20th Annual International Symposium
on
Man and His Environment in Health and Disease

Special Focus

Innovative Aspects and Treatment of Chronic Disease, Nutrition and Chemical Sensitivity

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 2002 conference will focus on "Innovative Aspects and Treatment of Chronic Disease, Nutrition and Chemical Sensitivity". For this year's conference, we have assembled a faculty of top international experts for you. This Conference presents the most current information available while providing guidelines to identify, diagnose, treat and to prevent environmentally triggered responses in the body.

GOALS OF THE MEETING

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

OBJECTIVES OF THE MEETING

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

INTENDED AUDIENCE

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

EDUCATIONAL FORMATS

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

CONFERENCE FORMAT

The AEHF Committee has selected some of the leading experts in the fields of chronic disease, nutrition and chemical sensitivity.
Each speaker's presentation will last approximately 20 minutes and will be followed by a 10 minute question and answer session. All speakers are encouraged to use any and all appropriate audio/visual aids. (A brief outline of the speech is included in this booklet.)

GIVEN IN COOPERATION

William J. Rea, M.D., F.A.C.S.
Symposium Chairman, American Environmental Health Foundation,
Environmental Health Center - Dallas,
Dallas, Texas

Bertie B. Griffiths, Ph.D.,
Environmental Health Center – Dallas
Dallas, Texas

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

20TH ANNUAL INTERNATIONAL SYMPOSIUM
ON
MAN & HIS ENVIRONMENT

SCHEDULE
Thursday, June 6, 2002

8:00 a.m. REGISTRATION

9:00 WELCOME: William J. Rea, MD

9:30 Douglas Seba, Ph.D., "Environmental Update 2002: Biochemical Cultural Terrorism"

9:50 Q & A

10:00 BREAK WITH EXHIBITORS

MODERATOR: Wallace Rubin, M.D.

10:30 Riki Ott, Ph.D., "Exxon Valdez Oil Spill Legacy (EVOS): Shifting Paradigms in Oil Ecotoxicology"

10:50 Q & A

11:00 Kaye H. Kilburn, M.D., "Arguments for One Brain Disorder from Chemicals"

11:20 Q & A

11:30 William J. Rea, M.D., "Weather Pattern Monitoring" & "Lipid-Soluble and Water-Soluble Antioxidant Profiles Measured in Chemically Sensitive Patients and Controls"

11:50 Q & A

12:00n BUFFET LUNCH WITH THE EXHIBITORS

MODERATOR: Kalpana D. Patel, M.D.

1:30 Russel J. Reiter, Ph.D, "Melatonin: Antioxidant par Excellence"

1:50 Q & A

2:00 C. Chan Gunn, M.D., "The Importance of Good Connections"

2:20 Q & A

2:30 Colin H. Little, M.D., T Cell Antigen Binding Molecules "TABM in Food and Chemical Sensitivity"

2:50 Q & A

3:00 BREAK WITH EXHIBITORS

3:30 Allan D. Lieberman, M.D., "Beauticians Beware/Disease in the Beauty Salon"

3:50 Q & A

4:30 ROUNDTABLE: Marsha Vetter Ph.D., M.D., Allan D. Lieberman, M.D., Rima E. Laibow, M.D.

6:00 ADJOURN

THURSDAY, JUNE 6, 2002
ABSTRACTS
AND
HANDOUTS

Abstract Information & Notes

Douglas Seba, Ph.D. Date of talk: Thursday, June 6, 2002 9:30am
Phone: 703/949-1055
P.O. Box 1417, #323 Fax: n/a
Alexandria, VA 22313 E-mail: n/a
Medical School Attended: University of Miami
Major and date of Graduation: Ph.D. in Environmental Oceanography from University of Miami, 1970
Residency: n/a
Board Certifications: n/a
Current Faculty Appointments: None
Current Job Description: Independent Marine Scientist
Other Information: 35 years experience with chemicals in the environment, especially their transport and fate
Disclosure Statement: None
SPEECH TITLE: **Environmental Update 2002: Biochemical Cultural Terrorism**
The information below has been provided by the speaker.
1.) Goals and objectives: To understand the very essence of this conference by reviewing major environmental phenomenon that leaves us vulnerable to Biochemical Terrorism
2.) Outline of talk/abstract: Genetics, Xenobiotics, fate and transport mechanisms, and increasing global dust will be reviewed for contemporary aspects
3.) Conclusion of what is to be learned: That adverse health effects can occur at vast distances from their environmental origin and thus place environmental physicians and patients in a constant state of Biochemical Terrorism.
4.) References: Drawn from a misc of media, web sites and scientific publications relevant to the current time line.

ENVIRONMENTAL UPDATE 2002: BIOCHEMICAL CULTURAL TERRORISM

Douglas Seba

The xenobiotic chemical culture that pervades modern society is one of the root causes of the evolution of environmental medicine and in particular the two decades of this conference. The events of 9/11 and subsequent bioterrorism are said to be seminal turning points in our history, and in one sense they are, in that the rest of society has learned to live with the same fear that the chemically sensitive patient has for the last two decades. A single spray of pesticide or whiff of perfume can be just as disabling to the chemically sensitive person as the hell of a bioweapon attack.

A few select samples of contemporary issues of the last year that illustrates these precepts will be presented including bioweapons, proteomics, and the first national stream reconnaissance for pharmaceuticals and hormones http://toxics.usgs.gov/pubs/ofr-02-94/index.html).
Equally important are changes in the political/legal landscape of xenobiotic chemicals such as changes in the definition of toxic dose or disability as well as the use of human trials for pesticides.

The fate and transport of endocrine disruptors from distant sources and mechanisms of re-concentration are continuing research interests of the presenter. Hormones are really the messengers of life and their disruption portends drastic effects on our eco sphere. Recent research will be reviewed.
The environmental scientists and physicians at this conference are probably the most qualified to help chemically sensitive patients to cope with daily exposures to xenobiotics, which, for them, are

equivalent to petite, individual acts of biochemical terrorism. By extension, these same professionals would be the most informed and capable to assist in incidents of true bioterrorism.

Abstract Information & Notes

Riki Ott, Ph..D. Date of talk: Thursday, June 6, 2002, 10:30am
Alaska Forum for Environmental Responsibility Phone: 907/424-3915
P.O. Box 1430 Fax: 907/424-3926
Cordova, AK 99574 E-mail: otter2@ctcak.net
Medical School Attended: Graduated with a doctorate from School of Fisheries, University of Washington, Seattle, WA. Took classes at UW School of Public Health.
Major and date of Graduation: Marine Toxicology, 1986
Current Faculty Appointments: Faculty mentor with Columbia Pacific University, Novato, California
Current Job Description: Environmental consulting and writing. Currently writing a book about the *Exxon Valdez* Oil Spill legacy and its public policy ramifications.
Other Information: A commercial fisherm'am, from 1985-1994, in Area E, Prince William Sound and the Copper River Delta, Alaska. Co-founder of Oil Reform Alliance, the Alaska Forum for Environmental Responsibility in 1994, the Copper River Watershed Project, and the Oiled Regions of Alaska Foundation.
Disclosure Statement:
SPEECH TITLE: **Exxon Valdez Oil Spill Legacy (EVOS): Shifting Paradigms in Oil Ecotoxicology**
The information below has been provided by the speaker.

1.) Goals and objectives: Demonstrate a shifting paradigm in ecotoxicity of crude oil following the *Exxon Valdez* Oil Spill (EVOS) by presenting an overview of acute, indirect, and chronic impacts of this spill to natural resources in Prince William Sound, Alaska.

2.) Outline of talk/abstract: Compare old and new paradigms in oil ecotoxicity. 1970s paradigm: focus on BTEX (benzene and its derivatives) with short-term acute impacts on fish and wildlife. 1990s paradigm: focus on PAHs (polycyclic aromatic hydrocarbons) with long-term, chronic, population-level effects on fish and wildlife.

Review history of paradigm shift: initial NRDA (natural resource damage assessment) studies based on 1970s paradigm report short-term injuries. Population-level collapses of pink salmon and herring stocks in 1992-1993 reveal gross inadequacies of old ecotoxicity models to explain reality of situation. Fishermen blockade Valdez Narrows, holding up oil tanker traffic for three days, and disband after promise from EVOS Trustee Council scientists to design comprehensive, ecosystem studies to determine full impacts of EVOS. Subsequent studies, conducted from 1994-2000, demonstrate new understanding of ecotoxicity of oil. First reviews of 400-peer reviewed papers completed in 2001. Overview of results from four main comprehensive studies including herring and pink salmon work by NOAA Auke Bay Lab in Juneau, Alaska, and studies funded through EVOS Trustee Council, the entity responsible for managing the $1 billion civil settlement for natural resource damages.
Brief review of attempts by Exxon to derail new science and stall unfavorable public policy changes.

3.) Conclusion of what is to be learned: Oil is much more toxic to natural resources than we thought. Levels of 1 to 10 ppb (parts per billion) PAHs causing functional sterility in a variety of fish, birds, and marine mammals. Federal water quality standards for PAHs, thought to be protective of marine life, are 300 ppb. Further, persistence of oil in some habitats, while not new, chronically exposes fauna to oil with population-level impacts. Implications of this new science for continued use of fossil fuels are discussed.

4.) References: Carls, M.G., Babcock, M.M. P.M. Harris, G.V. Irvine, J.A. Cusick, and S.D. Rice. 2001. Persistence of Oiling in Mussel Beds after the Exxon Valdez Oil Spill. Mar. Environ. Res. 51:167-190
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Rice, S. D., and R. Heintz. 2000. "A Shifting Paradigm for Impacts of Oil Pollution" with a bibliography of papers relevant to the paradigm shift. NMFS Auke Bay Lab, AK.
Rice, S. D., R. Heintz, M. G. Carls, J. W. Short, and A. Moles. In press. Long term Biological Damage...What Do We Know and How Should it Influence our Decisions on Response, Assessment and Restoration? International Oil Spill Conference (IOSC), March 2001.
Rice, S. D., J. W. Short, R. Heintz. In press. Oil and Gas Issues in Alaska: Lessons Learned about Long-term Toxicity Following the Exxon Valdez Oil Spill. Proceedings of the Oil and Gas Workshop. Continuing Studies in Science at Simon Fraser University
Rice, S. D., J. W. Short, R. A. Heintz, M. G. Carls, and A. Moles. 2000. Life history consequences of oil pollution in fish natal habitat. Pp. 1210-1215 In: Peter Catania (ed.), Energy 2000: The Beginning of a New Millennium. Technomic Publishing Co., Lancaster, England.
Rice, S. D., R. E. Thomas, R. A. Heintz, A. C. Wertheimer, M. L. Murphy, M. G. Carls, J. W. Short, and A. Moles. Submitted. Impacts to pink salmon following the Exxon Valdez oil spill: persistence, toxicity, sensitivity, and controversy. Reviews in Fishery Science.

Abstract Information & Notes

Kaye H. Kilburn, M. D. Date of talk: Thursday, June 6, 2002 11:00amUniversity 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
Major and date of Graduation: 1954
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, President & Director, Neuro-Test, Inc.
Disclosure Statement: None
SPEECH TITLE: **Arguments for One Brain Disorder from Chemicals**
The information below has been provided by the speaker.
1.) Goals and objectives: Analysis of the chemical syndromes to understand that the brain is the target and that manifestations can be measured.
2.) Outline of talk/abstract: An umbrella approach recommends neurobehavioral testing of people exposed to chemicals usually indoors to show that brain function measurements objectify the disorder.
3.) Conclusion of what is to be learned: Chemical syndromes MCS, CFS, FM, GWS, etc. are manifestations of brain damage.
4.) References: K.H. Kilburn: Chemical Brain Injury, New York: John Wiley and Sons, 1998.

ARGUMENTS FOR ONE BRAIN DISORDER FROM CHEMICALS

Kaye H. Kilburn, M.D.
Ralph Edgington Professor of Medicine
USC Keck School of Medicine

Confusion has reigned since the symptoms-based descriptions of chronic fatigue syndrome, myalgic encephalopathy and multiple chemical sensitivity. The absence of objective abnormalities from physical findings, imaging or other tests meant diagnosis depended on complaints/symptoms. The lack of specificity and quantification has impeded the search for causes and rational therapy. The shifting sands of beliefs substite for direct evidence. After 50 years a change in orientation seems overdue.
To take the next step regarding chemical sensitivity-chronic fatigue, perhaps we need better understanding of information at hand concerning the central role of the brain. I think that we have failed to ask the right questions. It is time to take stock. Fixed beliefs have polarized people into opposing camps. The plethora of clues suggests fresh perspectives on sensitivity and fatigue are needed to solve the problem (1). Common features, Table 1, and conditions of exposure, Table 2, help define the problem.
I will begin with three generalizations that seem obvious and then discuss five key concepts.

Generalizations:
Disorders due to chemicals are frequent.
Sensitivity to chemicals has 15 names and designations that are so similar that they must describe the same phenomena (2). Lumping them makes sense (Table 3).
Many people are affected by chemicals spread indoors (3,4,5) but some experienced massive releases into the atmosphere. At Bhopal and in the Gulf War hundreds of people were affected at once. I have studied less dramatic exposures that occurred at Lobelville TN, Tucson and Phoenix AZ and Bryan TX (6).

Perspectives-Overview:
Chemicals impair human brain function, which is the key to understanding and further research.
Limbic structures communicate with cortex and hypothalamus to direct diverse effects including to disorder moods and to raise the frequencies of symptoms (7).
Often patients identify a specific chemical or a group that first bothered them but subsequently find many chemicals trigger ill effects (6).
Concentrations of chemicals in air, absorbed into blood, fat and the brain and incorporated need to be correlated with impaired central nervous system function. Certain function tests make comparisons easy. People's indoor environments suggest which chemicals should be analyzed in expired air, blood and other tissues (8,9).

Therapeutic interventions, even avoidance should have record keeping and research to measure any effects of reducing exposures and body burdens of chemicals and drugs. Giving antioxidants and oxygen at the same time is irrational.

To overcome zealous splitting that led to semantic confusion and conceptual paralysis the chemically associated syndromes or complaint aggregates described (after 1950) should be viewed as one, Table 3. Taken together they signal a growing problem. The 15 titles for this disorder remind one of the parable of 6 blind men of Indostan observing an elephant (2). (It's like a wall, a spear, a snake, a tree, a fan and a rope.) Careful consideration including abnormal brain function in most people after several community wide chemical exposure's points to the brain as being primarily affected by chemicals. It modifies moods, stimulates (up regulates) or depresses (down regulates) functions and disturbs perceptions with unpleasant feelings.

The human brain directs an orchestra of extremely diverse responses. The brain initiates rapid or deep breathing, fast or abnormal heart rhythms, dilates or contracts blood vessels, increases or decreases stomach and bowel activity (emptying versus stasis), and modifies vision, balance and the speed of response (10).

The brain is generally or focally over stimulated to cause seizures, epilepsy or under-stimulated, sedated producing sleep and coma. Drugs that over stimulate, the convulsants: strychnine, picrotoxin and phenylenetetrazol have generalized effects as do amphetamines, LSD, PCP and OXY (Oxycontin). In contrast, sedative and narcotic (pain killing) drugs induce coma. Usually the central nervous system responds as a whole. For chemicals to stimulate or depress brain functions they must be delivered to it. The human lung is the ultimate absorptive organ. To facilitate intake of oxygen it has a minimal membrane thickness, 1/10,000, that of skin and the area of a tennis court (80 square meters)(11). Oxygen absorption can reach 4 liters per minute during exercise. Hydrocarbon solvents, hydrogen sulfide, ammonia, and chlorine enter blood and brain like oxygen. Inhaled ether causes unconsciousness in seconds. The brain controls all bodily operations and functions.

Conscious, cortical impulses conducted to the limbic "system" and hypothalamus coordinate the autonomic nervous system and influence metabolism, growth and reproduction via autonomic adrenergic and cholinergic (vagus) nerves and the ductless endocrine glands, thyroid and adrenal. Papez proposed in 1937 (12) that the circuit for emotions consists of the amygdala, hippocampus and fornix and mammillary body, the limbic structures. Through the cingulate gyrus, the frontal association cortex, parietal and temporal association cortices access limbic structures. Thus the cerebral cortex stimulates the limbic "system" to influence breathing, circulation, digestion, muscle tone and movements and endocrine functions (11).

Eons of evolution produced our brain's complex and varied functional capacities. These carryover systems have differing susceptibilities to man made (environmental) chemicals.

Measurements of brain functions identify what processes such as balance, reaction time, blink reflex or color recognition and mental ability starting with verbal recall have been impaired, Table 4. Detailed accounts of these tests and the results of testing individuals and groups of chemical exposed people have been published (6,13-15). There is evidence for actuation of autoimmunity by chemical's toxic to the human brain (16,17). Only measurable functions, 8 physiological tests and 11 psychological ones yield reliable numbers and in groups of subjects distribute symmetrically around the mean (18). Three examples selected from 25 studies illustrate these connections, Table 5. Chlorpyrifos, polychlorinated biphenyls and diesel exhaust impair the human brain.

The high brain toxicity of chlorpyrifos (sold as Dursban) and sprayed indoors to kill insects has been realized. It persists for many months on carpets, drapes, wall coverings and surfaces (19) and occupants develop insidious neurobehavioral impairment and symptoms (20). It has been banned for indoor uses in the United Stated by Environmental Protection Agency (EPA)(21). It is well known that single high doses cause cholinergic crises but less appreciated that insidious poisonings frequently present as chemical sensitivity and brain impairment. Pyrethroids, synthetic chrysanthemum insecticides that have been sprayed in airliner cabins to persist have poisoned flight attendants and people indoors at home with permanent effects.

Similarly PCBs persist for years and heating transforms them into dibenzofurans increasing their neurotoxic potency 1,000 to 10,000 fold (13). Diesel exhaust is often pulled indoors and into cars and concentrated after absorption to cause insidious poisoning (14). People's impairment is similar to that from being aboard locomotives or in shops repairing diesel engine. Suspicion plus thorough detective work makes causal links.

The therapeutic lesson is to minimize exposures found to be noxious from informal challenges or deliberate tests. Avoidance of the chemical or chemicals is key. Most are inhaled indoors in tight buildings. But sometimes foods that are suspected from history taking must be removed and reintroduced one at a time. A time saver when patients are distressed is to isolate them in a ceramic-glass-stainless steel environment with air cleaned by charcoal and hepa filters for the ultimate avoidance.

Therapeutic trials should measure patient's brain functions before and after intervention. Metabolic and blood flow imaging during burden reduction and challenge testing may be useful. Therapeutic protocols should be modeled on the trials for cancer treatment drugs.

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TABLE 1
Chemical Sensitivity-Chronic Fatigue: common features
Indoors
Irritability of nose, eyes and airways
Extreme Fatigue
Depression
Memory and concentration loss
Headache
Dizziness and balance
Loss of libido
Pain

TABLE 2
Mimicking asthma the symptoms are related to chemical exposure occur at:
Low doses below 5% or 2.5% or
even .001% of occupational tolerance
Resolve after removal
Recur on re-exposure
Threshold decreases
Multiple substances trigger
Multiple symptoms are common
Inappropriate laboratory tests are normal
Neurobehavioral functions are impaired

TABLE 3
CHEMICAL BRAIN INJURY
"A ROSE BY ANY OTHER NAME WOULD SMELL AS SWEET"
Multiple chemical sensitivity

Chronic fatigue syndrome
Indoor air illness, sick building syndrome
Fibromyalgia
Depression
Asthma, hyper reactive airways disorder
Gulf War syndrome
Organophosphates
Mold and mycotoxin disorders
Cacosmia: smell dysfunction,
Chronic Lyme disease,
Neuroendocrine hyperactivity
Degenerative brain diseases, PD-ALS,
Traumatic brain injury,
ADHD: Attention deficit hyperactivity disorder

TABLE 4
Test Part of Brain

Simple Reaction Time & Visual Two Choice Reaction Time	retina, optic nerve and cortex integrative radiation to motor cortex
Sway-Balance	inputs: ascending proprioceptive tracts, vestibular division 8th cranial nerve, cerebellum, vision, visual integrative and motor tracts
Blink Reflex Latency	sensory upper division trigeminal nerves (V), pons, facial nerves (VII)
Color Confusion Index	center macular area of retina, with optic cones, optic nerve, optic cortices
Visual Fields	retina-optic nerve-optic cortex
Hearing	auditory division of 8th cranial nerve
Verbal Recall Memory	limbic system of temporal lobe, smell brain
Problem Solving, Culture Fair & Digit Symbol	cerebral cortices: optic-occipital parietal cortex
Vocabulary	long-term memory, frontal lobes
Information, Picture Completion & Similarities	long-term memory, frontal lobes
Pegboard Performance	optic cortex to motor cortex
Trail Making A & B	(eye-hand coordination)
Fingertip Number Writing	parietal cortices, sensory area of pre Rolandic fissure
Profile of Mood States	limbic system for emotional memory

TABLE 5
Specific chemicals as possible cause in indoor air:
Toluene diisocyanate,
Trimellitic anhydride (TMA),
Organophosphate insecticide spray residuals
Organochlorine-chlordane residuals
Pyrethroids residuals
Molds and mycotoxins: trichothecenes
Organic solvents; chlorinated ones (cacosmia)
Formaldehyde, chlorine, ammonia
Typical outdoor chemicals:
Chlorine, hydrogen chloride, hydrogen sulfide, diesel exhaust, Gulf War syndrome

Abstract Information & Notes

William J. Rea, M.D. Date of talk: Thursday, June 6, 2002, 11:30am
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
Major and date of Graduation:
Residency: University of Texas Southwestern Medical School
Board Certifications: American Board of Surgery; American Board of Thoracic Surgery; American Board of Environmental Medicine
Current Faculty Appointments: Capital University of Integrative Medicine, Washington, D.C.
Current Job Description: President, Environmental Health Center - Dallas
Disclosure Statement: None

SPEECH TITLE: **Weather Pattern Monitoring**
The information below has been provided by the speaker.

1.) Goals and objectives: To understand weather patterns.

2.) Outline of talk/abstract: Weather patterns including pollen and mold counts, humidity, wind patterns, temperature and pollutant levels have been monitored at the Environmental Health Center - Dallas. In the last two years, new EPA monitoring stations around Dallas have allowed us to better access the total environmental load that our patients are exposed to. At certain times of the year, the load is extremely high, sometimes resulting in factors that were impossible to see in the past, i.e. particulate matter < 2.5 μ was found to be extremely elevated on certain days when other pollutants both manmade and natural were found.

3.) Conclusion of what is to be learned: It has now been shown these levels can precipitate heart attacks and are responsible for other poor bodily functions.

SPEECH TITLE: **Lipid-Soluble and Water-Soluble Antioxidant Profiles Measured in Chemically Sensitive Patients and Controls**
The information below has been provided by the speaker.

1.) Goals and objectives: To understand the ramifications of lipid soluble and water soluble antioxidant diagnosis and treatment in chemically sensitive patients.

2.) Outline of talk/abstract: The serum concentrations of antioxidants have been measured for the lipid-soluble antioxidants coenzyme Q10 (Ubiquinol), -Tocopherol (vitamin E), -Tocopherol, Lycopene, -Carotene, -Carotene, vitamin A (Retinol) and for the water-soluble antioxidant Ascorbate (vitamin C) by Pantox Laboratories, San Diego, California, in 139 chemically sensitive patients treated and 30 selected controls.

3.) Conclusion of what is to be learned: The statistical analysis of the data showed large variations of the individual values of the antioxidants in both the chemically sensitive and controls, but did not reveal any significant difference between the two groups. This may be the result of the relatively small numbers of controls and indicate the necessity of further studies with an increased control group.
Notes:

Abstract Information & Notes

Russel J. Reiter, Ph.D. Date of talk: Thursday, June 6, 2002, 1:30pm
Dept. Cellular & Structural Biology,
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San Antonio, TX 78229-3900 E-mail: reiter@uthscsa.edu
Medical School Attended: 3 honorary doctor of medicine degrees
Major and date of Graduation:
Residency:
Board Certifications:
Current Faculty Appointments: Professor of Neuroendocrinology
Current Job Description: Research Scientist
Other Information: Editor-in-Chief, Journal of Pineal Research; Editorial Board of 14 other journals; Numerous research awards including A. Ross McIntyre Gold Medal, U.S. Senior Scientist von Humboldt Award (Germany), Lisoni Lincee Award (Italy), Inaugural Distinguished Scholarship Award (Univ. Texas), Invited speaker at 300+ international symposia, etc.
Disclosure Statement: None

SPEECH TITLE: **Melatonin: Antioxidant Par Excellence**
The information below has been provided by the speaker.

1.) Goals and objectives: 1) To identify the mechanisms which make melatonin and especially effective antioxidant, 2) To explain the antioxidant cascade by which a single melatonin molecule scavenges several free radicals.

2.) Outline of talk/abstract: See abstract

3.) Conclusion of what is to be learned: Melatonin's efficacy and ubiquity as a direct free radical scavenger and indirect antioxidant.

4.) References:
R.J. Reiter, News Physiol. Sci. 15:246-250, 2000;
R.J. Reiter et al, Cell Biochem. Biophys. 34:237-256, 2001;
R.J. Reiter et al, Curr. Topics Biophys. 24:171-183, 2001

MELATONIN: ANTIOXIDANT PAR EXCELLENCE
Russel J., Ph.D.

Melatonin (*N*-acetyl-5-methoxytryptamine) is a superior and ubiquitously-acting free radical scavenger and antioxidant for several reasons including the following: 1) Melatonin directly detoxifies a variety of oxygen and nitrogen-based free radicals and non-radical reactants; 2) the metabolites that are formed when melatonin scavenges radicals are themselves radical scavengers (thus, there is an antioxidant cascade which greatly increases the efficacy of melatonin as a scavenger; 3) melatonin stimulates a number of antioxidative enzymes including superoxide dismutase, glutathione peroxidase and catalase which rid the cell of toxic agents; 4) melatonin inhibits one prooxidative enzyme, i.e., nitric oxide synthase; 5) melatonin stimulates the production of another important intracellular antioxidant, glutathione; 6) melatonin increases efficiency of mitochondrial electron transport thereby likely reducing the free radical generation; 7) melatonin stimulates ATP product which enhances the ability of cells to repair free radical damage; 8) melatonin is readily absorbed via any route and enters all cells and subcellular organelles; 9) melatonin has no known prooxidative activity unlike some other classical

antioxidants. Finally, melatonin is now known to be available in edible foods, e.g., tart cherries, and therefore can easily be included in a healthy diet.

Abstract Information & Notes

C. Chan Gunn, M.D. Date of talk: Thursday, June 6, 2002, 2:00pm
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Canada
Medical School Attended: Cambridge University, U.K.
Major and date of Graduation: Natural Sciences
Residency:
Board Certifications:
Current Faculty Appointments: Clinical Professor, University of Washington
Current Job Description: President ISTOP
Disclosure Statement: None

SPEECH TITLE: **The Importance of Good Connections**
The information below has been provided by the speaker.

1.) Goals and objectives: The role of the Peripheral Nervous System is critical. It is subjected to constant attrition. Resulting Peripheral Neuropathy causes widespread dysfunction.

2.) Outline of talk/abstract: Every enervated structure needs constant nerve stimulation for control and maintenance of cellular function

3.) Conclusion of what is to be learned: What happens when nerves go wrong? Dysfunctions in major organs and musculoskeletal system reviewed.

4.) References: "Prespondylosis" and Some Pain Syndromes following Denervation Super sensitivity - *Spine*, Volume 5, number 2. March/April 1980, p. 185-192, C Chan Gunn

Notes:

Abstract Information & Notes

Colin H. Little, M.D. Date of talk: Thursday, June 6, 2002, 2:30pm
324 Stephensons Road Phone: (61)3 9888 1345
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Australia E-mail: littlec@bluep.com.au
Medical School Attended: University of Melbourne
Major and date of Graduation:
Residency: Western General Hospital
Board Certifications: FRACP, MRCP (UK), FACA
Current Faculty Appointments:
Current Job Description: Physician & Allergist, Researcher into sensitivity disorders.
Disclosure Statement: None

SPEECH TITLE: **T Cell Antigen Binding Molecules TABM in Food and Chemical Sensitivity**
The information below has been provided by the speaker.

1.) Goals and objectives: Assessment of the value of measuring TABM (T cell antigen binding molecules) to specific foods and chemicals

2.) Outline of talk/abstract: Data from published studies (see references) presented. Recent work to measure TABM to other foods and chemicals also discussed

3.) Conclusion of what is to be learned: Measurement of TABM to specific foods and chemicals may be of diagnostic value in patients with sensitivity disorders and may help clarify the mechanisms

4.) References:
Infection & Immunity, July 200 p 3840-3847.
Clin Immunol & Immunopathol, Nov 1998, p 160-170.
Arch of Environmental Health, Jan 199, p 6/14 and Sept 2000, p 304-318.

T CELL ANTIGEN BINDING MOLECULES (TABM IN FOOD AND CHEMICAL SENSITIVITY)
Colin H. Little, M.D.

The mechanisms underlying adverse reactions to foods and chemicals are only partially understood. Many aspects of the subject remain controversial. Further problems have arisen as a result of the difficulty in documenting adverse reactions.

There are at least five processes which may contribute to adverse reactions to foods and chemicals, and also to yeast and molds:

a) Metabolic abnormalities - examples are lactose intolerance and Aspirin sensitivity. In many cases metabolic abnormalities are proposed in the absence of supportive data from research. Also, where they may be metabolic disturbances, it is unclear whether they are primary or secondary to reactions.

b) Conditioning - in some cases this is a potent factor, particularly with adverse reactions to chemicals, Where this process is an element in adverse reactions, it is often difficult to know whether it is a major factor. It should be remembered that animal studies have shown that conditioned responses can closely duplicate immune reactions.

c) Toxicity - with regard to adverse reactions to chemicals (and perhaps molds), there is some debate as to whether sensitive individuals are particularly susceptible to the toxic effects of chemicals. However adverse reactions to chemicals are reported at concentrations far below those reputed to produce toxic affects. It is important that a distinction is made between a susceptibility to the irritant effects of chemicals and a specific sensitivity to chemicals, although the two may quite possibly co-exist.

d) Psychiatric - this proposes that patients "imagine" adverse reactions to foods and chemicals. The process is equated with a form of hysteria where unresolved personal conflicts are converted to physical symptoms. Adverse reactions are believed by the patient to be the basis for these symptoms. In view of the close relationship between the effects of stress and immune reactions on the central nervous system, dissecting these processes clinically can be difficult.

e) Immune reactions - inappropriate immune responses to foods or chemicals could potentially account for the diverse range of incitants reported by patients and the acquisition of a sensitivity disorder. However the failure to develop diagnostic immune tests, particularly in the case of adverse reactions to chemicals, has contributed to skepticism regarding the role of immune processes. I will present data addressing to this issue.

Involvement of immune reactions to foods and chemicals implies a loss of immune tolerance. This process of intolerance is now the subject of intense research which suggests that it is a physiologically active process, i.e., there are mechanisms to safeguard immune tolerance which are constantly operative throughout life. Increasingly, studies are being directed towards events which may undermine such tolerance, particularly regarding inappropriate signaling by dendritic cells.

When confronted by non replicating antigens, there are four possible immune outcomes:
1. Inadequate stimulus - for transient/weakly antigenic immune stimuli, no immune response is elicited.

2. Suppression of the immune response - for persistent/strongly antigenic immune stimuli, the response may be switched off by regulatory T cells. There appear to be at least two types of such cells involved, Tr1 and CD4+ CD25+ T cells. Tr1 cells may act by the production of IL-10. CD4+CD 25+ appear to require cell-to-cell contact and seem as particularly important in the prevention of auto immune diseases. Tr1 cells are thought to protect against contact dermatitis and can be induced by desensitizing injections for inhalant allergies.

3. TH1 response - this involves the proliferation of IFN in response to specific antigens and may cause substantial tissue damage. TH1 cells are thought to be implicated in rheumatoid arthritis, coeliac disease and contact dermatitis.

4. TH2 / TH3 response- TH2 response refers to antibody production, particularly IgE and IgG. The presence of T cells producing the cytokines IL-4 and IL-5 is central to this response.

However, often in association with TH2 responses, a second population of T-cells (TH3) may be induced which suppress TH1 responses. I will present data suggesting that TH3 cells may be important in some adverse reactions to foods and particularly to chemicals. The effects of TH3 cells are mediated by the cytokine, TGF. A novel class of antigen specific T-cell proteins (TABM) are thought to be necessary to deliver TGF to where antigen is localized, as will be discussed below.

In clinical practice we see three types of disorder where we suspect immune processes may be involved:
1. Contact dermatitis - research has demonstrated that tissue damage is mediated by TH1 or TC1 cells and that IL-10 producing Tr cells have an important protective role.

2. Food intolerance- IgE, IgG and TABM specific for food antigens may be involved. TH1 responses to foods appear to occur only in the exceptional case, for example in coeliac disease. For "incomplete" antigens, such as food additives, TABM are possibly implicated.

3. Chemical sensitivity - for "complete" antigens, where there is a stable bond between chemicals and protein, IgE and IgG antibody can be important. We now have considerable data to suggest that TH3 responses could be pertinent where adverse reactions occur to simple chemicals that do not bind readily to proteins.

Before discussing particular studies, it is important to outline the nature of TABM. This class of molecules we believe to be produced by TH3 cells. The response of TH3 cells is directed to TH1-inducing epitopes on antigens. The action of TH3 cells is thought to be mediated primarily by the cytokine TGF. TH3 cells inhibit TH1 responses, primarily by the action of TGF. These cells may be elaborated in association with a TH2 response where specific antibody is produced. The dual action of both TH2 and TH3 cells is the basis for "immune deviation", where there is humoral immunity and suppression of cellular immunity. This outcome often occurs in chronic infections where a persistent TH1 response would result in substantial tissue damage.

We believe TH3 cells act via the production of TABM. These molecules are antigen specific but are associated with the cytokine TGF (and sometimes IL-10). The function of these molecules may be to deliver the cytokines to where antigen is localized.

TABM have been reported in literature, sometimes as "suppressive factors", over the past twenty years. The recent development of a monoclonal antibody specific to epitopes unique to TABM, has facilitated their study. TABM have the following properties:

1. High molecular weight - molecular sieving studies indicate a molecular weight ranging from $3x10^5$ Dalton - more than 10^6 Dalton.

2. Serum concentrations of 10-50mcg/ml. This is far lower than antibody concentrations, but TABM may be directed to different epitopes.

3. Association with cytokines, particularly TGF and IL-10.

4. Secretion by T cells. We have demonstrated that TABM are produced by T cell (but not B cell) lines in vitro

5. Association with the enzyme elastase.

6. The activation of TGF occurs when TABM bind antigen. This crucial process implies that TABM only "deliver" TGFb in an active state when antigen is present. The activation of TGF is thought to be due to the action of elastase.

We have measured IgG and TABM in several clinical contexts.

1. Milk intolerant adults - in this group there were high levels of IgG antibody to casein and betalactoglobulin in patients in comparison to controls. The main subclass of IgG antibody involved was IgG1. Also, there were elevated TABM levels to the cows milk proteins casein, betalactoglobulin and alpha lactalbamin in the patient group. Subsequent studies have demonstrated that TABM are mainly directed to alpha lactalbamin.

2. Solvent sensitive subjects - In patients sensitive to the solvent toluene, there were elevated levels of TABM specific to benzoic acid (a metabolite of toluene) conjugated to Albumin. Levels were higher in patients with a longer history of chemical exposure.

3. Subjects with recurrent vulvovaginal candidiasis - patients with this disorder showed elevated levels of both IgG antibody and TABM to Candida mannan in comparison with controls. TABM was purified from a subject with a high titre of TABM to Candida mannan using affinity columns. This TABM was associated with the cytokine TGF. It was shown to inhibit IFN production by peripheral blood white cells stimulated with Candida extract.

We have also demonstrated the presence of TABM specific to melanoma antigens in patients with melanoma, and to filiarial antigens in patients with chronic microfilaremia.
TABM were purified from the serum of a patient with high titre to Candida mannan, as mentioned above. This TABM was associated with TGF. Interestingly, when antigen was added to the purified TABM, the associated TGF was activated. However this process was dose dependent : activation occurred at intermediate concentrations of mannan but not at high or low concentrations.

In an extensive study, TABM specific to benzoic acid conjugated to Albumin (termed BA- TABM) was purified from the serum of a patient with a high titre of this TABM using affinity columns. This BA-TABM was associated with large amounts of TGF 1 and TGF 2, and also the enzyme elastase. The addition of benzoic acid conjugated to Albumin to this purified TABM was associated with the activation of the associated TGF. Once again this effect was dose dependent, occurring at intermediate but not high or low concentrations of the conjugated chemical. Remarkably, a similar effect on the activation of TGF was also observed when benzoic acid was added to the BA-TABM, i.e., unconjugated benzoic acid interacted with the BA-TABM, resulting in the activation of TGFB. This finding implies that TABM may bind to unconjugated chemicals with cytokine activation. As will be mentioned in a second talk, such a process may have neurological effects.

Abstract Information & Notes

Allan D. Lieberman, M.D. Date of talk: Thursday, June 6, 2002, 3:30pm
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Medical School Attended: Chicago Medical School
Major and date of Graduation:
Residency: Children's Memorial Hosp. - Chicago
Board Certifications: American Board of Environmental Medicine
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: **Beauticians Beware/Disease in the Beauty Salon**
The information below has been provided by the speaker.

1.) Goals and objectives: Present a case report as means of establishing causation of disease from exposures from chemicals used in the beauty salon.

2.) Outline of talk/abstract: Case presentation, review of current literature.

3.) Conclusion of what is to be learned: Cosmetologists must be aware of what hazardous materials they are using and their possible consequences.

4.) References:

BEAUTICIANS BEWARE/DISEASE IN THE BEAUTY SALON

Allan D. Lieberman, M.D.
The case report is an important contribution to the medical experience and literature as it brings awareness to the possible cause of both old and new diseases. For those of us practicing environmental medicine, recognizing the cause of our patients' disease or disorder and then eliminating it or avoiding it becomes the most important aspect for helping the patient.
Beauticians beware is a case report documenting the beauty salon as a potential hazard for cosmetologists and more importantly their clients.

20TH ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT

SCHEDULE

Friday, June 7, 2002

8:45 ANNOUNCEMENTS/MODERATOR: Stuart Lanson, M.D.

9:00 Mohamed B. Abou-Donia, Ph.D., "Disruption of the Blood Brain Barrier and Neuronal Death after Combined Exposure to Stress, Pyridostigmine Bromide (PB), DEET, and Permethrin"

9:20 Q & A

9:30 C. Chan Gunn, M.D., "How to Recognize Early Neuropathy"

9:50 Q & A

10:00 BREAK WITH EXHIBITORS

10:30 Allan Magaziner, D.O., "Role of Complementary Medicine in Cardiovascular Disease"

11:15 Q & A

11:30 Bertie B. Griffiths, Ph.D., "M.I.M. (Mycotic Immune Modulator) Its Role in Clinical Medicine"

11:50 Q & A

12:00n BUFFET LUNCH WITH THE EXHIBITORS

MODERATOR: Kaye H. Kilburn, M.D.

1:30 Dick van Steenis, MBBS, "Industrial Air Pollution and the Country Doctor"

1:50 Q & A

2:00 Ronald Finn, M.D., "Twenty Years as an Environmental Physician"

2:20 Q & A

2:30 Stuart Lanson, M.D., "Homeopathy: An Old Modality for the Potential Treatment of the New Bioterrorism"

2:50 Q & A

3:00 BREAK WITH EXHIBITORS

3:30 C. Malcolm Beck, "The Carbon Cycle and Health"

3:50 Q & A

4:00 Kou Sakabe, M.D., "The Present Status of Chemical Sensitivity in Japan"

4:20 Q & A

4:30 CASE STUDIES & PANEL/ MODERATOR: Allan D. Lieberman, M.D.

Adrienne Buffaloe, M.D., "Treatment of Hyperemesis Gravidarum by Provocation Neutralization

6:00 Adjourn

FRIDAY, JUNE 7, 2002
ABSTRACTS
AND
HANDOUTS

Abstract Information & Notes

Mohamed B. Abou-Donia, Ph.D. Date of talk: Friday, June 7, 2002, 9:00am
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Box 3813 E-mail: donia@acpub.duke.edu
Durham, NC 27710
Medical School Attended: University of California, Berkeley, CA
Major and date of Graduation: Agricultural Chemistry, 1967
Residency: North Carolina
Board Certifications: American Board of Toxicology: Academy of Toxicological Sciences
Current Faculty Appointments: Professor of Pharmacology and Cancer Biology
Current Job Description: Teaching Toxicology to medical and graduate students carrying out research
Disclosure Statement:

SPEECH TITLE: **Disruption of the Blood Brain Barrier and Neuronal Death after Combined Exposure to Stress, Pyridostigmine Bromide (PB), DEET, and Permethrin**
The information below has been provided by the speaker.

1.) Goals and objectives: To investigate the interaction between Pyridostigmine bromide, DEET, permethrin, and stress on the development of neurotoxicity.

2.) Outline of talk/abstract: 1) Body weight and clinical condition, 2) Neurobehavioral assessment, 3) Acetylcholinesterase and plasma butyrylcholinesterase, 4) Integrity of the blood brain barrier, 5) Neuropathological and Immunohistochemical studies.

3.) Conclusion of what is to be learned: Combined exposure to stress and chemicals, PB, DEET and permethrin at real-life doses, can significantly increase the BBB permeability, decrease AChE activity, and induce considerable neuronal cell death in several brain regions.

4.) References: Abou-Donia, et al, (2001). Locomotor and sensorimotor performance deficit in rats following exposure to Pyridostigmine bromide, DEET, and permethrin, alone and in conclusion. Toxicol Science 60: 305-314.
Abdel-Rahman, A., A.K. Shetty, and M.B.,Abou-Donia (2002). Disruption of BBB and Neuronal Cell Death in Cingulate Cortex, Dentate Gyrus, Thalamus, and Hypothalamus in a Rat Model for Gulf War Syndrome. Neurobiol Diseas. (In press).

DISRUPTION OF THE BLOOD BRAIN BARRIER AND NEURONAL DEATH AFTER COMBINED EXPOSURE TO STRESS, PYRIDOSTIGMINE BROMIDE, DEET, AND PERMETHRIN

Mohamed B. Abou-Donia

A combined exposure to high doses of pyridostigmine bromide (PB), N,N-diethyl m-toluamide (DEET), and Permethrin leads to a significant toxicity and neurological dysfunction (Abou-Donia et al., J. Toxicol Environ. Health, 48: 35-56, 1996). We investigated the effects following combined exposure to low doses of these chemicals with stress, simulating the daily exposure experienced by veterans to these chemicals during Persian Gulf War. Two groups of male Sprague-Dawley rats were administered PB (1.3mg/kg/d, oral), DEET (40mg/kg/d, dermal), and permethrin (0.13mg/kg/d, dermal) for 28 days.

Animals in one of the above two groups were subjected to stress every day for the duration of the experiment by placing them in a Plexiglas® restraint tube for 5 minutes. Two additional groups of animals (one subjected to stress and vehicle treatment, and another treated with vehicle alone) served as controls.

Three sets of five animals from each of the above four groups were processed for: 1) evaluation of the blood brain barrier (BBB) permeability using injections of [^{3}H]hexamethonium iodide and 10% type IV horseradish peroxidase (HRP); 2) acctylcholinesterase (AChE) activity and m2 muscarinic ACh receptor biochemical assays; and 3) Hematoxylin and Eosin (H&E) staining and micortubule associated protein-2 (MAP-2) immunostaining. Animals subjected to either chemical treatment or stress alone did not show changes in body weight, brain [^{3}H]hexamethonium iodide uptake, brain AChE, plasma ChE but exhibited a slight increase in BBB permeability by HRP and a decreased m2- muscarinic ACh receptor ligand binding, in comparison to control animals. In addition, these animals exhibited either no or minimal neuronal cell death.

In contrast, animals subjected to both chemical treatment and stress exhibited a dramatic increase in BBB permeability (with focal perivascular accumulation of HRP in both cerebrum and the brainstem), a significant decrease in brain AChE activity, a decrease in m2 muscarinic ACh receptor ligand binding density in midbrain and cerebellum, and a significant neuronal cell death associated with a reduced MAP-2 expression in the cerebral cortex and the hippocampus. These results underscore that, when combined with stress, exposure to even low doses of PB, DEET, and permethrin, that produce minimal effects by themselves, leads to a significant brain injury.

Abstract Information & Notes

C. Chan Gunn, M.D. Date of talk: Friday, June 7, 2002, 9:30am
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Canada
Medical School Attended: Cambridge University, U.K.
Major and date of Graduation: Natural Sciences
Residency:
Board Certifications:
Current Faculty Appointments: Clinical Professor, University of Washington
Current Job Description: President ISTOP
Disclosure Statement: None

SPEECH TITLE: **How to Recognize Early Neuropathy**
The information below has been provided by the speaker.

1.) Goals and objectives: Early neuropathy - which can also cause chronic pain - is important but generally missed

2.) Outline of talk/abstract: Early signs of Peripheral Neuropathy are clinically detectable, but not obvious to most laboratory or radiological tests.

3.) Conclusion of what is to be learned: Many illnesses are caused by a loss of nerve stimulation. Brief discussion on how physical stimulation, such as acupuncture, helps.

4.) References:

a. Early and subtle signs of Low Back Sprain - *Spine,* Volume 3 Number 3, September 1978 p. 267 - 281,
C Chan Gunn & William E. Milbrandt
b. Epiphenomena of Radiculopathy - *Outline of a talk given to the Royal Society UK* - February 1, 1996,
C Chan Gunn
c. Acupuncture in Peripheral Nervous System - *Theory and Basic Science*, Chapter 9, p 137 - 150,
C Chan Gunn
Notes:

Abstract Information & Notes

Allan Magaziner, D.O. Date of talk: Friday, June 7, 2002, 10:30am
Magaziner Center for Wellness Phone: 856/424-8222
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Cherry Hill, NJ 08003 E-mail: n/a
Medical School Attended: Chicago College of Osteopathic Medicine
Major and date of Graduation: 1983
Residency:
Board Certifications: American Osteopathic Board of Family Practice, American Board of Environmental Medicine
Current Faculty Appointments: Clinical Instructor, Department of Family Practice, University of Medicine & Dentistry of New Jersey - Robert Wood Johnson Medical School
Current Job Description: Founder & Medical Director, Magaziner Center for Wellness & Anti Aging Medicine; President-Elect & Program Chairman, American College for Advancement in Medicine
Other Information: Author of Total Health Handbook: Your Complete Wellness Resource and Complete Idiot's Guide to Living Longer and Healthier
Disclosure Statement: None

SPEECH TITLE: **Role of Complementary Medicine in Cardiovascular Disease**
The information below has been provided by the speaker.

1.) Goals and objectives: 1) To identify new emerging risk factors in atherosclerosis
2) To understand the role of inflammation in vascular disease

3) To identify foods and nutrients that are effective in reducing cardiovascular risk.

2.) Outline of talk/abstract: See abstract

3.) Conclusion of what is to be learned: Participants will gain a better understanding of the newly emerging risk factors of cardiovascular disease. Although the traditional thinking still places great emphasis on high cholesterol and the use of statin medications for treatment, this presentation will evaluate other factors that can no longer be overlooked. The fact remains that half of all patients with heart attacks do not have high cholesterol and, therefore, other causes and treatments will be explored. This presentation will provide scientific and clinical evidence supporting the use of natural agents in lowering cardiovascular risk factors in both prevention and treatment.

4.) References: 1) Heber et al., Am J Clin Nutr 1999; 69:231-236; 2) Rissanen et al., Br J Nutr 2001; 85(6): 749-754; 3) Rao, AV et al., Drug Metabolism and Drug Interactions 2000; 17(1-4): 211-218; 4) Fernandez, JC et al., Clinical Drug Investigations, 21(2): 103-113, 2001; 5) Castano et al., J Geront 2001; 56A: 186-192

ROLE OF COMPLEMENTARY MEDICINE IN CARDIOVASCULAR DISEASE
Allan Magaziner, D.O.
Participants will gain a better understanding of the newly emerging risk factors of cardiovascular disease. Although the traditional thinking still places great emphasis on high cholesterol and the use of statin medications for treatment, this presentation will evaluate other factors that can no longer be overlooked. The fact remains that half of all patients with heart attacks do not have high cholesterol and, therefore, other causes and treatments will be explored. This presentation will provide scientific and clinical evidence supporting the use of natural agents in lowering cardiovascular risk factors in both prevention and treatment..

Abstract Information & Notes
C. Malcolm Beck Date of talk: Friday, June 7, 2002, 11:30am
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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:

SPEECH TITLE: **The Carbon Cycle and Health**
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:

CARBON CYCLE AND HEALTH
C. Malcolm Beck
When first measured years ago, the atmospheric CO_2 (carbon dioxide) was at 280 ppm. (Parts per million). Now atmospheric CO_2 is over 380 ppm and expected to reach 600 ppm in a few years. The rise is blamed on burning fossil fuels; however, back in ancient history, all of the carbon in the fossil fuels was combined with oxygen in CO_2 before the plants collected it and Nature (probably the Biblical Flood) put it under ground.

Sixteen years ago a scientist speaking to an environmental group stated that "All we need to do to offset the carbon dioxide going into the air from burning fossil fuels is to increase the organic content of our farm lands 1 tenth of 1 percent each year. That would take the CO_2 out of the air and put it back in the soil where it belongs". However, he didn't tell us how to do this. This statement got me to thinking and studying. With the help of soil scientists, we concluded, if we weighed the carbon missing from our farm and ranch lands it would just about equal the excess carbon that is in the air.

When soil scientists first began recording in this country the soil organic content was between 3 and 8 percent. Now, the soil organic content is down to 20 percent or less than the original recorded amount. Some farms lands are down to .2 percent.

Bad farming practices, over tilling and over-use of high analysis, carbonless, chemical fertilizers have destroyed the organic content and health of our food producing soils. This is causing serious environmental problems of soil erosion, water shortages, air pollution and nitrate toxicity in much of

our drinking water. These environmental problems adversely affect the health of plant, animal and human life on earth.

Worldwide, humans churn out 8 billion tons of carbon dioxide every year. There are 455 million acres of cropland in the US. and 578 million acres of grassland pasture. If we increased the organic content of just our cropland in the US a puny one percent we would take out 4.55 billion tons (over half of what the world generates annually) of carbon dioxide out of the air and return it to the soil.

When moisture and temperature is best for plant growth, it is also the best environmental conditions for the soil macro and micro life to degrade expired life forms. The decaying activity on and in the soil releases an abundance of CO_2 , much greater than all the animal life above ground generates and releases. However, CO_2 is slightly heavier than air, it can remain near the ground allowing plants to recapture it. Then, using energy from the sun, the plant separates the carbon from the oxygen, and then uses the carbon to make carbohydrates, a food and energy for all life. The released oxygen is the catalyst that allows us to use the food and energy.

Research by Dr. Joe Bradford, a USDA/ARS scientist, discovered the farmers that practice conservation tillage and no till agriculture see their soil organic content go up one tenth of one percent each year. Holistic management ranchers and organic farmers also see the organic content of their soil go up each year.
If we would humble ourselves and study Nature's ways of soil improvement instead always trying to dominate her; our soil, plants and all higher life, us included, would be much healthier and happier.
Carbon dioxide facts:

The higher the CO_2 concentration the better the plant grows until the concentration gets over ten times the normal or about 2800-ppm.

In a closed greenhouse, on a bright sunny day, growing plants run short of CO_2 causing the growth to slow. Covering the floor and walkways with a decaying organic mulch can produce a lot of CO_2, this improves the growth, flowering, fruit production and health of plants.

Photosynthesis rates are greatest when sunlight intensity is 1/4 to 1/3 less than maximum. But, if sunlight gets too low the stomata close and restrict the entrance of CO_2. However, research has shown that CO_2 can also pass through the epidermis of some plants.

When light intensities are too high on a bright, hot afternoon photosynthesis will shut down in many plants. And in some plants Photo-oxidation occurs, the leaves consume oxygen, causing them to bleach out.

Even though a succulent plant is mostly water it still pulls a lot of CO_2 out of the air to create carbohydrates that it sends out through the roots to feed microbes in the rhizosphere (around the root), which help the plant in numerous ways. Up to 80 percent of the carbohydrates a plant manufactures is sent to and out the roots. This attracts billions of microbes-bacteria, fungi and other soil life that is not only beneficial, but also necessary for healthy plant growth.

Much of the CO_2 that is released in the soil form decay is dissolved in water to form carbonic acids (H2CO3) which combines with soil minerals, dissolving and making them available for plant uptake. Plants need to transpire to pull minerals dissolved in water toward their roots. Soil low in necessary mineral nutrients could cause a plant to transpire more causing moisture loss from the soil. 99 percent of the water the plant pulls from the soil is transpired to the air.

A high mineral nutrient level in the soil will allow a plant to grow better and faster with less transpiration.

A high concentration of carbon dioxide in the atmosphere also allows the stomata to quickly get enough carbon so it needs to stay open a shorter period and stay shut longer, which also saves moisture lost form transpiration, which conserves soil moisture.

Reference. Yearbooks of agriculture. Plant physiology books. College chemistry and physics books. 44 years of my own research on the farm and in greenhouses. Many science publications.
Notes:

Abstract Information & Notes
Dr. Dick van Steenis MBBS Date of talk: Friday, June 7, 2002, 1:30pm
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Pembrokeshire SA73 1DF E-mail: vsteenis@pgen.net
Wales, UK
Medical School Attended: Adelaide (Australia)
Major and date of Graduation: December 1958. M.B.B.S.
Residency: Queen Elizabeth Hospital, South Australia
Board Certifications: None
Current Faculty Appointments: None
Current Job Description: Retired General Medical Practitioner. Research in Toxicology and Epidemiology since January 1995.
Disclosure Statement: None

SPEECH TITLE: **Industrial Air Pollution and the Country Doctor**
The information below has been provided by the speaker.

1.) Goals and objectives: Explain how mishandling of hazardous waste and pollutants affect multiple body systems.
2.) Outline of talk/abstract: Cocktails of PM2.5 particles and other pollutants are causing respiratory and cardiovascular diseases, depression, ME, birth defects, premature deaths, etc.
3.) Conclusion of what is to be learned: Necessity of careful history of victims including pollutant exposures, plus necessity of better training in Toxicology and Epidemiology.
4.) References:
Clean Air Task Force. October 2000. Death, Disease & Dirty Power.
www.cleartheair.org
Belgian Platform environment & Health. July 2001 Report on the health impact of the MIWA waste incinerator in St. Niklaas. www.milieugezondheid.be
Notes:

Abstract Information & Notes
Professor Ronald Finn, M.D. Date of talk: Friday, June 7, 2002, 2:00pm
8 Prestwick Drive Phone: 0151-924-6657
Blundellsands Fax:
Liverpool L23 7XB E-mail: joronfinn@eggconnect.net
Medical School Attended:
Major and date of Graduation:
Residency:
Board Certifications:

Current Faculty Appointments:
Current Job Description:
Disclosure Statement: None

SPEECH TITLE: **Twenty Years as an Environmental Physician**
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:

TWENTY YEARS AS AN ENVIRONMENTAL PHYSICIAN
Professor Ronald Finn. M.D. FRCP

My twenty years as an Environmental Physician coincides almost exactly with the duration of the Dallas Conferences. This is the 20th Dallas Symposium and I have taken part in most of them.

When I look back over this period, I think of the lessons I have learned and the conclusions I have reached, and the purpose of this talk is to pass these thoughts on to you. In order to give an impression of the type of case that I have dealt with, I propose to describe some typical Environmental cases, and how they were managed; and finally to draw some general conclusions and lessons from these cases.

Environmental Cases.
The clinical cases to be described will include severe headache, migraine, irritable bowel syndrome, aphthous ulcers, emphysema and coronary artery disease.

Conclusions.
The work of most physicians is dominated by the control of symptoms in chronic disease by means of medication. Basically we don't cure but we manage disease. There is a quantum difference in treating Environmental cases. When the treatment is successful the patient is cured; he has no symptoms and no disease. This always gave me a high degree of personal satisfaction, which I did not experience to the same degree in other types of clinical work. I also have no doubt after 20 years that Environmental Medicine is a valuable therapeutic discipline.

Environmental Medicine is essentially chemical sensitivity in which an individual reacts to a chemical (simple or complex) to which most other subjects do not react.

There are two mechanisms. The first are true allergies mediated through the immune system, and the second, which is the largest group are chemical intolerance. We are all biochemically unique and react in different ways to environmental chemicals; thus a group of medical students will all react differently to the same quantity of alcohol. Some will show no effect but others will show various reactions including collapse, vomiting or aggressive behavior. The reason for the different reactions is probably inherited differences in the efficiency of various enzymes systems.

My main conclusion is that chemical sensitivity exists at two levels of clinical complexity. The cases described responded to the removal of one or at most two environmental chemicals. The more complex cases who react to many environmental chemicals require a much more sophisticated approach including desensitization, nutritional support, detoxication and the possible use of an Environmental Unit. Complicated cases should be referred to a physician specializing in Environmental Medicine, but it is important to appreciate that there are many simpler problems, similar to the cases described, which

can be successfully treated by any practicing physician with a minimum of environmental knowledge, and without the need for any special facilities.
How does one treat simple cases? We teach our medical students that diagnosis is a process of logical deduction, but in practice most doctors work by pattern recognition. They have seen a patient with appendicitis and recognize it when they see it again. Thus with experience you readily recognize the type of case that is likely to respond to an environmental approach, and the most likely environmental causes in each case. One then simply withdraws one or two of the likely agents for a trial period. One only diagnoses what one knows, and the important point is to be aware that chemical sensitivity exists. I personally always consider, however briefly, the possibility of an environmental approach in all cases.

What of the future? There are very large numbers of patients with simple environmental sensitivities who control their symptoms with long term medication. They often have restricted life styles due to their symptoms, and long term medication may occasionally cause serious side effects. It would therefore be helpful if all medical students could be taught the elements of simple environmental therapy. It would only require about two seminars, and the benefits to patients would be considerable.

Abstract Information & Notes
Stuart Lanson, M.D. Date of talk: Friday, June 7, 2002, 2:30pm
Scottsdale Allergy, Asthma, & Environmental
Health Center Phone: 480/994-9512
8416 E. Shea Blvd, Ste. 101 Fax: 480/994-3773
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Medical School Attended: USC Medical School
Major and date of Graduation: 1966
Residency: General Surgery 1969-70 Cedars of Lebanon, LA
Otolaryngology 1970-73 Univ. of Illinois Eye & Ear
Board Certifications: Diplomate in Board of Otolaryngology, Diplomate in Board of Environmental
Current Faculty Appointments: None
Current Job Description: Director of Scottsdale Allergy, Asthma, & Environmental Health Center
Other Information: FACS; FAAOO; FAAEM; FAAO; FAAFPRS Licensed in Homeopathy since 1995, Arizona.
Disclosure Statement: None

SPEECH TITLE: **Homeopathy: An Old Modality for the Potential Treatment of the New Bioterrorism**

The information below has been provided by the speaker.
1.) Goals and objectives: Historical review of homeopathic principles that can potentially be used to prevent and treat a variety of infectious diseases considered terrorist threats.

2.) Outline of talk/abstract: History and principles of homeopathy are reviewed. Advantages and disadvantages of homeopathy are reviewed as it is applied to the treatment of infectious diseases. Types of prescribing are reviewed. Homeoprophylaxis is discussed in regards to the advantages and disadvantages of using nosode remedies. Recommendations are made as to an approach to the treatment of specific infectious diseases such as small pox and anthrax using constitutional, genus epidemicus and nosode therapies

3.) Conclusion of what is to be learned: Homeopathic treatment for infectious disease has its limitations for public health. However, it has great usefulness in the prevention and/or treatment of certain infectious diseases in select individual patients when standard allopathic treatments are not

available or ineffective, or when the side effects of the vaccination or treatment would be too severe for a particular patient.

4.) References:

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Greene, Brian. The Elegant Universe. The Edge of Knowledge. 14. Vintage Books, 1999.

Homeopathy: An Old Modality for the Potential Treatment of the New Bioterrorism
Stuart Lanson, M.D.

Historical and theoretical review of homeopathy as a prevention and treatment of bioterrorism is discussed. Principles, advantages, disadvantages, schools of treatment, types of remedies, prescribing, constitutional therapy, genus epidemicus, and nosodes are discussed. Historical examples of a variety of infectious diseases treated with homeopathy with multiple references are used. Prophylactic homeopathic for over two dozen infectious diseases is reviewed. An approach to the treatment of bioterrorist infectious diseases with homeopathy is offered.
Notes:

Abstract Information & Notes
Bertie B. Griffiths, Ph.D. Date of talk: Friday, June 7, 2002, 3:30pm
Environmental Health Center-Dallas Phone: 214/368-4132
8345 Walnut Hill Ln., Suite 220 Fax: 214/691-8432
Dallas, TX 75231 E-mail: n/a
Medical School Attended: Graduate of the University of Wisconsin and University of the West Indies, Faculty of medicine. Recipient of Degrees in microbiology, virology, and postdoctoral training in Infectious Diseases and Immunology. Rockefeller fellowship to study Entomology and Virus epidemiology in Brazil and Trinidad. Appointments: Professor and Consultant in Microbiology and Infectious Diseases.

Major and date of Graduation:
Current Job Description: Director of EHC-D Laboratory.
Disclosure Statement:

SPEECH TITLE: **M.I.M. (Mycotic Immune Modulator) Its Role in Clinical Medicine**
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:

M.I.M.
(Mycotic Immune Modulator) Its Role in Clinical Medicine
Bertie B. Griffiths, Ph.D., William J. Rea,, M.D., Kevin Mascarenhas, M.D.

Introduction

•The objective of this presentation is to show that a metabolite isolated from the culturing of Aspergillus niger stimulates human lymphocytes. This element termed M.I.M (mycotic immune modulator) is being utilized, presently, therapeutically in our clinic on patients with a compromised T&B cell profile, and which have not responded to other treatments.

•In the presence of an antigen in cell culture systems, or in association with a major histocompatibility complex, the primary resting phase of the T lymphocytes can be interrupted. Various mitogens, or biological response modifiers may be utilized to activate lymphocytes. Some of these are concanavalin A (Con A), phytohaemagglutinin (PHA), pokeweed mitogen (PWM) (Greaves and Janossy,1972), purified protein derivative (Corrier,1991; Cram et al.,1976), bacterial species (Forsgren et al.,1980), transfer factor (Griffiths,1994).

•The effect(s) that certain mycological secondary metabolites have on lymphocytes, DNA and protein have been documented: Fusarium secondary metabolites inhibit proliferation of murine spleen lymphocytes (Bondy et al., 1991), and varied mycotoxins induce immunosuppression (Corrier, 1991). Gliotoxin induces DNA fragmentation in macrophages, inhibition of protein synthesis and causes irreversible inhibition of murine T cell proliferation (Waring, 1990). Sorenson et al. (1987) observed that Trichothecene mytoxins in aerosolized conidia of Stachybotrys atra significantly inhibited protein synthesis and thymocyte proliferation.

Materials and Methods

Molds

•Strains of Aspergillus niger were propagated on BBL sabouraud dextrose agar.
•Plates were exposed in the environment for 20-30 minutes, and subsequently incubated at 30 degrees Celsius.
•Colonies were subcultured in liquid media (BBL saboraud dextrose broth) and shake cultured at room temperature for 24-72 hours.
•Cultures were centrifuged, supernate concentrated, and chromatographed.

Column Chromatography

•DEAE- Celluse columns were developed following standard procedures.
•Elution was done by varying concentrations of potassium phosphate buffer (pH7.0)

•Eluants were examined spectrophotometrically, peaks combined, concentrated, and inoculated into cell cultures grown from peripheral lymphocytes.
•Fraction eluted with 0.1m buffer possessed the highest T lymphocytes stimulation ability; these fractions were combined, concentrated, dialyzed against 0.005 phos. buffer and used for clinical trials.

Cell Cultures
•Venous blood was collected and erythrocytes separated by use of heparinized CPT vacutainers (Becton Dickinson Co.)
•Lymphocytes were harvested, washed and inoculated into micro titer plates containing RPml 1640 medium, and incubated at 37 degrees Celsius for 48 hours.

Mitogenic assay of lymphocytes
•Micro titer cell cultures were inoculated with varying concentrations of the mold eluant as experimental mitogen, Con A and pHA at 100/ug as positive control, and with saline as negative control.
•All cultures were incubated at 37 degrees Celsius for 48 hours.
•Cells were harvested, re-suspended in DNA buffer and stained with propidium iodide and examined flow cytometrically.

Flow Micro Fluorometric Analysis
•Cells tagged fluorometrically for DNA were allowed to pass through a laser beam tuned to 488 nm. Florescence was measured electronically on the Coulter Epics Elite Spectrophotometer and recorded as histograms.
•DNA distributed in the cell cycle was calculated on accumulated data by parametric analysis.

Clinical Application
•The dialyzed concentrates were sterilized by seitz filtration, diluted and 0.01 ml. inoculated intradermally into 35 non-patient volunteers, and 20 patients.
•Patients were selected on the basis of having significantly compromised T&B cell profiles following appropriate treatments.

Results
•Differences in blastogenic response were observed when human T lymphocytes were inoculated with varied concentrations of the mold extract, Con A and PHA mitogens, and normal saline.
•Figures 1a, b and c show the typical flow cytometric DNA histograms of DNA distribution of unchallenged T lymphocytes, or when challenged with normal saline (negative control), and when challenged with Con A and PHA (positive controls), respectively. Figures 2a, b and c show the results of challenge with M.I.M dilutions1: 5, 1:25, 1:125. Table shows comparative dose response in the growth cycle of T lymphocytes stimulated with the mold extract and PHA.
•None of the 35 volunteers that were inoculated with M.I.M showed any adverse manifestation. On the contrary, they were eager to pursue a regime of treatment.
•At present, of the 20 patients being treated, one patient reported hoarseness and coughing and was advised to discontinue the treatment.
•Another patient, who was previously ill with lymphatic leukemia, and was being treated by his private physician reported at the Environmental Health Center. Here, he was found to have low values of T lymphocytes. After he was treated with 10 doses of M.I.M, the values of the T lymphocytes increased above normal values. This patient is now off M.I.M and is doing well.
•All other patients reported excellent manifestations. Progressive reports on their T&B cells profiles are not yet scheduled to date of this abstract.

Conclusion

•Our studies show that M.I.M, A product of fungal metabolism can significantly stimulate human T lymphocytes, invitro and invivo.

•Mitogenic response assay indicates a blast response of 88% at a concentration of 1:125 dilution. No adverse reaction was observed also, in higher dilutions,1:5, and 1:25.

•In comparison to the mitogenic response of the standard mitogens, Con A and PHA, M.I.M elicited approximately 9-10 times greater than these mitogens; it takes 100 Ug. of Con A and PHA to induce a blast of 92% and M.I.M to induce 88% blast at dilution 1:125.

•The deleterious effects of mycotoxins have been extensively documented in published literature. Effects include: Depression of T&B lymphocytes activity, suppressed immunoglobulin production, reduced complement or interferon activity and inhibition of DNA, RNA and protein synthesis -- to this end, there is ongoing investigation at the Environmental Health Center.

•Apart from the 2 patients who discontinued treatment with M.I.M, the data obtained at present show that M.I.M caused an increase in DNA synthesis by mitogenic effect; and the magnitude of the effect is dose dependent.

•M.I.M offers promising expectations of being a biological response modifier through its immunopotetiating mechanism(s).

Summary

•Aspergillus niger was grown in saboraud dextrose broth. Cultural extracts were concentrated and chromatographed on DEAE cellulose column, dialyzed against 0.005 molar phosphate buffer, inoculated into cell cultures of human peripheral T lymphocytes, and incubated at 37 degrees Celsius for 48 hours.

•Parallel tests were done with mitogens, PHA and Con A as positive controls and with normal saline as negative control.

•Cells were harvested and stained with propidium iodide and fluorescent isothiocyanate (Fit C) and examined microflourometrically. This mycotic extract was found to be significantly mitogenic on the basis of dose response.

•Dilution of 1:125 of this extract, now called M.I.M produced no adverse effect upon intradermal inoculation into 35 volunteers. Patients were treated similarly.

Notes:

Abstract Information & Notes

Kou Sakabe, M.D. Date of talk: Friday, June 7, 2002, 4:00pm
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Japan E-mail: sakabe1@attglobal.net
Medical School Attended: Tokai University Hospital
Residency: Tokai University Hospital
Board Certifications: 1) Japanese Society of Industrial and Occupational Medicine
2) Japanese Association of Physical Medicine, Balneology and Climatology
Current Faculty Appointments: Division Head of Clinical Ecology
Current Job Description: Clinical Ecologist, Environmental Toxicologist
Disclosure Statement:

SPEECH TITLE: **The Present Status of Chemical Sensitivity in Japan**
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:

THE PRESENT STATUS OF CHEMICAL SENSITIVITY IN JAPAN

Kou Sakabe, M.D.

Chemical sensitivity (CS) with 221 cases (men; 57, women; 164) who visited Environmental Medical Center; the Kitasato Institute from Aug. 2000 to Aug. 2001 were retrospectively investigated. The patients were mainly referred from patients'union of CS and from the doctors with suspicion of CS. The main triggering cause of CS was air pollution in 192 out of 195 cases. CS due to poor indoor air quality was 114 of 195 patients. Detection of objective disturbances in the nervous system seemed to be the most important to establish a consistent diagnosis of CS. The results obtained are as follows: 1) Disturbance of the visual modulation transfer function was detected in 63 of 111 cases. 2) Abnormal findings in electro-pupillography (an objective estimation of light reaction of the pupil) were detected in 63 of 116 cases. 3) Abnormal smooth pursuit eye movement was detected in 107 of 109 cases. 4) Abnormalities in the accommodation function of eye focusing and in the blood flow of the brain by single photon emission CT (SPECT) and near-infrared oxygen monitoring (NIRO) were often detected. These findings suggested that central nervous system of CS patients was mostly deranged and that neuro-ophthalmological examination could show positive findings.

221 patients with a variety of conditions were placed on a treatment program comprising as follows: a) patient education, b) injection therapy, c) nutrition replacement, d) physical therapy, e) low dose desensitization and d) selective environmental control. The overall success rate will be shown in presentation slides.

Notes:

Abstract Information & Notes

Adrienne Buffaloe, M. D., M. Ed. Date of talk: Friday, June 7, 2002, 4:30pm
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New York, NY 10016 E-mail: abuffaloe@aol.com
Medical School Attended: Columbus College of Physicians & Surgeons
Major and date of Graduation: 1989
Residency: Bellevue Hospital
Board Certifications: n/a
Current Faculty Appointments: NY University Hospital, John Jay College of Criminal Justice
Current Job Description: President Townsend Foundation - nonprofit for environmental issues
Disclosure Statement:

SPEECH TITLE: **Treatment of Hyperemesis Gravidarum by Provocation Neutralization**
The information below has been provided by the speaker.

1.) Goals and objectives: To resolve hyperemesis gravidarum by provocation neutralization with early placental hormones.

Hyperemesis gravidarum is a serious illness potentially resulting in abortion of the fetus or death of the mother. The early swift management can result in an excellent outcome for both patients. In-office provocation neutralization of early placental hormones has proved effective in resolving the condition within one day, allowing the mother to have a normal oral intake of fluids and nutrition. This pregnancy resulted in a normal full term delivery without complications.

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned: Provocation neutralization with early placental hormones is an effective method to treat hyperemesis gravidarum

4.) References:

1) Emergency Medicine: A Study Guide 5th edition New York: McGraw Hill, 2000.
2) Cunningham FG et al Williams Obstetrics 20th edition Stamford, CT: Appleton & Lange 1997.

CASE ONE

Slide One

G4 P3013 27 year-old black female presents with two days of vomiting during first trimester of pregnancy

Slide Two

Hyperemesis Gravidarum
Adrienne Buffaloe, M. D.
Epidemiology~
Nausea and vomiting-most common symptom of first trimester pregnancy
Morning sickness------hyperemesis----------death

Slide Three

Etiology/ Pathophysiology~
Unknown but contributing factors thought to be decreased gastric emptying time, decreased stomach acidity, increased chorionic gonadotrophin levels, circulating placental microvilli, neurosis.

Slide Four

Ho: hormones expressed by placenta first trimester: Chorionic Somatotropin (Placental Growth Factor and Choriomammatropin); and Placental Growth Factor

Slide Five

Signs & Symptoms~
Vomiting, blood ketones, increased urine ammonia, decreased urine chlorides, polyneuritis, weight loss, dehydration
Coffee-ground emesis, late finding, precursor to death.
T under 101, P<130

Slide Six

Diagnosis~
Clinical

Slide Seven

Treatment~
Provocation/Neutralization with Chorionic Somatotropin (Placental Growth Factor and Choriomammatropin); and Placental Growth Factor
IV Fluids, Antiemetics, Small carbohydrate meals. Abortion if T> 101, P>130, jaundice, dilirium, retinal hemorrhages.

Slide Eight

Prognosis~
Early hormonal titration viable intervention. Need more patients to document efficacy.

20TH ANNUAL INTERNATIONAL SYMPOSIUM
ON
MAN & HIS ENVIRONMENT

SCHEDULE

Saturday, June 8, 2002

8:45 ANNOUNCEMENTS/MODERATOR: Theodore R. Simon, M.D.

9:00 Mohamed B. Abou-Donia, Ph.D., "Subchronic Dermal Application of DEET and Permethrin, Alone and in Combination to Rats Impede Sensorymotor Performance and Causes Brain Neuronal Cell Death"

9:20 Q & A

9:30 William J. Rea, M.D. "Molds & Mycotoxins"

9:50 Q & A

10:00 BREAK WITH EXHIBITORS

10:30 Stuart Lanson, M.D., "Successful Treatment of Knee Implant Syndrome"

10:50 Q & A

11:00 Wallace Rubin, M.D., "Diagnosis and Treatment of Chronic Inner Ear Disease"

11:20 Q & A

11:30 Richard Jaeckle, M.D., "Depression & Forgetfulness, A Case Presentation"

11:50 Q & A

12:00n OPEN LUNCH

MODERATOR: Richard Jaeckle, MD

1:30 Rima E. Laibow, M.D. "Anticipatory States in Detoxification: Neuro Bio Feed Forward"

1:50 Q & A

2:00 Nancy A. Didriksen, Ph.D., "Interpreting Halstead-Reitan Neuropsychological Test Results in Chemically-Exposed Individuals Using Comprehensive Norms"

2:20 Q & A

2:30 Theodore R. Simon, M.D., "Environmental Sensitivity versus Cardiac Disease"

2:50 Q & A

3:00 BREAK WITH EXHIBITORS

3:30 Riki Ott, Ph.D., "Exxon Valdez Oil Spill Legacy: Cleanup Workers"

3:50 Q & A

4:00 Colin H. Little, M.D., "Immune - CNS Connections in Food/Chemical Sensitivity"

4:20 Q & A

4:30 CASE STUDIES & PANEL/ MODERATOR: Nancy A. Didriksen, Ph.D.

6:00 RECEPTION WITH THE EXHIBITORS

SATURDAY, JUNE 8, 2002
ABSTRACTS
AND
HANDOUTS

Abstract Information & Notes

Mohamed B. Abou-Donia, Ph.D. Date of talk: Saturday, June 8, 2002, 9:00am
Duke University Medical Center Phone: 919/684-2221
Laboratory of Neurotoxicology Fax: 919/681-8224
Dept. of Pharmacology and Cancer Biology
Box 3813 E-mail: donia@acpub.duke.edu
Durham, NC 27710
Medical School Attended: University of California, Berkeley, CA
Major and date of Graduation: Agricultural Chemistry, 1967
Residency: North Carolina
Board Certifications: American Board of Toxicology: Academy of Toxicological Sciences
Current Faculty Appointments: Professor of Pharmacology and Cancer Biology
Current Job Description: Teaching Toxicology to medical and graduate students carrying out research
Disclosure Statement:

SPEECH TITLE: **Subchronic Dermal Application of DEET and Permethrin, Alone and in Combination to Rats Impede Sensorymotor Performance and Causes Brain Neuronal Cell Death**
The information below has been provided by the speaker.

1.) Goals and objectives: To study the interaction between DEET and permethrin on neurobehavioral performance and brain tissue integrity

2.) Outline of talk/abstract: 1) Body weight and clinical condition, 2) Neurobehavioral assessment, 3) Morphological and histochemical analysis of brain tissues, 4) Morphometric analysis in different brain regions

3.) Conclusion of what is to be learned: Subchronic (60 days) dermal application of DEET and permethrin, alone or in combination to adult rats, leads to impairment of sensorimotor performance and a diffuse neuronal cell death in the cerebral cortex, hippocampal formation and the cerebellum.

4.) References:

Abou-Donia, et al, (2001). Effects of Daily Dermal Application of DEET and Permethrin, Alone and in Combination, on Sensorimotor Performance, Blood-Brain Barrier, and Blood-Testis Barrier in Rats. J. Toxicol Environ Health 62:523-541

Abdel-Rahman, A.A., A.K. Shetty, and M.B.,Abou-Donia (2001). Subchronic Dermal Application of N, N-Diethyl m-Toluamide (DEET) and Permethrin to Adult Rats, Alone or in Combination, Causes Diffuse Neuronal Cell Death and Cytoskeletal Abnormalities in the Cerebral Cortex and the Hippocampus, and Purkinje Neuron Loss in the Cerebellum. Experi. Neurol. 172:153-171.

SUBCHRONIC DERMAL APPLICATION OF DEET AND PERMETHRIN, ALONE AND IN COMBINATION TO RATS IMPEDE SENSORYMOTOR PERFORMANCE AND CAUSES BRAIN NEURONAL CELL DEATH

Mohamed B. Abou-Donia

To determine the effect of subchronic dermal application of these chemicals on brain, we evaluated histopathological alterations in the brain of adult male rats following a daily dermal dose of DEET (40 mg/kg in 70% ethanol) or permethrin (0.13 mg/kg in 70% ethanol) or a combination of the two for 60 days. Control rats received a daily dermal dose of 70% ethanol for 60 days. Animals were perfused and brains were processed for morphological and histopathological analyses following the above regimen.

In animals receiving either DEET or Permethrin, degenerating (eosinophilic) neurons were diffusely observed in distinct regions of the motor and somatosensory cortex, the hippocampus, and the cerebellum. In contrast, dying neurons were infrequent in animals receiving both DEET and Permethrin. However, the density of surviving neurons in cerebral cortex, hippocampus and cerebellar Purkinje cell layer of these animals were dramatically less than control animals, suggesting that in animals receiving both DEET and permethrin neuronal cell death occurs earlier than animals receiving either DEET or permethrin alone. Analysis of glial fibrillary acidic protein immunoreactivity revealed significant hypertrophy of astrocytes in all three treated groups with maximal changes in rats receiving both DEET and permethrin. Further, surviving neurons in the latter group exhibited abnormal dendrites, characterized by wavy and beaded appearance with microtubule associated protein-2 immunostaining.

Thus, subchronic dermal application of DEET and permethrin either alone or in combination leads to diffuse neuronal cell death in cerebral cortex, hippocampus and cerebellum of the adult brain; the neurotoxic effect is more pronounced when DEET and permethrin are applied together. Collectively, the above alterations can lead to many physiological, pharmacological and behavioral abnormalities, particularly motor and sensory deficits and learning and memory dysfunction.

Abstract Information & Notes

Wallace Rubin, M.D. Date of talk: Saturday, June 8, 2002, 9:30am
3434 Houma Blvd., Suite 201 Phone: 504/888-8800
Metairie, LA 70006 Fax: 504/455-6796
E-mail: wrubinmd@bellsouth.net
Medical School Attended: University School of Medicine
Major and date of Graduation: 1946
Residency: Tulane University School of Medicine
Board Certifications: American Academy of Otolaryngology, Head & Neck Surgery, American Board of Otolarnygology; AAOA
Current Faculty Appointments: Clinical Professor, Louisiana State University School of Medicine
Current Job Description: Solo Practice
Disclosure Statement: None

SPEECH TITLE: **Diagnosis and Treatment of Chronic Inner Ear Disease**
The information below has been provided by the speaker.

1.) Goals and objectives: To delineate diagnostic and treatment techniques

2.) Outline of talk/abstract: The diagnostic and therapeutic direction for the evaluation of the neurotological patient should be oriented to confirm an etiological mechanism.

3.) Conclusion of what is to be learned: This approach would then logically culminate in a systematic etiological investigation and treatment

4.) References:

1. Rubin, W.: "How Do We Use State of the Art Vestibular Testing to Diagnose and Treat the Dizzy Patient? An Overview of Vestibular Testing and Balance System Integration" Neurologic Clinics, Diagnostic Neurotology, Vol.8, No. 2:225-234, May 1990.

2. Rubin, W., Brookler, K.H.: "Dizziness: Etiologic Approach to Management" Thieme Medical Publishers, September, 1991.

3. Rubin, W. " Differential Diagnosis of Disorders Causing Dizziness: American Journal of Otology Vol. 14; No. 3:309-312, 1993.

DIAGNOSIS AND TREATMENT OF CHRONIC INNER EAR DISEASE

Wallace Rubin, M.D.

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

The questions to be answered by the neurotological evaluation are:

- What neurotological tests can be used for site of lesion confirmation?
- Which biochemical, metabolic, and hormonal tests are indicated?
- What modalities of therapy can then be efficacious?
- Do you perform a biochemical, metabolic, and hormonal screen? Which tests do you use? How do you make these decisions?

This is the challenge.

VESTIBULAR
DYSEQUILIBRIUM
#######
EQUILIBRIUM
MULTIMODAL
VESTIBULAR
VISUAL
PROPRIOCEPTIVE
#######
EQUILIBRIUM
DYNAMIC
ADAPTATION
COMPENSATION
#######
SENSORY DETECTION OF BODY POSITION
#######
EXECUTION OF APPROPRIATE MUSCULOSKELETAL RESPONSES
#######
INTEGRATION OF SENSORIMOTOR INFORMATION

#######
HEARING MECHANICS
COLLECTOR VIBRATOR CONCENTRATOR POP OFF VALVE
#######
INNER EAR
ENERGY CONVERTER MECHANICAL TO ELECTRICAL
#######
CHEMICAL OBSTACLE COURSE
INTESTINAL WALL
BLOOD BRAIN BARRIER
ENDOLYMPHATIC DUCT
#######
CHEMICAL MESSENGER DAISY CHAIN
ADRENAL GLAND PITUITARY GLAND HORMONAL SYSTEM
IMMUNE SYSTEM HYPOTHALAMUS
#######
NEW CONCEPTS
BIOCHEMICAL
IMMUNOLOGIC
NUTRITIONAL
NEUROTRANSMITTER
#######
BIOCHEMICAL
BLOOD DYSCRASIAS THYROID SUGAR FAT KIDNEY LIVER
#######
IMMUNOLOGIC
BACTERIAL VIRAL ALLERGY AUTOIMMUNE
#######
IMMUNOLOGIC
ALLERGIC SKIN TESTING - I.S.T. - INHALANTS RAST STUDIES - INHALANTS FOOD AND CHEMICAL AUTOIMMUNE - NO GOLD STANDARD TEST
#######
HORMONAL
ADRENAL PITUITARY THYROID PANCREAS SEX HORMONE HYPOTHALAMIC
#######
EVALUATION OF PATIENT WITH VERTIGO
A. HISTORY
B. OBJECTIVE EVALUATION
C. ETIOLOGIC INVESTIGATION
D. DIAGNOSIS
E. TREATMENT
####### #######
A. HISTORY B. OBJECTIVE EVALUATION
1. PERSONAL 1. PHYSICAL EXAMINATION
2. GENERAL MEDICAL 2. TESTING
3. TRAUMA A) AUDIO
4. OTHER EXPOSURES B) VESTIBULAR
3. IMAGING

4. MISCELLANEOUS-SUCH AS B.E.A.M.
####### #######
C. ETIOLOGIC INVESTIGATION D. DIAGNOSIS - BASED UPON:
1. BIOCHEMICAL A. HISTORY
2. IMMUNOLOGIC B. OBJECTIVE EVALUATION
3. IMAGING C. ETIOLOGIC INVESTIGATION
#######
E. TREATMENT
1. MEDICAL
2. SURGICAL
3. REHABILITATION
#######
WJ M 51 EXPOSURE 6-14-95
BENZENE VALVE DIZZINESS NAUSEA HEADACHE BUZZING RT EAR
#######
WJ M 51 5-6-96
NO PRIOR HEALTH PROBLEMS
SEEN BY ELEVEN DOCTORS USING CANE HEARING LOSS RT EAR
ABNORMAL ENG RT EAR ABNORMAL ROT POST
#######
BIOCHEMICAL IMMUNOLOGIC
CHOL 239 TRIG 476
ELEVATED
- TOLUENE XYLENE
- TRIMETHYLBENZENE
- DICHLORBENZENE
#######
ALLERGENS
ALTERNARIA CANDIDA
RHIZOPUS MITES
ASPERGILLUS
CORN SOY WHEAT
POTATO
BREWERS YEAST
TREATMENT
AVOIDANCE DIETARY INJECTION IMMUNIZATION

Abstract Information & Notes
Stuart Lanson, M.D. Date of talk: Saturday, June 8, 2002, 10:30am
Scottsdale Allergy, Asthma, & Environmental
Health Center Phone: 480/994-9512
8416 E. Shea Blvd, Ste. 101 Fax: 480/994-3773
Scottsdale, AZ 85258 E-mail: N/A
Medical School Attended: USC Medical School
Major and date of Graduation: 1966
Residency: General Surgery 1969-70 Cedars of Lebanon, LA
Otolaryngology 1970-73 Univ. of Illinois Eye & Ear
Board Certifications: Diplomate in Board of Otolaryngology, Diplomate in Board of Environmental
Current Faculty Appointments: None
Current Job Description: Director of Scottsdale Allergy, Asthma, & Environmental Health Center

Other Information: FACS; FAAOO; FAAEM; FAAO; FAAFPRS Licensed in Homeopathy since 1995, Arizona.
Disclosure Statement: None

SPEECH TITLE: **Successful Treatment of Knee Implant Syndrome**
The information below has been provided by the speaker.

1.) Goals and objectives: To demonstrate evaluation and treatment of a patient with implant syndrome.

2.) Outline of talk/abstract: A case history of a patient with knee implant syndrome is reviewed. Treatment strategies are discussed.

3.) Conclusion of what is to be learned: Successful treatment of a knee implant syndrome is possible. Various strategies for appropriate treatment are reviewed.

4.) References:

SUCCESSFUL TREATMENT OF AN IMPLANT SYNDROME
Stuart Lanson, M.D.

- Treatment of a knee implant syndrome was successful because the principles of treatment of implant syndrome were followed including:
- Removal of Implant
- Neutralization of implant material
- Reduce immune activation with immunotherapy
- Detoxification support reduce chemical body burden
- Nutritional support to up regulate essential metabolic pathways
- Oxygen Multistep Therapy to treat underlying vasculitis related to implant reaction
- Determine what safe materials can be used as an implant
- What was learned included:
    - Oxygen therapy adds another dimension to healing in implant syndrome
    - Modified testing protocols for implants
    - Prior pollutant load was significant
    - Stainless steel implants can be specially fabricated to avoid reactive metals.

Notes:

Abstract Information & Notes
William J. Rea, M.D. Date of talk: Saturday, June 8, 2002, 11:00am
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 Southwestern Medical School
Board Certifications: American Board of Surgery; American Board of Thoracic Surgery; American Board of Environmental Medicine
Current Faculty Appointments: Capital University of Integrative Medicine, Washington, D.C.
Current Job Description: President, Environmental Health Center - Dallas
Disclosure Statement: None

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

1.) Goals and objectives: To understand the problems with massive mold exposure and what to do clinically.

2.) Outline of talk/abstract: 55 patients (33 F & 22m, 1-76 y.o.d., 365 mean age) who had a toxic exposure to molds either in the home or workplace were studied. Molds found in the contaminated area had a wide range of findings including Cladosporium, Alternaria, aspergillus, stachybotyrus, penicillium and many others. These predominant molds were found in colonies with high levels too numerous to count.

Cause was always some kind of water leak. Sources were from seepage through a sewer pipe to roof leaks, shower and bathtub plumbing leaks, building leaks to faulty construction. The range of time spent in the moldy buildings was from 3 months to over 2 years. The patient's main symptoms were rhinosinusitis, bronchitis, short-term memory loss, confusion, lack of concentration, chronic fatigue, fibromyalgia, chronic cough and swollen nasal passages. Clinically the patients had a positive stressed Romberg's and Tandem Romberg's.

Laboratory studies showed that all were sensitive to molds on intradermal skin testing. 75% showed alteration in T&B lymphocytes and cell-mediated immunity was 96%. Computerized balance tests were run and 2 out of 3 were abnormal correlating with the clinical imbalance.

Five (5) patients had a triple camera SPECT brain scan with 100% being positive for neurotoxicity. 26 patients had autonomic nervous system imbalance either measured by the Iriscorder and/or the heart rate variability machine. 26 had dysfunction. Multi area surface thermography was measured in 10 with 10 being abnormal. Mold and mycotoxin antibodies were performed in 13 with 9 being abnormal.

Treatment consisted of massive avoidance of molds and toxic chemicals, injection therapy for molds and secondary foods and chemicals, nutrient therapy both oral and intravenous, heat depuration, physical and massage therapy. In some cases, autogenous lymphocytic factor was given. The results of treatment were quite remarkable with 82% improving.

3.) Conclusion of what is to be learned: It is clear that individuals exposed to high levels of molds and mycotoxins develop the symptoms of rhinosinusitis, bronchitis, short-term memory loss, lack of concentration, fibromyalgia and chronic fatigue and who have a positive stressed and tandem Rhomberg's sign should be treated as a chemically sensitive patient.

Broadspread avoidance of toxics, food rotation, nutrient supplementation and sauna are necessary to clear their symptoms and keep them well. Avoidance of mold exposure alone will not allow them to revert back to normal.
Notes:

Abstract Information & Notes
Richard Jaeckle, M.D. Date of talk: Saturday, June 8, 2002, 11:30am
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:

SPEECH TITLE: **Depression & Forgetfulness, A Case Presentation**
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:

Depression & Forgetfulness
A Case Presentation

Richard G. Jaeckle, MD
A 65 y/o retired nurse had developed depression and forgetfulness slowly and insidiously over several years. Antidepressants were of limited value, but somewhat helpful. As her condition became more alarming, she sought neurological consultation. The neurologist found no diagnosable illness, and only age-related changes on the MRI. He was perplexed and incredulous that she could not repeat any parts of a simple story that was used as a memory test, despite being able to drive and function apparently normally otherwise. A trial of Aricept was recommended but was not pursued.

The nurse had worked for twenty years and then retired to raise a family. There was no family history of depression. She and her family had always prided themselves for their memory and recall, so her forgetfulness was especially troubling. She frequently misplaced items and could not remember turning off electrical appliances, so she would recheck herself. Her speech pattern was characterized by forgetfulness of words, and she initially substituted phrases. Not recalling the word "cashier", she would substitute "the one who collects the money". "You know" and "you know what I mean" became more common. Recently, she found it difficult to finish a thought.

She began to dread social contact, and found it difficult to speak on the phone. She was acutely aware of and pained by her illness. Her laboratory results revealed only borderline anemia and leukopoenia, but were otherwise unremarkable. She was well nourished, took vitamins regularly, and had taken Armour thyroid 120 mg for thirty years and HRT for twenty years.

COMMENT
Since forgetfulness and memory disturbances are considered part of the diagnostic criteria for depression, it is standard practice to view this illness as "functional" and to prescribe sysmptom-oriented mediation, i.e., antidepressants. Should one take the approach of a neurological illness, the whole format changes. Her forgetfulness becomes aphasia, more specifically a conduction aphasia, which specifically denotes a difficulty repeating what one has heard and impairments in handwriting and word finding. Indeed, her earliest symptom, dating the onset of the illness ten years, was the difficulty making change in her volunteer job at her daughter's school (acalculia). Her forgetfulness of words and speaking around the deficit is a type of paraphrasia. Her most recent difficulty was inability to write checks or even to sign her name, leading her to depend more on credit or debit cards.

The presentation will focus on the laboratory evaluation, diagnosis, and progress in treatment of this patient.

Abstract Information & Notes

Rima E. Laibow, M.D. Date of talk: Saturday, June 8, 2002 1:30 p.m.
Interactive Health Care Sciences Phone: 845-680-0700
348 Rt. 9W, P. O. Box 688 Fax: 845-680-0500
Palisades, NY 10964 E-mail: rlalex@earthlink.net
Medical School Attended: Albert Einstein College of Medicine
Major and date of Graduation: 1970
Residency: Psychiatry-Adult, Psychiatry-Child
Board Certifications: Diplomate: Forensic Examination, Neurotherapy
Current Faculty Appointments: N/A
Current Job Description: Senior Medical Director
Other Information: President, Founder: Neurotherapy Certification Board, Past President: Quantitative EE Technician Board.
Disclosure Statement: None

SPEECH TITLE: **Anticipatory States in Detoxification: Neuro Bio Feed Forward**
The information below has been provided by the speaker.

1.) Goals and objectives: To present a novel strategy for assisting and enhancing recovery from environmental and endogenous toxicity.

2.) Outline of talk/abstract: 1) Definition of Neuro Feed Forward, 2) theoretical implications and distinctions between feedback and feed forward; 3) clinical application 4) conclusions

3.) Conclusion of what is to be learned: The central nervous system and autonomic nervous system are important regulators and targets of toxicity and detoxification. Regulation of their function can assist in both neuro psychiatric and seratic detoxification.

4.) References:

Bovnias, M, Stubblebine, A, Laibow, R: "Anticipatory Mental Imaging and "Neuro Bio Feed Forward" in Neurotoxicology AEHF 19th Annual Symposium. 2001 Dallas

Laibow, R, Stubblebine, A, Bovnias, M, "Neuro-Psychotoxicology and Q-BB6: Neuro Bio Feedback Potential in Diagnosis and Rehabilitation"
_____ Technology and Occupational, Med. 1996 p. 325-338

ANTICIPATORY STATES IN DETOXIFICATION: NEURO BIO FEED FORWARD
Rima E. Laibow

Recent investigation into the PsychoNeuroImmune (PNI) Axis, Autonomic Nervous System (ANS) and the Hypothalamic Pituitary Adrenal (HPA) Axis has made it clear that the so called "Mind Body Connection" is a misnomer. The mind and body, rather than being discrete entities which are somehow connected are, rather, a single, unitary entity, the "Whole" of "(W)Holistic". From this point of view, the previously impossible-seeming system-wide effects of either somatic or NeuroBioFeedback are solidly grounded in science rather than the perplexing mystery which non-organ system based effects have seemed to be for most of Biofeedback's history.

In our clinic, NeuroBioFeedback (NBF) is coupled with visualization, goal setting, psychotherapy and respiratory strategies as well as detoxification, nutritional medicine, etc. We have found that without those internally focused, cognitive/perceptual and self-modulatory strategies, NBF treatment results

were neither profound nor durable while, with them, results were durable, robust and unexpectedly profound, often beyond all reasonable expectation. Tissue regenerated and function returned (e.g., a function pituitary gland regenerated following an automobile accident) or function returned in the absence of tissue regeneration (e.g., full neurological function in a surgically left hemispherectomized patient).

Responses were so startling and unexpected, we initiated a self-funded research program to explore and exploit these results. In the course of this research we learned that biological change is governed by anticipatory states which take place at all levels from the molecular to the interpersonal (and trans personal) and at all developmental states from the embryonic onward. The maintenance of homeostasis, for example, requires the ability to sense and anticipate alterations of, and then return to, the desired state of the organism or sub-organismic system. As molecular, macromolecular, tissue and system-wide information is monitored from inside the organism by the brain, mental/emotional images or percepts are formed from this internally generated information which represented by changes in field potential, molecular signaling, ground substance regulation signaling and brain wave frequency. The neuronal configuration thus produced is primarily unconscious. These physiological processes may also be associated with conscious mental/emotional images.

HPA and PNI modulation are associated with mental/emotional imaging. Regulation of these processes is associated with mixed unconscious and conscious phenomena as memory, myth, meaning, metaphor and other mental/emotional processes are mediated by, and reciprocally mediate, internal events throughout the system, near simultaneously.

These sub-cortical and cortical perceptions condition the construction of mental/emotional imaging and, therefore, neuronal connectivity, and thereby impact physiological functions at all levels throughout the organism. Because they impact neuronal connectivity, all toxicants are neurotoxicants to a greater or lesser degree. Either disease or toxification can lead to alterations in the neuronal connectivity which, in turn, deforms the information used as a template for homeostatic reformatting or correction. This deformation of neuronal connectivity can be considered either reversible or irreversible unless modulation of anticipatory states is employed. Externally mediated healing events as well as internally mediated ones "reset" the anticipatory mental imaging taking place in the brain. "Miracles" stabilize the positive anticipatory mental images. NeuroBioFeedback is a technologically mediated means of stabilizing these anticipatatory states which is best called "NeuroBioFeedForward (NBFF)".

When neuronal connectivity is altered by toxicants and reference mental images or anticipatory images are distorted ("reversible damage") or destroyed ("irreversible damage") physiological functions are likewise distorted or destroyed. Either organic degradation/changes (e.g., receptor density, neuronal viability) or functional ones can create aberrant anticipatory images or make the achievement of normal ones difficult or impossible.

NBFF, coupled with reformatted conscious and unconscious anticipatory states, appears to have the capacity to elicit two types of change. By engaging the HPA, PNI and ANS in a process of operant conditioned EEG-focused learned behavior it is often possible to elicit neuronal and systemic repair and bring about a return to normal or near normal function even in the face of significant destruction of tissue or other organic pathology or toxicity. Significantly for our most damaged patients, when tissue repair is not possible, the holographic nature of the CNS can be tapped so that information present throughout the system can be used to create new anticipatory images or return to those held in the field of the organism before the injury which disregulated it. In addition to fostering a return to prior, higher levels of function (which predate toxification via this bio-anticipatory holgramatic repair) the joint interaction of the HPA, PNI and ANS have both the theoretical and clinical capacity to up-regulate systemic mechanisms of detoxification and tissue repair.

Thus, chemical sensitivity, environmental toxicity, emotional and organic disease all share a common mechanism of operation (distorted anticipatory imaging) and, hence, share the possibility of positive response to operant conditioned altered neuronal function through NBFF and similar techniques. This repair requires strong support from physical, environmental and nutritional medical approaches so that the micro- to macro-anticipatory images have a substrate field in which they can be fulfilled.
Notes:

Abstract Information & Notes
Nancy A. Didriksen, Ph.D. Date of talk: Saturday, June 8, 2002, 2:00pm
100 North Cottonwood Drive, Suite 106 Phone: 972/889-9933
Richardson, TX 75080 Fax: 972/889-9935
E-mail: n/a
Graduate School Attended: University of North Texas
Major and date of Graduation: Health Psychology / Behavioral Medicine, 1986
Internship: Environmental Control Unit - Northeast Community Hospital, Bedford, TX
Board Certifications: N/A
Current Faculty Appointments: Adjunct Professor of Psychology - University of North Texas
Current Job Description: Private practice - evaluating and treating chemically sensitive and other chronically-ill patient populations
Disclosure Statement: None

SPEECH TITLE: **Interpreting Halstead-Reitan Neuropsychological Test Results in Chemically-Exposed Individuals Using Comprehensive Norms**
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:
Notes:

Abstract Information & Notes
Theodore R. Simon, M.D. Date of talk: Saturday, June 8, 2002, 2:30pm
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 University
Major and date of Graduation: Nuclear Medicine 1980
Residency: Nuclear Medicine
Board Certifications: Nuclear Medicine
Current Faculty Appointments: N/A
Current Job Description: Private Nuclear Medicine Practice
Disclosure Statement:

SPEECH TITLE: **Environmental Sensitivity versus Cardiac Disease**
The information below has been provided by the speaker.

1.) Goals and Objectives: Frequently, environmental disease and cardiac disease have similar clinical presentations. The treating physician must distinguish between these processes. This talk provides objective, safe, rapid, and generally accepted tools for triaging patients who represent diagnostic

challenges. If cardiac disease is found, these tools will provide differential diagnostic, therapeutic, and prognostic guidance

2.) Outline: Diagnostic algorithms will be presented. Testing procedures, results, and implications will be discussed with case material and both scientific and practical underpinnings. Emphasis on choosing the proper test and understanding potential findings will provide efficient and effective cardiac triage techniques.

3.) Conclusions: Nuclear Medicine can safely, rapidly, and reliably identify, characterize, and guide cardiac disease in a patient population where usual clinical rules are not readily applicable
4.) References:

Notes:

Abstract Information & Notes
Riki Ott, Ph..D. Date of talk: Saturday, June 8, 2002, 3:30pm
Alaska Forum for Environmental Responsibility Phone: 907/424-3915
P.O. Box 1430 Fax: 907/424-3926
Cordova, AK 99574 E-mail: otter2@ctcak.net
Medical School Attended: Graduated with a doctorate from School of Fisheries, University of Washington, Seattle, WA. Took classes at UW School of Public Health.
Major and date of Graduation: Marine Toxicology, 1986
Current Faculty Appointments: Faculty mentor with Columbia Pacific University, Novato, California
Current Job Description: Environmental consulting and writing. Currently writing a book about the *Exxon Valdez* Oil Spill legacy and its public policy ramifications.
Other Information: A commercial fisherm'am, from 1985-1994, in Area E, Prince William Sound and the Copper River Delta, Alaska. Co-founder of Oil Reform Alliance, the Alaska Forum for Environmental Responsibility in 1994, the Copper River Watershed Project, and the Oiled Regions of Alaska Foundation.
Disclosure Statement:

SPEECH TITLE: ***Exxon Valdez* Oil Spill Legacy: Cleanup Workers**
The information below has been provided by the speaker.

1.) Goals and objectives: Demonstrate that workplace exposure to oil and other chemicals during the *Exxon Valdez* Oil Spill (EVOS) cleanup resulted in short- and long-term health problems, including chemical sensitivity, among thousands of cleanup workers. Present overview of Exxon's worker safety program and program shortfalls; analysis of available monitoring, clinical, and injured worker data sets; and findings from relevant court records and interviews

2.) Outline of talk/abstract: Review worker safety program including collection and relevance of monitoring data (from Exxon's contractor Med-Tox) and clinical data (from Exxon's contractor Veco), and failings of program (from publications, court records, congressional records, and media articles). Overview and discussion of results of three data sets including Med-Tox monitoring data, showing average workers were exposed to 12-times the OSHA PEL for oil PAHs (polycyclic aromatic hydrocarbons); Veco clinical data, showing a total of 6,722 cases of URIs (upper respiratory infections) were reported during 1989 with 300-500 cases reported *every week*; and Veco's injured worker data set (N = 1,600), showing the largest category of injuries after "physical injuries" (breaks, strains, and sprains) was respiratory damage.

Brief review of reports by the Alaska Department of Labor and NIOSH on the spill cleanup and thwarted efforts by these entities to independently monitor the cleanup. Brief review of Exxon's analysis of and conclusions from cleanup worker injury records.

Discussion of symptoms described by cleanup workers in court records and during personal interviews. Comparison of symptoms with Gulf War vets and others with chemical sensitivity

3.) Conclusion of what is to be learned: Thirteen years after the *Exxon Valdez* Oil Spill, hundreds of former cleanup workers were found to be suffering from poor health. Many symptoms described by former workers were consistent with chemical sensitivity. At the time of this writing (12/13/01), it is too early to conclude that chemical sensitivity symptoms are definitively linked to the cleanup work, except for former workers who filed spill-related, toxic tort lawsuits in the early 1990s. However, law firms such as Masry and Vititoe (of the movie *Erin Brockovich* fame) are actively pursing personal injury, toxic tort lawsuits for spill-related long-term health damage, and more about definitive linkages may be known by May 2002.

Meanwhile, a team of epidemiologists and environmental doctors is needed to review the available data sets.

4.) References:

Alaska Department of Health and Social Services. 1989. Public Health Advisory on Crude Oil. 28 April. *EVOS Field Hearing,* 1063-65.

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Laborers' International Union of North America. 1989. Letter from Angelo Fosco, general president, to Chairman Miller, enclosing prepared statement of Mano W. Frey, executive president, AFL-CIO on Recommendations for the *EVOS* Cleanup and other attachments. 6 May. *EVOS Field Hearing*, 1029-1069.

Laborers' National Health and Safety Fund. 1989. Report of the Public Health Team Assessing the EVOS Cleanup. Washington, DC, 24 April. *EVOS Field Hearing*, 1036-1062.

Laborers' National Health and Safety Fund. 1989. Letter to Jim Sampson, Alaska Department of Labor, regarding Veco worker training program deficiencies. 28 April. *EVOS Field Hearing*, 1067-1068.

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Lobdell, H. 1989. Testimony of Laborers' International Union of North America. US House of Representatives Committee on Merchant Marine and Fisheries, Subcommittee on Coast Guard and Navigation, Seattle, WA, 16 June.

McDowell, T. 1989. Seldovia Town Meeting with Spill Agency Representatives. 24 August 1989.

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EXXON VALDEZ OIL SPILL LEGACY: CLEANUP WORKERS PRELIMINARY FINDINGS FROM THE 1989 CLEANUP

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Thirteen years after the 1989 *Exxon Valdez* Oil Spill (EVOS), we started an investigation to determine if this spill and subsequent 1989 cleanup had any acute health effects on 11,000 workers. A literature review was conducted for environmental pollutants present during the cleanup, including crude oil, crude oil mist, seawater mist, diesel fumes, the dispersants Inipol EAP22, Corexit 9527 and 9580M, and the cleaning solutions Simple Green, De-Solv-It, and Citriklean. These environmental pollutants were all found to have known acute and chronic health effects. A synthesis of available documents, including court records, congressional hearings, and federal reports, on Exxon's worker safety program revealed that this program was severely flawed for multiple reasons. Available health and medical records from the State of Alaska (1,771 claims filed), Exxon (6,722 reported cases of Upper Respiratory Infections), and toxic tort lawsuits show that workers reported acute health symptoms identical to the potential

health effects listed for the environmental pollutants present during the 1989 cleanup. Further, the symptoms were strikingly similar - respiratory problems (leading injury), headaches, nose and ear bleeds, increased susceptibility to sinus infections, increased chemical sensitivity, and central nervous system problems such as dizziness, nausea, brain fag, coordination loss, and tingling appendages. Significantly, Exxon failed to report the URIs in violation of federal and Alaska laws.

Our findings indicate that the chemicals present during the 1989 cleanup were hazardous to human health; that workers were exposed to dangerous levels of dangerous chemicals; and that workers developed acute health symptoms from exposure to these chemicals during the cleanup. Interviews with 62 former workers found strong evidence of chronic health effects potentially from 1989 EVOS cleanup. The possibility of conducting a longitudinal study of the exposed population of 1989 cleanup workers is discussed.

***Exxon Valdez* Oil Spill (EVOS)**
March 24, 1989 in Prince William Sound (PWS), Alaska
Exxon: 11 million gallons
State of Alaska: 35 million gallons ± 20%
Exxon: 1,300 miles (2,090 km) of beaches
NOAA: 3,240 miles (5,221 km) of beaches
(est. 40% of oil stranded on PWS beaches)
Sources: State of Alaska 1991 (unpublished); Mearns 1996; Spies et al. 1996

EVOS Hazardous Waste Cleanup: April - September 15, 1989
Operations in Brief Types of Jobs
11,000 people Oil Response Technicians
21 million hours Bioremediation Application Technicians
1,600 vessels Dispersant Crews
1,000s of aircraft sorties Decontamination (laundry) Crews
150 miles of beaches "treated" Support (supply, trash, wildlife, divers,
medical, monitors, supervisors, etc.)

Beach Segments & Treatment Types
Total Segments 1,149: 519 PWS, 426 Gulf of Alaska, 138 Kenai Peninsula
Treatment Type # Segments % Effort
Manual: Pickup, remove debris & tar mats, 425 34%
hand-wipe, tilling/raking, etc.
*Hydraulic: Steam clean, water deluge, flooding, 486 39%
moderate or high pressure and hot
water wash (discontinued after 1989), spot wash
Mechanical: Till, sediment removal, berm relocation 54 4%
Chemical: Dispersants tested, not approved for use in 1989-1991 3 --
Bioremediation (1989-1992)
Inipol EAP22 (395,500 liters, 104,510 gal) 290 23%
Customblen (59,000 kg) 2
No treatment
TOTAL number of segments treated = 1,260 100%
*/ Note: Specifics for hydraulic operations. Beach crews: 60-120ºC, 345-55 kPa, 946 L/m.
13 MAXI barges: 60ºC, 552-827 kPA, 511 L/m. OMNI barges: 60ºC, 552-827 kPA, 1,893 L/m.
Source: Mearns 1996

Purpose of This Study:

Examine Health Effects of the EVOS Cleanup on Workers to

Determine if the EVOS cleanup had acute health effects on workers;
Determine if there is a potential for chronic health effects; and
Involve health care professionals and/or academics in longitudinal study of EVOS cleanup workers.
¡ This is a Work in Progress !

Methods & Resources

1. Literature review of compounds of concern
2. Evaluation of Exxon's worker safety program
    - NIOSH Health Hazard Evaluation 1991
    - Teitelbaum expert witness deposition
    - Court records for toxic tort lawsuits (1990s)
    - Exxon 4-hour safety training video
    - Congressional oversight hearing 1989
    - Seldovia town meeting video 8/24/89
3. Preliminary analysis of exposure assessments
    - Exxon's air quality monitoring data and statistical summary
    - Reller (1993) analysis of Exxon's data
4. Analysis of injury and illness data
    - Exxon's clinical data summary of 6,722 URIs
    - Alaska Worker Compensation Board report of 1,771 claims
    - Interviews with 62 former cleanup workers

Environmental Pollutants Present during 1989 EVOS Cleanup

Weathered crude oil, oil mist (airborne PAHs)
Seawater mist
Diesel fumes & exhaust
Bioremediation products: Inipol EAP22, (liquid), Customblen (pellets)
Dispersants: Corexit 9580M, Corexit 9527
Cleaning solutions: Simple Green, De-Solv-It, Citriklean

Compounds of Concern in Products Used during 1989 EVOS Cleanup

Product	Compounds of Concern
Inipol EAP22	2-butoxyethanol, laurel phosphate
Customblen	
Corexit 9527	2-butoxyethanol
Corexit 9580M	
Simple Green	2-butoxyethanol
De-Solv-It	Limonene, volatile napthas
Citriklean	

Table 1. Acute (a), Chronic (c), or Both () Health Symptoms

Symptom	Oil/skin	Oil Mist	PAH/air	SW Mist	Diesel
Respiratory damage					
CNS disorders					
Liver disorders	c	c	c		
Blood disorders (leukemia)	c		c		
Blood disorders (anemia)	c	c	c		
Kidney disorders	c	c	c	c	

Skin cancer c
Endocrine disruption c c
Immune suppression

Table 2. Acute (a), Chronic (c), or Both () Health Symptoms
Symptom Inipol SimpGrn Corexit DeSolvIt
Respiratory damage
CNS disorders
Liver disorders c c c
Blood disorders c c c
Kidney disorders c c c

Hazardous Waste Worker Safety Program
Purpose: "Provide information that will enable workers to perform tasks free of injury or illness" (NIOSH 1991)
Elements:
Occupational medicine structure
Comprehensive industrial hygiene (IH) plan
Worker safety training using Hazard Communication Standard
Implementation and effectiveness monitoring
Proper medical attention before, during, and after job

Occupational Medicine Structure
Element Reviewers Exxon
Lead medical physician OP ERP
Lead medical team OPs, IHs IHs
in residence rotated
Field medical teams OP, IH, nurses IH, nurses
every shift/2,500 not enough
Problem: Essential triage function to prioritize projects and assign resources to minimize illness outbreaks was unaddressed.
Sources: NIOSH 1991, Teitelbaum 1994

Comprehensive Industrial Hygiene Plan
Element Reviewers Exxon
Faulty assumptions/design PAHs/air VOCs/air
Monitor compds of concern
- VOCs, oil mist 33 1,725
- PAHs/airborne 25 30
- Diesel fumes 11 0
- Seawater mist 0 0
- Dispersants 0 0
- Cleaning solvents 11 112

Inter-lab QA/QC program need none
Preventative care
- Train field team to recognize/ need none
- report problems
- Lead OP access to medical records need none
- Timely reporting of illness/injury 0 reported 6,722 URIs

Problem: IH plan not designed to be responsive to problems of workers and thousands of workers became sick

Sources: NIOSH 1991, Teitelbaum 1994

Hazardous Waste Worker Safety Training

Element Teitelbaum Exxon
24-40 hours not adequate 4 hours
Trainers apprised of site specific risk not adequate 0 hours
Hazard Communication Standard applied failed

- MSDS right-to-know none
- Recognize/report symptoms right-to-know "benign"
- Access to info right-to-know "read labels"
- PPE required optional

Problem: Sick workers did not recognize/report symptoms with some exceptions (e.g., Inipol crews)
Sources: NIOSH 1991, Teitelbaum 1994; US Congress 1989; McDowell 1989; LIUNA 1989

Implementation & Effectiveness Monitoring

Element Reviewers Exxon
Wearing of PPE not consistent not enforced
Supply of PPE not adequate out of stock
PPE for PAH/air, Inipol not adequate "right gear"
Decontamination of PPE not adequate out of stock
Federal/state oversight not adequate obstructed
Problem: Workers became sick and most did not link health effects with cleanup work.
Sources: NIOSH 1991, Teitelbaum 1994; US Congress 1989; McDowell 1989; court records

Proper Medical Attention Before, During & After Job

Element Reviewers Exxon
Prescreening physicals needed none

- Long shifts (dyssynchronicity) cause illness ignore
- Preexisting illness susceptible ignore
- Job descriptions, work assessment needed none

During work

- Occupational medical staff OP IH
- Hospitalization OP regular doctors

Follow up

- Hazardous waste cleanup recommended none

Problem: Workers did not link health effects with cleanup work, were not aware how to improve health, and became susceptible to chronic problems.
Source: Teitelbaum 1994; court records; Phillips 1999; Murphy 2001

Table 3. Total Hydrocarbon (HC), Oil Mist, & PAH/airborne Exposures

Oil Mist[1] PAH/airborne[2]
Exxon (1989, air quality data)
Geometric mean ± 95% CI 0.615 ± 4.0 ppm 2.297 ± 1.15 ppb
Range, ppm 0 to 20.0 ppm 0 to 8.64 ppb
N 114 29
NIOSH (1991)
Geometric mean, ppm ± 95% CI 0 (LOD 0.4 ppm) 1.109 ± 0.47 ppb
Range, ppm 0.2 to 4.1 ppb
N 33 23

Extrapolation[3]
Extrapol. Exxon 9.225 ± 60 ppb
Extrapol. NIOSH 0.07 ± .03 ppm
1 / Oil Mist: Exxon measured long chain aliphatics (IHASL SOP #2.11.2). NIOSH claimed no detection (nd) for non-volatile or semi-volatile organic compounds (NIOSH Method No. 5026; LOD 0.4 ppm).
2/ PAHs/airborne: Exxon used NIOSH Method No. 5515. NIOSH used a GC/HRMS/SIM analysis (EPA Method 3630), which was 10 to 100 times more sensitive than NIOSH Methods 5506 and 5515 (LOD 25 to 200 ng/gram). These methods measure priority pollutants, not alkylated PAHs: most of airborne PAHs are alkylated PAHs.
3/ From Short & Heinze (1997): 1.5% of oil mist estimates PAH levels in Prudhoe Bay crude.

Table 4. Maximum Over Exposures

Compound	Max. Exposure	NIOSH Limit	Over Exposure
Benzene - Exxon	7.8 ppm	0.05 ppm	160
Benzene - NIOSH	0.3 ppm	0.05 ppm	6
Butoxyethanol	99 ppm	12 ppm	8.2
Carbon monoxide - Exxon	100 ppm	17 ppm	5.9
Hydrogen sulfide - Exxon	199 ppm	5 ppm	40
Nitrogen dioxide - NIOSH	0.25 mg/m^3	1 mg/m^3	0.25
Oil mist - Exxon	20 mg/m^3	0.05 mg/m^3	400
PAHs/airborne - Exxon	8.64 mg/m^3	0.05 mg/m^3	173
PAHs/airborne - NIOSH	4.1 mg/m^3	0.05 mg/m^3	82

Table 5. Over Exposures at the Upper 95% CI

Compound	Upper 95% CI	NIOSH Limit	Over Exposure
Benzene - Exxon	0.66 ppm	0.05 ppm	13
Benzene - NIOSH	0.06 ppm	0.05 ppm	1.2
Butoxyethanol - Exxon	21 ppm	12 ppm	2
Carbon monoxide - Exxon	18 ppm	17 ppm	1
Hydrogen sulfide - Exxon	33 ppm	5 ppm	7
Nitrogen dioxide - NIOSH	0.10 mg/m^3	1 mg/m^3	0.1
Oil mist - Exxon	4.6 mg/m^3	0.05 mg/m^3	92
Oil mist - extrapol NIOSH[1]	0.1 mg/m^3	0.05 mg/m^3	2
PAHs/airborne - Exxon	3.45 mg/m^3	0.05 mg/m^3	69
PAHs/airborne - NIOSH	1.58 mg/m^3	0.05 mg/m^3	32

1/ Oil mist - extrapol. NIOSH is from Table 4, using Short & Heinze (1997).

Table 7. Nature of Illness & Injury during EVOS Cleanup
Review of Alaska Dept. of Labor (1990) report, Table 3.5, p. 30.

Nature of Illness or Injury	Subtotal	Total	Percent
TOTAL		1,771	
Injury		1,135	n/a
Illnesses		636	100%
Respiratory symptoms		433	68%
Respiratory system	264		
Exposure to low temperatures	6		
Infection, parasite	46		
Pneumoconiosis	1		
Symptoms, ill-defined	113		

Occupational disease 3
Chemical symptoms
141 22%
Poisoning, systemic 34
Eye, other diseases 15
Burn, chemical 13
Dermatitis 44
Other disease NEC 35
CNS
26 4%
Nervous system 19
Cerebrovascular 5
Mental disorders 2
Other
Inflammation of joints 17 3%
Hepatitis 3 -
"No illness" 16 3%
Note: NEC = not elsewhere classified

Table 8. Source of Illness & Injury during EVOS Cleanup.
Review of Alaska Dept. of Labor (1990) report, Table 3.6, p. 31.

Source of Illness or Injury*	Subtotal	Total	Percent
TOTAL		1,771	
Injury		932	n/a
Illnesses		839	100%
Oil and/or other chemicals		123	15%
Petroleum	52		
Chemical	55		
Liquid	8		
Clothing	8		
Respiratory symptoms		259	31%
Cold environment	206		
Infection	49		
Building, structure	4		
Other		457	54%
Boiler	10		
Box, container	33		
Non classified	234		
Other sources	180		

*Note: Injuries were selected from the following categories - amputation, heat (burn), bruise, cut, fracture, abrasion, and strain. Illnesses were selected from the following categories - chemical, occupational disease, respiratory system, other classifiable, non classifiable.

Table 9. Nature of Chronic Illness based on Interviews with 62 former EVOS Cleanup Workers

Nature of Illness	Subtotal	Total	Percent
TOTAL		153	100%
Respiratory symptoms		52	34%
Respiratory system	36		
Sore throat	2		
Sinus	11		
Sinus polyps	3		

CNS 36 24%
Headaches 12
Nausea 4
Memory loss 10
Coordination loss 2
Tingly appendages 2
Stress, mood swings, depression 4
Insomnia 2
Chemical symptoms 19 13%
Poisoning, systemic 1
Chemical sensitivity 8
Skin rash, dermatitis 7
Cysts (porphyria) 3
Fatigue 13 8%
Achiness of joints 9 6%
Heart problems (arrhythmia, attacks) 9 6%
Other (< 5%)
Stomach disorders 3 2%
Diabetes 3 2%
Thyroid disorders 3 2%
Liver disorders 2 1%
Blood disorders (anemia, leukemia) 2 1%
Suppress immune system 2 1%

Table 10. Job Types of 62 former EVOS Cleanup Workers

Job Description*	Subtotal	Total	Percent
TOTAL		68	100%
Oil Response Technician - ORT		56	83%
Beach crew (wash rocks)	46		
Skiff/boat operator	6		
Barge (crane operator)	4		
Trash or wildlife (carcass) collection		4	6%
Decontamination (laundry) crew		3	4%
Inipol crew		2	3%
Diver		2	3%
Dock, boat wash		1	1%

*/ Note: 7 workers listed two jobs during the cleanup; 1 worker's job is unknown (dead).

Other Points from Interviews with 62 Former Cleanup Workers

5 of 62 workers are dead (1 of leukemia, 2 of heart attacks; 4 were ORT beach crew, 1 burned trash).
18 of 62 consider themselves disabled (respiratory lead symptom with 13 of 18; all ORTs, 16 of 18 beach crew, 2 of 18 skiff operators).
20 of 62 had 3 or more symptoms (all ORTs, 19 of 20 beach crew, 1 of 20 skiff operator).

Table 11. Total Upper Respiratory Infections (URIs) by Week
(from Veco 1989, URIs Breakdown, in *Stubblefield v Exxon*)

Week	URIs	# Workers	Percent
5/7	13	5,736	0.2
5/14	55	6,584	0.8
5/21	101	8,010	1.3
5/28	273	8,381	3.3
6/4	251	8,850	2.8

6/11 411 8,844 4.6
6/18 497 9,445 5.3
6/25 548 9,297 5.9
7/2 448 9,347 4.8
7/9 439 9,464 4.6
7/16 424 9,855 4.3
7/23 412 9,997 4.1
7/30 363 10,040 3.6
8/6 378 9,745 3.9
8/13 381 9,689 3.9
8/20 367 9,189 4.0
8/27 348 9,285 3.7
9/3 362 8,779 4.1
9/10 375 8,855 4.2
9/17 276 4,985 5.5
Total URIs = 6,722
Geometric mean = 385 URIs $\pm$ 39.45 @ 95% CI
Geometric mean percent URIs of total workers = 4.3%
First three weeks excluded from geometric mean because of concern that URIs were not reported initially.

Conclusions
Environmental pollutants present during the 1989 EVOS cleanup could cause acute and chronic health symptoms.
Workers were exposed to dangerous levels of dangerous environmental pollutants during 1989 EVOS cleanup.
Workers developed acute health symptoms from exposure to environmental pollutants during the 1989 EVOS cleanup.
There is evidence of chronic health problems potentially stemming from the 1989 EVOS cleanup.
Most EVOS cleanup workers are unaware of the potential short- and long-term health symptoms from their 1989 cleanup work.
There is a need for an epidemiological study on former EVOS cleanup workers.

Abstract Information & Notes
Colin H. Little, M.D. Date of talk: Saturday, June 8, 2002 4:00pm
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Medical School Attended: University of Melbourne
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Residency: Western General Hospital
Board Certifications: FRACP, MRCP (UK), FACA
Current Faculty Appointments:
Current Job Description: Physician & Allergist, Researcher into sensitivity disorders.
Disclosure Statement: None

SPEECH TITLE: **Immune - CNS Connections in Food/Chemical Sensitivity**
The information below has been provided by the speaker.

1.) Goals and objectives: Improved understanding as to how reactions to foods and chemicals affect the CNS.

2.) Outline of talk/abstract: Data from published studies presented evaluating the effect of TABM (T cell derived antigen binding molecules) and antigen on sensory nerve fibers

3.) Conclusion of what is to be learned: When TABM bind antigen, the associated cytokine (TGF) is activated. This cytokine acts on sensory nerves with probable relay to centers in the CNS

4.) References:

1. Arch of Environmental Health, Sept 2000, p 304-318
2. Life Sciences 1995, 57:1011-1026 Pain 1995, 63:289-302
3. Brain Research Bulletin 1997, 43:357-64
4.

IMMUNE - CNS CONNECTIONS IN FOOD AND CHEMICAL SENSITIVITY

Colin H. Little, M.D.

There have been major developments in understanding how immune activation influences brain function in the last few years. Cytokines are thought to be the mediators of this connection. The "purpose" of immune processes acting on the central nervous system is to elicit "sickness behaviour". Such behaviour is believed to mobilise resources to combat infection. Components of sickness behaviour include fever, reduced motor activity, decreased exploratory behaviour and a reduction in food and water intake. There is also a reduction in social and sexual behaviour. In animal studies immune stimuli can also be shown to impair learning and memory and cause a decrease in activities related to body care. Such animals show less brain self- stimulation which may relate to effects on motivation.

Interleukin-1 (IL-1) and TNF □ are the cytokines most studied with regard to actions on the central nervous system. However other cytokines such as IL-6 and perhaps TGF□ may be important. It was originally thought that these cytokines acted directly on the brain, crossing the blood-brain barrier where it is patent, for example the sites of the circum-ventricular organs. Although such processes may be involved where cytokine concentrations are particular high, at lower concentrations the action of cytokines appears to be mediated by their effects on sensory nerves. This suggests that the nervous system has a "sixth sense", responding to immune events producing cytokines.

Immune responses occur particularly at the interface with the environment - the gut, respiratory tract and skin. The vagus nerve is a major sensory nerve for the gut and respiratory tract. Division of the vagus nerve in animals prevents many of the effects on the central nervous system associated with the intra peritoneal injection of IL-1. Recent work indicates that sensory nerves in the skin may also relay to specific brain centres following immune stimulation. Similar considerations apply for the Trigeminal nerve which provides much of the sensory nerve supply to the upper respiratory tract. However to date most studies on immune - brain communication have involved the vagus nerve.

Considerable work has been done to trace in detail how immune stimuli acting on sensory branches of the vagus nerve relay to specific centres in the central nervous system. In the periphery dendritic cells and other immune cells are intimately associated with vagal fibres. This close association enables cytokines released by immune cells to activate vagal afferent fibres. The sensory branches of the vagus nerve relay to the dorsal vagal complex of the medulla, particularly the nucleus tractus solitarius (NTS).

The peripheral administration of IL-1 activates neurones within the NTS. There is subsequent relay from the NTS to the dorsolateral pons and ventrolateral medulla. Projections occur from these sites to

the hypothalamus, amygdala, thalamus and cortex. In addition there is an important relay from the NTS to the raphe magnus in the medulla and from there to the dorsal horns of the spinal cord. Activation of this latter pathway facilitates pain sensation. Signalling of the hypothalamus is implicated in fever and loss of appetite. Similarly, the amygdala and hippocampus are implicated in behavioural changes and impaired learning and memory associated with immune activation.

It should be noted that activation of these pathways does not produce a stereotyped effect on behaviour. Rather a motivational state is induced which will be influenced by other events affecting the central nervous system. Animal studies have shown that competing influences, for example maternal behaviour, can "override" some of the effects of immune activation on behaviour. Also the effects of stress considerably overlap those induced by immune events within the nervous system, sometimes acting at the same anatomic sites such as the hippocampus.

Although research to date concerning immune - brain communication has mainly deployed cytokines such as IL-1 and TNF or microbial products such as Lipopolysaccharide (LPS), it is quite possible that adverse reactions to foods or chemicals, if mediated by immune processes, could also affect the central nervous system. We have explored the possible role of TABM in such a process. This was done using an animal model to study the effects of purified TABM (specific to benzoic acid i.e. BA-TABM) and its associated cytokine, TGF, on sensory nerves.

We have demonstrated that TGF enhances the release of neuropeptides from sensory nerves. This effect is quite similar to that of IL-1 on sensory nerve fibres. The purified TABM (BA- TABM), associated with TGF, has a similar effect, which is dose related. These actions are not observed in Capsaicin treated animals, indicating that c fibres are involved. Finally, the effects of the BA-TABM on sensory nerves was influenced by the addition of antigen, either conjugated or unconjugated benzoic acid. Depending on its concentration, benzoic acid could either enhance or diminish the effect of BA-TABM on neuropeptide release from sensory nerves. Finally, the action of BA-TABM was blocked by anti TGF antibody.

It is possible that adverse reactions involving TABM affect adjacent sensory nerves, via the action of TGF activated when TABM bind antigen. This could alter local tissue function and signal centres within the central nervous system, as outlined above. Such a process may for example explain the disturbances in gut motility and symptoms such as malaise and headache reported in some patients with food intolerance.

The possibility of adverse reactions involving IgG antibody also acting on sensory nerve fibres, with secondary effects on other tissues is perhaps underrated. IgG1 antibody in particular, on binding to antigen, may induce mediator release from mast cells. This process can occur directly, or by the activation of complement. Mediators derived from mast cells are known to act on sensory c fibres, inducing the release of neuropeptides. Such a process could have wide spread clinical effects.

Our understanding of the affects of immunity on the brain is still limited. Although research has dealt mainly with the administration of cytokines such as IL-1 or microbial extracts, it seems reasonable to presume that immune responses induced by adverse reactions to foods or chemicals may have similar effects. Studies on the cytokine TGF could be particularly instructive. As the components of the system whereby immunity affects the brain are progressively identified, this may open up new approaches to a number of clinical problems, including depression.
Notes:

20TH ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT

SCHEDULE

Sunday, June 9, 2002

8:45 ANNOUNCEMENTS/MODERATOR: Ronald Finn, MD

9:00 Kalpana D. Patel, M.D., "Neurotoxicity Aspects of Solvents & Heavy Metals"

9:20 Q & A

9:30 Kaye H. Kilburn, M.D., "Brains, Molds and Mycotoxins"

9:50 Q & A

10:00 BREAK WITH EXHIBITORS

10:30 Russel J. Reiter, Ph.D, "Melatonin: Treatment of Free Radical - Related Diseases"

10:50 Q & A

11:00 C. Malcolm Beck, "Volcanic Rock, Energy and Health"

11:20 Q & A

11:30 SUMMARY AND CLOSE: Ronald Finn, M.D.

SUNDAY, JUNE 9, 2002
ABSTRACTS
AND
HANDOUTS

Abstract Information & Notes

Kalpana D. Patel, M.D. Date of talk: Sunday, June 9, 2002, 9:00am
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Medical School Attended: BJ Medical School
Major and date of Graduation: 1967
Residency: Bexar County Hospital, San Antonio, Texas
Board Certifications: Pediatrics/Environmental Medicine
Current Faculty Appointments: President of American Board of Environmental Medicine, Associate Prof. Pediatrics Suny Buffalo
Current Job Description: Director of Environmental Health Center Buffalo
Disclosure Statement: None

SPEECH TITLE: **Neurotoxicity Aspects of Solvents & Heavy Metals**
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 Syndrom, 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 deputation, and physical therapy to reduce total toxic load of chemicals and toxic heavy metals.

4.) References:
Notes:

Abstract Information & Notes
Kaye H. Kilburn, M. D. Date of talk: Sunday, June 9, 2002, 9:30am
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Disclosure Statement: None

SPEECH TITLE: **Brains, Molds and Mycotoxins**
The information below has been provided by the speaker.

1.) Goals and objectives: Present neurobehavioral findings in 20 patients exposed to molds.

2.) Outline of talk/abstract: Ten New York and ten California-Arizona patients showed impaired function associated with homes or apartments that had molds growing due to water intrusions. They resemble other chemically exposed patients.

3.) Conclusion of what is to be learned: Molds appear to contribute to or cause some indoor air problems.

4.) References:
K.H. Kilburn: Inhalation of molds and mycotoxins. European J. Cancer, in press, 2001

BRAINS, MOLDS AND MYCOTOXINS
Kaye H. Kilburn, M.D.
Ralph Edgington Professor of Medicine
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Forty five patients exposed to molds and mycotoxins indoors showed neurobehavioral and pulmonary impairment. All were exposed in homes with water intrusion into walls from structural defects, leaky pipes, air-conditioning condensers and icemaker tubes. Two thirds were women. Ages varied from 17 to 79, average 45 years, and education from 9 to 20 years, average 14 years. Abnormalities were determined by testing 12 physiological functions: balance, reaction time, blink reflex, grip strength, color discrimination, visual field performance, hearing and vibration. Fourteen psychological tests measured problem solving (Culture Fair), digit symbol substitution, recall memory, concentration, concept juggling, peg placement, trail making, vocabulary, and long term memory (using information, picture completion and similarities).

Pulmonary volumes and flows were assessed by spirometry. Respiratory flows and vital capacities were measured from a full inspiration while subjects stood and expired using a nose clip into a volume displacement (Ohio) spirometer. This maneuver was repeated until two forced expirations agreed within 5% (16). Records of volume and flows were traced with a digitizer and measured by a computer. Prediction equations adjusted for height, age, sex and smoking status (15).

Abnormal tests, neurobehavioral and pulmonary, were defined as beyond the confidence limits of predicted values that adjusted for age, sex, height, weight and years of educational achievement. The 45 patients averaged 6 abnormalities (range 0-21) and median 5. Ten had 9 or more, abnormally fast sway for balance with eyes closed was found in 20 and prolonged choice reaction time in 15, 16 had poor vibration sense in the feet. Mental tests were less frequently abnormal. Profile Of Mood States scores were elevated at 67 (mean) and symptom frequencies averaged 5 (mean), twice normal values. Thirteen had airways obstruction showed by abnormal FEV1/FVC or FEV1 and 7 more had small airways obstruction due to reduced FEF 75-85 for a total of 20, 44%.

These observations alert us to the probability that mold exposures produce sub-clinical neurobehavioral abnormalities short of tremors (3), convulsions and hallucinations (2). Studies of larger numbers of affected people and characterization of their mold exposures should identify associations to answer questions about causation. If molds are incriminated serum measurements of antibodies to mycotoxins rather than molds, may provide the best indication, particularly trichothecenes and toxic chemical compounds. These agents had two 6 carbon rings may be a link in understanding the indoor air mystery. Mold exposures indoors are associated with neurobehavioral impairment and evidence of small airways obstruction (33,34). Newly observed abnormalities are for balance, simple and choice reaction time, errors in color discrimination, recall memory and cognitive and problem solving ability. These 45 subjects showed no evidence of malingering.

There are three major mycotoxin related human diseases (1). Vascular obstruction and necrosis occur, the adrenergic activity of ingested ergot alkaloids from Claviceps species which also stimulate the hypothalamus and midbrain, depress the vasomotor center and act centrally to cause emesis. Secondly, alimentary toxic aleukia was described in Russia from eating moldy overwintered grain containing Fusarium (Poae and Sporotrichicides) after the siege of Stalingrad in World War II. The toxins are sesquiterpinoids related to the trichothecenes of Stachybotrys. Third is liver cancer from consumption of Aflatoxin B_1 contaminated food, particularly in malnourished people (35). The association of hepatoma with aflatoxin consumption above 100 □g/kg in foods is strong in Uganda and other sub-Sahara African countries. Beginning in 1967 the association of aflatoxin in peanuts, dry corn and millet and hepatoma was found in Kenya (36) and in Thailand (37). Many animal studies show that as little as 1 □g/kg of aflatoxin on the diet produces hepatomas in rats and lesser doses causes them in trout. Teratogenic effects in animals from cytochalasin and damage to developing rodent's lungs (1) suggest problems that human populations may develop, especially people who are displaced and ill fed.

To investigate the connection of molds growing indoors to impairment of brain and lung, inspection of walls for mold growth should be combined with samples of dust, usually wiped from surfaces, and examined for spores. Molds can be grown on agar by uncovering plates or taking air samples obtained with an Anderson sampler. Indoor and outdoor samples should be quantified and compared. There are chemical assays of dust for mycotoxin (aflatoxin, coumadins, zearalenones and trichothocene) using thin layer chromatography on silica gel and identification by charring and by treating with 4 (p-nitrobenzyl) pyridine. Liquid chromatography is used for final separation with identification using mass spectrometer.

There is no blood assay for mycotoxins but reactive antibodies can be measured to trichothene, aflatoxin and zearalenones.

Rapid development of analytical capacity is predicted, driven by the need to have information to correlate with the human impairments and the desire to show quantitative relationships. Opportunities for mold growth on paper surfaces within walls must be stopped. All the molds feed on cellulose. For prevention perhaps impregnating the paper with a growth inhibitor such as boric acid during its manufacture would suffice. Cleaning inner plaster board surfaces, even with chlorinated water or other oxidants, is ineffectual so replacement of moldy plaster board seems mandatory for remediation.

Conclusion

Mycotoxins, an age old problem (1,2) contribute to the adverse effects of indoor air (14) on neurobehavioral functions and airflow. Mold amelioration reduces respiratory problems in school children (34). Reducing mold toxins in foods decreases hepatocellular cancers. Further studies using similar methods, particularly objective neurobehavioral measurements and mycotoxin levels in air and serum should characterize this syndrome and may correlate with specific molds toxins encountered indoors.

Notes:

Abstract Information & Notes

Russel J. Reiter, Ph.D. Date of talk: Sunday, June 9, 2002, 10:30am
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Other Information: Editor-in-Chief, Journal of Pineal Research; Editorial Board of 14 other journals; Numerous research awards including A. Ross McIntyre Gold Medal, U.S. Senior Scientist von Humboldt Award (Germany), Lisoni Lincee Award (Italy), Inaugural Distinguished Scholarship Award (Univ. Texas), Invited speaker at 300+ international symposia, etc.
Disclosure Statement: None

SPEECH TITLE: **Melatonin: Treatment of Free Radical Related Diseases**
The information below has been provided by the speaker.

1.) Goals and objectives: 1) To identify those diseases and disease states where melatonin has been shown to be an effective treatment; 2) To summarize the nature of what constitutes a free radical-related disease.

2.) Outline of talk/abstract: See abstract

3.) Conclusion of what is to be learned: Why melatonin is an effective co-treatment in certain disease states and future perspectives on its use.

4.) References:

1. R. Gitto... R.J. Reiter et al, Pediatr. Res. 50:756-760, 2001;
2. F. Fulia... R.J. Reiter et al, J. Pineal Res. 31:343-349, 2001;

3. M. Karbownik.. R.J. Reiter et al, Int. J. BioChem. Cell Biol. 33:735-753, 2001.

MELATONIN: TREATMENT OF FREE RADICAL RELATED DISEASES
Russel J. Reiter, Ph.D.

Melatonin is a non-toxic molecule over a very wide range of doses even when given for prolonged periods (years). Recent studies show its utility in treating free radical-related diseases. In particular, melatonin has been found to reduce the severity of septic shock in premature newborn infants.

Melatonin treatment in these children reduces the clinical measures (white blood cell count, C reactive protein, etc.) of sepsis and improves their survival. Likewise, in newborn infants who experience asphyxia during birth, pharmacological melatonin treatment improved their outcome as well. Both these conditions in newborns as free radical-related. In adults, melatonin treatment has been shown to greatly reduce the signs of tardive dyskinesia following treatment with neurally active drugs. Also, co-treating cancer chemotherapy patients with melatonin prolongs their survival, improves their general well being, and reduces the severity of myeosuppression and thrombocytopenia caused by chemotherapeutic agents. Finally, melatonin has been shown effective in slowing the progression of Alzheimer's disease.

Abstract Information & Notes
C. Malcolm Beck Date of talk: Sunday, June 9, 2002, 11:00am
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Disclosure Statement:

SPEECH TITLE: **Volcanic Rock, Energy and Health**
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:

VOLCANIC ROCK, ENERGY & HEALTH
C. Malcolm Beck

In the beginning there were no plants, or any life, on Earth. There was no life on Earth because there was no soil to support life--but there was no soil because it takes life forces to create productive soil. At some point the Almighty saw fit to breathe life on Earth. It was a very primitive form of life; it could live on rock, feed on and etch away at the rock. This micro life would exude, die and decompose on the rock. This process went on and on until very small amounts of our first soil was formed.

Even though extremely small, the life, death and decay of these life forms created conditions for more and higher life forms. After countless centuries of creating soil more complex and, still higher, forms of life could exist. Finally Man, the most complex of all life could exist and be sustained.

Basalt, lava and granite were some of the first rock that was etched, eroded and dissolved by the first life forms into soil. Because of the once extreme heat and possibly because of the centrifugal force of

the spinning earth these rocks held a slight magnetic energy, or paramagnetic energy, this energy was recognized by the ancients and used in their mighty rock structures.

Paramagnetic energy was studied by Phillip S. Callahan PhD and published in a book in 1995 entitled, "Paramagnetism". I studied all of Callahan's writings and learned to know and visit him personally. And have done several years of research using paramagnetic materials. I discover that heat could make certain clay and rock minerals paramagnetic. And that high centrifugal force can also make non-ferrous materials paramagnetic.

My most astounding discovery was, how paramagnetic rock could enhance the growth of plants and help plants overcome stresses of heat, cold, diseases, insects and even herbicides.

Blended in fertile soil or mixed with compost, paramagnetic materials perform the best. When used in the soil around tomato plants they withstood sustained 28-degree temperature without the slightest evidence that a freeze ever occurred when all other tomato plants for miles around were frozen to the ground. I also used compost and paramagnetic rock sand blended in the soil when 7 olive were planted. I gave 14 of the same age and species of olive to surrounding neighbors to put in their landscape. The following year when all the trees were 3 to 4 ft. tall we had a hard freezes, the temperature dropped to 16-degree. My trees fed with paramagnetic sand had a few slightly nipped leaves while the other 14 olive trees were frozen to death.

Every test preformed with the paramagnetic material was checked against a control and with every test there were positive results.

I now have a meter to test the degree of paramagnetism of materials and have discovered that materials lose paramagnetism while exposure to the elements. However, the exposure time is measured in centuries.

The first plants to evolve on earth were growing in mineral rich, paramagnetic soil. This low level energy evidently has beneficial influences on plants and possible all life. With plants it causes the sap to load up with minerals and sugars, which are known to give plants resistance to insects and diseases. The concentration of minerals and sugars in the sap also gives it much lower freezing point.

Research and logic has proven. It takes a healthy soil to grow a healthy plant and a healthy plant to grow a healthy body and a healthy body supports a sound mind. Paramagnetic soils are one component of that health.