan

Medical School Attended: Tokyou University
Residency: Tokyo University
Board Certifications: MD, Ph.D., Fellow of AAEHF
Current Faculty Appointments: Professor of Environmental Medicine. Neuro-Ophthalmology
Current Job Description: Director of Clinical Environmental Health Center, Kitasato Hospital, Ketasato Institute.
Other Information: 1994 - 1998 Dean of Kitasato University, School Of Medicine

Mikio Miyata, M.D.
Current Job Description: Chief of Clinical Environmental Health Center, Kitasato Hospital, The Kitasato Institute.

Disclosure Statement: None

SPEECH TITLE: **"Double Blind Challenge Test of Toluene and Formaldehyde in Patients with Chemical Sensitivity(cs) and Controls Especially Method of the Diagnosis Due to Objective Methods"**

The information below has been provided by the speaker.

1.) Goals and objectives: Establishment of diagnosis in patients with chemical sensitivity (CS) using objective methods within the clean room by removing the masking effect.

2.) Outline of talk/abstract: Patients with CS reacted, for example, in formaldehyde as low as 8ppb and this was demonstrated by change in blood circulation of the brain detected by Near Infrared Oxygen Monitoring (NIRO) method. The results accord with other tests such as eye tracking test as well as psychological test.

3.) Conclusion of what is to be learned: Those tests were significant to establish the diagnosis in patients of CS with low dosage exposure of chemicals.

4.) References:
Ishikawa S et al: Chemical sensitivity and its clinical characteristics in Japan.
Asian Medical Journal 43:7-15,2000

Abstract Information & Notes

Bertie Griffiths, Ph.D. Date of talk: Sunday, June 10, 11:30 a..m.

Environmental Health Center - Dallas Phone: 214/368-4132
8345 Walnut Hill Lane, Suite 220 Fax: 214/691-8432
Dallas, TX 75231

Medical School Attended: Graduate of the University of Wisconsin and University of the West Indies, Faculty of medicine.
Residency:
Board Certifications:
Current Faculty Appointments: Professor and Consultant in Microbiology and Infectious Diseases
Current Job Description: Director of EHC-D Laboratory

Disclosure Statement: None

Other Information: Recipient of Degrees in microbiology, virology, postdoctoral training in Infectious Diseases and Immunology. Rockefeller fellowship to study Entomology and Virus epidemiology in Brazil and Trinidad.

SPEECH TITLE: Title not available at time of printing.

The information below has been provided by the speaker.

1.) Goals and objectives:

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:

4.) References: