21st Annual International Symposium
on
Man and His Environment in Health and Disease

Special Focus

Innovative Aspects and Treatment of Molds, Mycotoxins 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 (ACCME) 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 21.5 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 2003 conference will focus on "Innovative Aspects and Treatment of Molds, Mycotoxins 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

1. # Plenary
2. # Panels Discussions
3. # Case Studies
4. # Question & Answer Sessions.

CONFERENCE FORMAT

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

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

GIVEN IN COOPERATION

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

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

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

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

Allan D. Lieberman, M.D.
Center for Occupational Environmental Medicine
North Charleston, SC

21st ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT

SCHEDULE

Thursday, June 19, 2003

7:00 a.m.	**REGISTRATION**
8:50	**WELCOME/MODERATOR: William J. Rea, M.D.**
9:00	**Douglas B. Seba, Ph.D.,** Independent Marine Scientist, Alexandria, VA: **"Environmental Update 2003: Molds, Dust, Global Warming"**
9:20	Q&A
9:30	**Tapani Tuomi,** Laboratory Chief, Finnish Institute of Occupational Health, Helsinki, Finland: **"Mycotoxins in Indoor Climates"**
9:50	Q & A
10:00	**BREAK**
10:30	**William J. Meggs, M.D.,** Professor of Toxicology, Dept. of Emergency Medicine, E. Carolina Univ. School of Medicine, Greenville, NC: **"Systemic Anaphylactic Reactions to Molds and Other Aeroallergens"**
10:50	Q & A
11:00	**William J. Rea, M.D.,** Director, Environmental Health Center B Dallas, Dallas, TX: **"Diagnosis of Mold & Mycotoxin Sensitivity"**
11:20	Q & A
11:30	**Andrew W. Campbell, M.D.**, Clinical Immunotoxicologist, Center for Immune, Environment and Toxic Disorders, Spring, TX: **"Immunological and Neurophysiological Abnormalities in Adults with Exposure to Molds"**
11:50	Q & A
12:00 p.m.	**Lunch in the Primebird Restaurant**
	MODERATOR: Wallace Rubin, M.D.
1:30	**Professor Tang G. Lee, AAA,** Professor**,** Faculty of Environmental Design, University of Calgary, Calgary, Alberta, Canada: **"Molds in Native Housing and SIDS Potential"**
1:50	Q & A
2:00	**William A. Croft, D.V.M., Ph.D.,** Private Practice, Mycotoxins, Environmental Diagnostic Group Inc., Madison, WI: "**Pathology of Trichothecene Mycotoxins in Man"**
2:20	Q & A
2:30	**Kaye H. Kilburn, M.D.,** Director of Environmental Sciences Lab, Ralph Edgington Professor of Medicine, University of S. California Medical Center**,** Keck School of Medicine, Los Angeles, CA: "**How Molds and Mycotoxins Affect Human Brains"**
2:50	Q & A
3:00	**BREAK**
3:30	**Kalpana D. Patel, M.D.**, Director of Environmental Health Center Buffalo, Northwest Center for Allergy & Environmental Medicine, Buffalo, NY: "**What is New and Different in the Diagnosis and Management of Different Skin Disorders B Itching Eczema and Urticaria"**
3:50	Q & A
4:00	**Katherine Warsco, Ph.D.,** Department of Interior Design, East Carolina University, Greenville, NC: "**Teaching Design for Good Indoor Air Quality"**
4:20	Q & A
4:30	**Michael R. Gray, M.D., M.P.H.,** Internal Occupational Medicine Certified Independent Medical Examiner, Progressive Health Care Group, Benson, AZ: "**Molds, Mycotoxins & Public Health: a Clinicians Perspective"**
4:50	Q & A
5:00	**Panel Discussion: How to evaluate moldy house?** Chris Rea, William J. Rea, M.D., Geoffrey Hutton, William Croft, D.V.M., Ph.D., and Larry Foster
6:00	**AJOURN**

THURSDAY, JUNE 19, 2003

ABSTRACTS
AND
HANDOUTS

Abstract Information & Notes

Douglas Seba, Ph.D.

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

Date of talk: Thursday, June 19, 2003, 9:00am

Phone: 703/949-1055
Fax: N/A
E-mail: N/A

Major and date of Graduation: Environmental Oceanography - 1970
Current Job Description: Independent Marine Scientist
Other Information: Forty years experience in Ecology and Chemicals

Disclosure Statement: None

SPEECH TITLE: **"Environmental Update 2003: Molds, Dust, Global Warming"**

The speaker has provided the information below.

1.) Goals and objectives: To review selected environmental phenomena that contributes to patient exposure to biochemicals and molds

2.) Outline of talk/abstract: Molds, xenobiotics, genetics, dust, global warming, fate and transport mechanisms, and wildlife anomalies will all 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 origins and put physicians and patients in a constant state of exposure and challenge.

4.) References: Taken from a broad spectrum of media, websites and scientific publications relevant to the moment.

Environmental Update 2003: Molds, Dust Global Warming

Douglas B. Seba

The world is both a moldy and dusty place. Both may be increasing in the natural environment perhaps aided by global warming. There also appears to be an increase in these moieties in indoor environments as we spend increasing amounts of time in air conditioning. Certainly structural mold insurance claims have increased greatly in states, like Texas and Florida, where climate change has increased warmth. Florida is also first among all large states in both total cancer and increase in pediatric cancers being over double that of California, for example. Dust from Africa, containing numerous molds, has also increased in Florida over the last few decades, as well as throughout the Caribbean and the entire United States as far west as New Mexico and north to Canada. Additionally, relative humidity has increase about 10% over the fifty years in the Caribbean and extending into the southeast U.S. All of these factors combine to make more nutrients and moisture available for mold growth. Asthma cases are also increasing nationally, but the increase in the Southeast is outpacing the rest of the country and there is a connection between molds/dust and asthma.

African dust is a quantitative source of hormonally active environmental agents. Global assessment of endocrine disrupters show pervasive distribution throughout the environment including the human body. Recent work with bisphenyl A at very low levels inducing highly significant increases in chromosomal aberrations in mouse eggs, frog deformities caused by interaction between parasites and atrazine, or degradated fluorinated telomers found in human blood are contemporary examples of these environmental agents.

These agents profoundly affect the state of your health as they trigger genes that would not otherwise be expressed. Thus, the nascent field of toxicogenomics will rapidly expanded as the role of endocrine disruptors as biomarkers is investigated. These continuing and emerging sciences will also change the focus of environmental regulation.

Examples of the items will be personally applied by the author to ongoing research in wildlife anomalies in the Bitterroot Mountains of Montana.

Abstract Information & Notes

Tapani Tuomi

Finnish Institute of Occupational Health (FIOH)
Arinatie 3 A
Helsinki, Finland FIN-00370

Phone: 358-9-47472926
Fax: 358-9-5061087
E-mail: tapani.tuomi@occuphealth.fi

Date of talk: Thursday, June 19, 2003, 9:30am

School Attended: Helsinki University of Technology
Major and date of Graduation: DR, Chemical Engineering (Applied Microbiology), 1995

Current Faculty Appointments: Docent in Environmental Chemistry and Microbiology, Helsinki Univ. of Technology

Current Job Description: Laboratory Chief, Laboratory of Chemistry and Microbiology, Finnish Inst. Of Occupational Health, Helsinki, Finland

Disclosure Statement: None

SPEECH TITLE: **"Mycotoxins in Indoor Climates"**

The speaker has provided the information below.

1.) Goals and objectives: To present current literature on the presence of mycotoxins in indoor climates and to discuss the possibility for carry-over of mycotoxins from contaminated indoor surfaces to air.

2.) Outline of talk/abstract: It has been recognized that mycotoxin-producing fungi can proliferate and produce mycotoxins in damp building materials in water-damaged building. Mycotoxins are also frequently found in deposited dust from indoor environments. There is very little evidence, however, on the presence of mycotoxins in indoor air. This suggests that the air-concentration of mycotoxins even in buildings hampered by long-standing water-damage and following mold-damage is below the limit of detection of contemporary methods of analysis. The talk will examine the spectra of mycotoxins found on building materials naturally contaminated by fungi, as well as the evidence on the presence of mycotoxins in inhalable air in damp buildings.

3.) Conclusion of what is to be learned: A wide range of mycotoxins are potentially present in indoor climates harboring moldy surfaces. It has proven difficult, however, that mycotoxins may contribute to the variety of symptoms experienced by patients exposed to moldy propagules in indoor climates.

4.) References: Skaug et al., 2001, Mycopathologia, 151:93-8, Page and Trout, 2001, AIHAJ 2001 Sep-Oct; 62(5):644-8, Peltola et al, 2001, Appl Environ Microbiol. 2001, 67:3269-74, Tuomi et al., 2000, appl. Environ. Microbiol, 66:1899-1904

Mycotoxins in Indoor Climates

Tapani Tuomi

As of present, analyzing for mycotoxins in indoor environments is difficult, if the goal is to assess the health consequences of extensive water damage on the occupants of a particular building. There is accumulating evidence on the presence of mycotoxins in crude building materials[1-7] as well as a body of indirect evidence linking the presence of mycotoxins in indoor environments to health problems[5, 8-15]. It is frequently maintained that mycotoxins present in bulk materials infested with toxigenic fungi are carried to indoor air by fungal propagules. It follows that the route of exposure to mycotoxins in indoor environments is inhaling dust particles containing toxigenic fungal propagules[2].

Dose-responses of humans to airborne mycotoxins are not known and it seems that mycotoxin concentrations in inhalable dust would have to be some 100-fold higher than what is frequently encountered in indoor environments for air sampling to be feasible on a general level. If air sampling is not attempted, deposited dust constitutes one step closer to the composition of indoor air with respect to mycotoxins. There are numerous studies from agricultural environments establishing that mycotoxins present in bulk material are - given the right circumstances - carried into dust. For instance, trichothecene concentrations of 0,1-1 µg/g dust, aflatoxin concentrations of 0,02-5 µg/g dust, ochratoxin A concentrations of 0,2-70 ng/g dust, and zearalenone concentrations of 20-100 ng/g dust have been reported during grain handling and from other agricultural settings[16-21]. In laboratory settings, Sorensen et al.[22] found satratoxin concentrations in the 10 µg/g dust-range, whereas Smoragiewicz et al.[6] detected trichothecenes in deposited dust from a moisture problem building in amounts exceeding 0.4-4 µg/g and Engelhart et al.[23] found sterigmatocystin (2-4 ng/g) in carpet dust from a damp indoor environment. It follows that samples of deposited dust should be considered alongside with bulk samples when assessing the presence of mycotoxins in indoor environments.

In agricultural settings, aflatoxin concentrations of 0,01 - 1000 ng/m^3 and eoxynivalenol (DON) concentrations of 3-20 ng/m^3 have been reported in air[17-18, 20-21, 24-25]. In indoor environments, satratoxin in concentrations of 0,1-0,5 ng/m3 and unidentified trichothecenes in concentrations of 1-35 ng/m^3 have been found[22-23]. It seems therefore, that irrespectively of the environmental setting, whether agricultural or indoor environments, measurement of airborne mycotoxins generally require use of high-volume samplers in combination with sensitive chemical or immunological methods of analysis. Risk-assessment on the inhalation of mycotoxins cannot be made based on the analysis of bulk samples of construction materials. Neither can mycotoxin contents of deposited dust serve as basis of risk-assessment. Therefore, with the development of more efficient methods of sampling and analysis, air sampling will help us better understand the health consequences of exposure to mycotoxins in indoor climates and perhaps will at some point enable estimation of dose-responses of humans to airborne mycotoxins.

In conclusion, a wide range of mycotoxins are potentially present in indoor climates harboring moldy surfaces. It has proven difficult, however, to establish the presence of mycotoxins in indoor air. This does not take away from the fact, however, that mycotoxins may contribute to the variety of symptoms experienced by patients exposed to moldy propagules in indoor climates.

REFERENCES: [1]Andersson et al., Appl Environ Microbiol, 1997, 63: 387-393; [2]Croft et al., Mycopathologia, 1986, 151:93-98; [3]Flappan et al., Environ Health Perspect, 1999, 107: 927-930; [4]Gravesen et al., Environ Health Perspect, 1999, 107: 505-508; [5]Johanning et al., Int Arch Occup Environ Health, 1996, 68: 207-218; [6]Smoragiewicz et al., Int Arch Occup Environ Health, 1993, 65: 113-7; [7]Tuomi et al., Appl Environ Microbiol, 2000, 66, 1899-1904; [8] Auger et al., Am J Ind Med, 1994, 25: 41-2; [9]Hodgson et al., J
Occup Environ Med, 1998, 40: 241-9; [10]Miller, Atm Environ, 1992, 26A: 2163-2172; [11]Morb Mortal Wkly Rep, 1994, 43: 881-883; [12]Morb Mortal Wkly Rep, 1995, 44: 67-74; [13]Morb Mortal Wkly Rep, 1997, 46: 33-35; [14]Rautiala et al., Am Ind Hyg Assoc J, 1996, 57: 279-84; [15]Smith et al., Fems Microbiol Lett, 1992, 79:337-43; [16] Lappalainen et al., Atmosph Environ, 1996, 30, 3059-3065; [17]Burg et al., Am Ind Hyg Assoc J, 1981, 42:1-11; [18]Burg et al., Am Ind Hyg Assoc J, 1982, 43:580-587; [19]Silas et al., Am Ind Hyg Assoc J, 1987, 48:198-201; [20]Selim et al., Am Ind Hyg Assoc J, 1998, 42:252-256; [21]Palmgren et al., Am Ind Hyg Assoc J, 1983, 44:485-488; [22]Sorenson et al., Appl Environ Microbiol, 1987, 53: 1370-5; [23]Engelhart et al., Appl Environ Microbiol, 2002, 68:3886-3890; [24]Ghosh et al., Am Ind Hyg Assoc J, 1997, 58:583-586; [25]Kussak, Ph.D. Thesis, Umeå University, Umeå, Sweden, 1995. [22]Johanning et al., Unpublished data pertaining to filter no. 1 in *Johanning et al., Proceedings: Indoor air 2002*; 23Yike et al., Appl Environ Microbiol, 1999, 65: 88-94.

Abstract Information & Notes

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

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

Phone: 252/744-2954
Fax: 252/744-3589
E-mail: meggsw@mail.ecu.edu

Date of talk: Thursday, June 19, 2003, 10:30am

Medical School Attended:	University of Miami
Major and date of Graduation:	M.D., 1979
Residency:	University of Rochester
Board Certifications:	Medical Toxicology, Allergy & Immunology, Internal Medicine, Emergency Medicine
Current Faculty Appointments:	Professor & Chief of Toxicology
Current Job Description:	Physician
Other Information:	Author of "The Inflammation Cure" to be published in Sept. 2003. Editor of "Health & Safety in Agriculture, Forestry, & Fisheries." Author of numerous research articles and textbook chapters.
Disclosure Statement:	None

SPEECH TITLE: "**Systemic Anaphylactic Reactions to Molds and Other Aeroallergens"**

The speaker has provided the information below.

1.) Goals and objectives:
$ To present the signs, symptoms, treatment, epidemiology, and prognosis of systemic anaphylaxis
$ To discuss the clinical situations in which systemic anaphylaxis can occur to aeroallergens
$ To discuss the mechanisms of systemic anaphylaxis

2.) Outline of talk/abstract: Aeroallergens are proteins found in the air on mold spores, pollen grains, and from animals. Humans become sensitized by the production of IgE antibodies to these antigens. Most commonly aeroallergen exposures are by inhalation, and the most common symptoms are rhino sinusitis, conjunctivitis, and asthma. Less commonly, reactions such as urticaria, dermatitis, and systemic anaphylaxis can occur from inhalation exposures to aeroallergens. The clinical presentation, diagnosis, treatment, and mechanism of systemic anaphylaxis will be discussed, and situations in which systemic anaphylaxis can occur from aeroallergen exposures will be presented. These include inhalation induced systemic anaphylaxis, dermal exposures, the alpine slide syndrome, and ingestion of honeybee pollen containing aeroallergens.

3.) Conclusion of what is to be learned: Aeroallergens can lead to systemic anaphylaxis from inhalation, ingestion, and dermal exposures.

4.) References:
Chivato T, Juan F, Montoro A, Laguna R. Anaphylaxis induced by ingestion of a pollen compound. J Investig Allergol Clin Immunol 1996 May-Jun; 6(3): 208-9.

Eriksson NE, Formgren H, Svenonius E. Food hypersensitivity in patients with pollen allergy. Allergy 1982 Aug; 37(6): 437-43

Mansfield LE, Goldstein GB. Anaphylactic reaction after ingestion of local bee pollen. Ann Allergy 1981 Sep; 47(3): 154-6

McGrath KG. Anaphylaxis. In Grammar LC, Greenberger PA, eds., *Patterson=s Allergic Diseases. 6th Edition.* Lippncott Williams & Wilkins. Philadelphia, 2002. Chapter 20, pp 415-436.

Patterson R, Harris KE. Idiopathic Anaphylaxis. In Grammar LC, Greenberger PA, eds, *Patterson=s Allergic Diseases. 6th Edition*. Lippncott Williams & Wilkins. Philadelphia, 2002. Chapter 20, pp 415-436.

Spitalny KC, Farnham JE, Witherell LE, Vogt RL, Fox RC, Kaliner M, Casale TB. Alpine slide anaphylaxis. N Engl J Med 1984 Apr 19;310(16):1034-7

Abstract Information & Notes

William J. Rea, M.D.

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

Phone: 214/368-4132
Fax: 214/691-8432
E-mail: wjr@ehcd.com

Date of talk: Thursday, June 19, 2003, 11:00am

Medical School Attended:	Ohio State University College of Medicine
Major and date of Graduation:	M.D., 1962
Residency:	UTSWS
Board Certifications:	American Board of Surgery, American Board of Thoracic Surgery, American Board of Environmental Medicine
Current Faculty Appointments:	Professor of Medicine, Capital University of Integrative Medicine, Washington, DC
Current Job Description:	President, Environmental Health Center - Dallas
Disclosure Statement:	None

SPEECH TITLE: **"Diagnosis of Mold and Mycotoxin Sensitivity"**

The speaker has provided the information below.

1.) Goals and objectives: 1.) To understand the cause. 2.)Understand the diagnostic tests. 3.) To understand their value in pulmonary function.

2.) Outline of talk/abstract: Mold Tests used in diagnosing - building inspection and mold plates, SPECT-Brain Scan, autonomic nervous system, evaluation through pupillography and HRV, balance test, psychological - neuro-evaluation, blood tests, skin tests, and urine test for mycotoxins.

3.) Conclusion of what is to be learned: How to diagnose and individual with mold exposure

4.) References: Chemical Sensitivity, Volume 2, 3

Abstract Information & Notes

Andrew W. Campbell, M.D.

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

Phone: 281/981-8989
Fax: 281/681-8787
E-mail: md@immunotoxicology.com

Date of talk: Thursday, June 19, 2003, 11:30am

Medical School Attended: Universidad Autonoma de Guadalajara, Mexico, School of Medicine

Major and date of Graduation: M.D., June 1974

Residency: 1974-1975 - Guadalajara, Mexico, Pediatrics, Obstetrics & Gynecology; 1977 - Resident, General Surgery Orlando Regional Medical Center, Orlando, Florida; 1978 - Resident, Family Medicine, Department of Family Practice, Medical College of Georgia, Augusta, Georgia

Board Certifications: American Board of Family Practice, American Board of Forensic Examiners, and American Board of Forensic Medicine

Current Job Description: Private Solo Practice

Other Information: Published several articles; Recent awards include Marquis Who=s Who in America and Marquis Who=s Who in Medicine and Healthcare

Disclosure Statement: None

SPEECH TITLE: **"Immunological and Neurophysiological Abnormalities in Adults with Exposure to Molds"**

The speaker has provided the information below.

1.) Goals and objectives: Understanding the effects of toxigenic fungi and mycotoxins as they affect humans, especially neurotoxicity.

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:

4.) References: Will provide list

Immunological and Neurophysiological Abnormalities in Adults with Exposure to Molds

Andrew W. Campbell, M.D.
William High, M.D., Ph.D.
Ebere Anyanwu, M.S., Ph.D.
Medical Center for Immune and Toxic Disorders
Houston, Texas

Objective: The objective of this study was to evaluate the immunological and neurophysiological effects in patients (378) who presented to our medical center with various adverse health problems due to documented exposure to indoor toxigenic molds. Exposure to indoor toxigenic molds and the subsequent effects on humans is ranked high among environmentally related disorders. Recently, occupational exposures in nonagricultural settings have been investigated using modern immunological laboratory tests. Few studies exist that take into account the combined immunological and neurophysiologic effects in humans.

Methods: We studied retrospectively patients with documented toxigenic mold exposure at measured levels in their residence using previous medical records, questionnaires, serum testing for antibodies to molds, serum immune function testing and neurophysiological testing including electroencephalogram (E.E.G.), brainstem auditory evoked response (B.A.E.R.), visual evoked potentials (VEP), and nerve conduction velocity (NCV).

Results: Findings from indoor environmental studies on the patients' residence (exposure site) were positive for specific levels of exposure to toxigenic molds including Penicillium, Aspergillus, Fusarium, Chaetomium, and Stachybotrys species. There was a positive correlation between findings from the neurophysiological and immunological studies and the exposure to indoor molds found in the residence. The objective immunological and neurophysiological findings were significantly abnormal, indicating both immunotoxic and neurotoxic effects.

Conclusions: A statistically significant number of patients with known chronic exposure to toxigenic molds developed immunologic and neurophysiologic abnormalities. Our findings revealed the extent to which toxigenic molds can affect the immunological and neurological systems of environmentally exposed individuals. Further work is encouraged in this regard.

Abstract Information & Notes

Professor Tang G. Lee, AAA

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

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

Date of talk: Thursday, June 19, 2003, 1:30pm

Major and date of Graduation:	Site planning and architecture, 1975
Current Faculty Appointments:	Professor of Architecture (Building Science and Environmental Health). Also Adjunct Professor at the University of Manitoba, and visiting scholar at the Lyle center for Regenerative Studies, California State Polytechnic University, Pomona
Current Job Description:	Conducting research investigations and teaching environmental health, particularly indoor air quality, building science and sustainability.
Other Information:	Conducts comprehensive indoor air quality investigations in an interdisciplinary team for those cases that could not be solved by other indoor air quality consultants. Also designs buildings such as medical clinics, institutions and residences that feature low toxicity.
Disclosure Statement:	None

SPEECH TITLE: **"Molds in Native Housing and SIDS Potential"**

The speaker has provided the information below.

1.) Goals and objectives: To understand the environmental conditions of native housing and its impact on occupant health. To develop appropriate housing design, construction and maintenance of native housing to minimize building deterioration and resulting health impacts.

2.) Outline of talk/abstract: Many native houses are built without regards to climate and cultural needs. Premature deterioration of these houses created conditions for microbial amplification. The resulting occupant symptoms reduced their potential for achieving a quality of life and may even aggravate SIDS. Recommendations for proper site planning, design and maintenance is presented.

3.) Conclusion of what is to be learned: Building deterioration will impact occupant health and well-being. Better quality buildings are needed to ensure occupant health.

4.) References:

Wilson, C.E. ***Sudden infant death syndrome and Canadian Aboriginals: bacteria and infections.*** FEMS Immunology and Medical Microbiology 25 (1999) 221-226. Federation of European Microbiological Societies, Elsevier Science.

Lee, T.G. and Stooke, T. ***Mould propagation resulting from air pressure differences across the building envelope.*** *Proceedings of the 9th International Conference on Indoor Air Quality and Climate (Indoor Air 2002)*, Monterey, California, June 30 B July 5.

Abstract Information & Notes

William A. Croft, D.V.M., Ph.D.

Environmental Diagnostic Group Inc.
521 Hilltop Dr.
Madison, WI 53711

Phone: 715/757-3756
Fax: 715/757-9302
E-mail: doccroft@hotmail.com

Date of talk: Thursday, June 19, 2003, 2:00pm

Veterinary School Attended:	University of Minnesota
Medical School Attended:	University of Wisconsin, Madison, Wisconsin
Major and date of Graduation:	Ph.D. in Medical Pathology from the University of Wisconsin, Madison, Wisconsin.
Current Job Description:	Study Human diseases within the environment from outbreak of human disease as a Medical Pathologist.
Other Information:	Was on Faculty of the University of Wisconsin as Medical Pathologist, was accepted by the National Institute of Health as a Medical Pathologist, qualified to research human diseases. Obtain over $900,000 of highly competitive research grants from the national Institute of Health while at the University of Wisconsin.
Disclosure Statement:	None

SPEECH TITLE: **"Pathology of Trichothecene Mycotoxins in Man"**

The speaker has provided the information below.

1.) Goals and objectives: To demonstrate the pathologic changes in the primary target organs after inhalation verses ingestion exposure to Trichothecene Mycotoxins in man.

2.) Outline of talk/abstract: A. History of Mycotoxicosis, B. Detection of "Sick Buildings" ingestion verses inhalation exposure. Signs and Symptoms expressed by over 6,000 patients exposed to Trichothecene Mycotoxins, attempting to establish diagnosis. C. The pathologic changes associated with inhalation exposure to trichothecene mycotoxins. D. The primary organs involved with inhalation Mycotoxicosis.

3.) Conclusion of what is to be learned: The primary target organs of this disease and how this mycotoxin affects every cell in the body.

4.) References:

a. Croft, W.A., Jarvis, B.B., and Yatawara, C.S.,: Airborne Outbreak of Trichothecene Toxicosis, In: Atmospheric Environ, 20(3), 549-552 (1986).
b. Croft, W.A., Jastromski, B.M., Croft, A.L., and Peters, H.A., "Clinical Confirmation of Trichothecene Mycotoxicosis In Patient Urine," In: Journal of Environmental Biology 23(3), 301-320 (2002).

The Pathology of Trichothecene Mycotoxicosis In Humans

1. The Fingerprint of the Agent Causing the Disease is Displayed Within the Cells or Tissue of The Body.
2. Degeneration and Necrosis of The Entire Central Nervous System, Cardiovascular, lung, Digestive Tract, Spleen, Liver, Kidney, Pancreas, Immune, Skin, Reproductive, Eye, Urinary Bladder and Prostate.
3. The Signs and Symptoms Described For Trichothecene Mycotoxicosis Match the Pathology Observed.
4. Every Cell in The Body is Affected or Susceptible to Trichothecene Mycotoxins When Exposed.
5. The Exposed Cells Are Not Allowed to Grow and Make Cellular Products in The Rough Endoplasm Reticulum Represents of First Mechanism of Action on The Cells.
6. The Burning or Denaturation of Tissue From the Epoxide Molecule is Another Mechanism of Action on The Cells of The Body Causing Intense Scarring of Organs. (Like Phenol)
7. The Rapidly Turnover Organs Systems Are Affected The Most Severe, G.I. Tract, Immune System and Reproductive, (like radiation damage)
8. The Central Nervous System is Severely Affected and is A Primary Target Organ. The Neurons in the Cerebral Hemispheres, White and Grey Matter, Brain Stem and even the Ependymal Cells. The Purkinje Cells of The Cerebellum Are Severely Affected That Affect Motion and Balance. The Dorsal and Ventral Motor Neurons Are Destroyed Causing Amyotrophic Lateral Sclerosis. Peroxidation of Peripheral Nerves is Also Observed. The Central Nervous System is The Organ Most Affected as Reported By People Exposed to Toxic Mold.
9. Lack of Cellular Production, Epoxide- Peroxidation of Lipid Membranes, Loss of Vessels, Loss of Oxygen From Severe Lung Scarring, and Loss of Proper Nutrients Due Loss of Functional Absorption of Intestine Affect the Brain and All Organs of The Body.
10. The Trichothecene Mycotoxins are Cumulative in Their Health Effects on Organ Systems.
11. Trichothecene Mycotoxins are “Hit and Run” Poisons and are not Stored in The Body.
12. Inhalation of Trichothecene Mycotoxins Are More Poisonous As Observed by The Intense Scarring of The Alveolar Tissue Than Consumption Due To The Neutralization of Mycotoxin by Bacteria.
13. Depression of the Immune System Allows for Increase Infections by Bacteria, Viral, Fungal and Cancer to Form.
14. Yeasts are allowed to Colonize the Intestine Tract Because They Are Resistant to Trichothecene Mycotoxins.
15. Yeast Can Cause Diabetes Mellitus, Gout and Prevent Proper Liver Function to Detoxify Xenobiotics.
16. Trichothecene Mycotoxins are Released Within the Urine and Feces as Evidenced by The Pathology Observed Within Those Tissues.
17. Children Exposed to Trichothecene Mycotoxins are 100 to 1000 X more susceptible because stems are killed not allowing for additional growth within the individual.
18. There is No Safe Level of Exposure to Trichothecene Mycotoxins.
19. The third Mechanism For Trichothecene Mycotoxicosis is To Develop Anaphylaxis to Mold Allergens When Mycotoxin Leaves The Body.

Dr. William Croft, (Medical Pathologist)

-

Stages of Mycotoxicosis: For Inhalation of Mycotoxin

The three Stages (1-3) ranging from lower to higher severity of poisoning were modified according to exposure via the air as opposed to ingestion already established (Forgacs *et al.,* 1962; Joffe, 1971). A separate Stage of convalescence occurs when a patient is completely removed from the contaminated premises and the source of mycotoxin or mold spores.

Stage 1: The primary changes are in the brain, respiratory and immune systems, mucus membranes and gastrointestinal tract. Signs and symptoms may include burning sensation in the mouth, tongue, throat, palate, esophagus, and stomach, which is a result of the action of the toxin on the mucous membranes and skin in the exposed areas. Moist areas of the body armpits, under breasts, belt line and groin are more sensitive or first affected. Patients may report burning within the eyes, ears and nose. Patients also reported that their tongues felt swollen and stiff. Mucosa of the oral cavity may be hyperemic. Mild gingivitis, stomatitis, glositis, and esophagitis developed. Inflammation, in addition to gastric and (small and large) intestinal mucosal, resulted in vomiting, diarrhea and abdominal pain. Excessive salivation, headache, dizziness, weakness, fatigue and tachycardia were also present.

There may be fever and sweating. The respiratory system develops burning sensations and congestion. Severe exposure to mycotoxin within the lungs may lead to congestion, edema and failure, due to caustic action. Body temperature remains normal and controllable by the patient. The poisoning appears and disappears relatively quickly in this Stage with the exception of, lungs and central nervous system. Initially (Stage 1), the patient's symptoms are very uncomfortable or painful. As the poisoning continues and the patient progress toward Stage 2, he or she becomes accustomed to the presence of the mycotoxin and a quiescent period follows due to lack of nerve sensation. Depending on exposure levels, the first Stage may last from 3 - 9 days. In scoring the 50 signs and symptoms listed in Tables-1 and 2, an average score range of 20-45 represents Stage 1.

Stage 2 : This Stage is often called the latent Stage or incubation period because the patient feels apprehensive, but is capable of normal activity in the beginning of this Stage. Every organ of the body is affected by degeneration and necrosis with continued exposure. The primary target organs for an individual become evident over time, due to biological variation. These are disturbances in the central and autonomic nervous systems resulting in headaches, mental depression, loss of short-term memory, loss of problem-solving ability, various neuropsychiatric manifestations, meningism, severe malaise and fatigue, narcolepsy, loss of temperature control, hyperesthesia or numbness of body areas, and cerebellar dysfunction including hypotonia, attitude and gait, dysmetria, asthenia, vertigo, disturbances of speech, and loss of balance (Best, 1961). Spinal cord degeneration may also be observed in gait and reflex abnormalities, such as the ability to drive vehicles, ride bicycles or pass sobriety tests (inability to tolerate ethyl alcohol). Attention deficient disorder may be observed in children. Various systems may include: **Eyes**: visual disturbances, floating objects, light sensitive, lack of tears, burning and itching. **Ears**: burning, itching, and loss of hearing. **Immune and hematopoietic**: progressive loss of white and red cells including a decrease of platelets and hemoglobin, and high susceptibility to bacterial, mycotic and viral infections, debilitating chemical and allergies. **Gastrointestinal**: metallic taste in mouth, tooth loss, gum problems, stomatitis, sores in gums and throat, nausea, vomiting, diarrhea or constipation, excessive flatulence, abdominal distention, hepatitis, pancreatitis, and diabetes mellitus. **Respiratory :** burning and bleeding from nasal membranes, respiratory difficulty, asthma, extreme susceptibility to cold, flu and pneumonia. **Skin:** thinning of hair on head, burning on face, rashes, irritation, and edema. **Renal**: proteinuria, possible hematuria. **Reproductive:** irregular ovarian cycles, increased menstrual flow, fibroid growths in uterus, cystic development in mammary glands, and tumors of mammary and prostate glands. **Musculoskeletal** : somatitis, muscle weakness, spasms, cramps, joint pain, enlargement of joints in hand, and clubbing of fingers. **Cardiovascular:** chest pain, palpitations, ruptures of atrial walls, myocardial infection and aneurysm of arteries.

The skin and mucous membranes may be icteric, pupils dilated, the pulse soft and labile, and blood pressure may decrease or increase. The body temperature does not exceed 38 degree C and the patient may be afebrile, or chilled. Visible hemorrhagic spots may appear on the skin. Thoughts of suicide may be prominent in the person's mind at this time or anytime in Stage 2. Human bonding is very important for survival.

Degeneration and hemorrhages of the vessels marks the transition from the second to the third Stage of the disease and may not be consistently observed. The degeneration of the vital organs including serious respiratory insufficiency or asthma and CNS degeneration will take the patient into Stage three along with development of necrotic angina. If exposure continues, depending on exposure levels, Stage 2 may continue from weeks to months or even years until the symptoms of the third Stage develop. Evaluating the 50 signs and symptoms (Table-1 and 2) by assigning a score (0-least intense to 5-most intense or severe) to each symptom, we have determined that an average score range of 45-180 represents Stage 2.

Stage 3: Severe degeneration of the vital organs. The transition from the second to the third Stage is sudden. In this Stage, the patient's resistance is already low, and violent severe symptoms are present, especially under the influence of stress, or associated with physical exertion and fatigue. The first visible sign of this Stage may be lung, brain or heart failure (heart attack), with or without the appearance of petechial hemorrhage on the skin of

the trunk, the axillary and inguinal areas, the lateral surfaces of the arms and thighs, the face and head, and in serious Cases, the chest. The petechial hemorrhages vary from a few millimeters to a few centimeters in diameter. There is increased capillary fragility and any slight trauma may cause the hemorrhages to increase in size.

Aneurysms of the brain or aorta may be observed by angiography. Hemorrhages may also be found on the mucous membranes of the mouth and tongue, and on the soft palate and tonsils. There may be severe interstitial thickening or scarring of the lungs, or respiratory failure. Nasal, gastric and intestinal hemorrhages and hemorrhagic diathesis may occur. Necrotic angina begins in the form of catarrhal symptoms and necrotic changes soon appear in the mouth, throat, and esophagus with difficulty and pain on swallowing. Severe degeneration of the skin on the face, eyelids, and loss of lashes is also often present.

Necrotic lesions may extend to the uvula, gums, buccal mucosa, larynx, vocal cords, lungs, stomach, and intestines and other internal organs such as the liver and kidneys and are usually contaminated with a variety of avirulent bacteria. Bacteria infection causes an unpleasant odor from the mouth due to the enzymatic activity of bacteria on proteins. Areas of necrosis may also appear on the lips and on the skin of the fingers, nose, jaws, and eyes. Regional lymph nodes are frequently enlarged. Esophageal lesions may occur and involvement of the epiglottis may cause laryngeal edema and aphonia (loss of voice). Death may occur by strangulation.

Patients may suffer an acute parenchymatous hepatitis accompanied by jaundice. Bronchopneumonia, pulmonary hemorrhages, and lung abscesses are frequent complications. Tumors may develop of various organs, including skin, urinary bladder, brain, mammary gland, bone, immune, liver, prostate, possibly resulting in death. The most common cause of death is brain failure due to both direct effects of the mycotoxin on the central nervous system and indirect effects due to respiratory failure or lack of oxygen to the brain caused by the severe caustic inflammation (fibrinous exudation) reaction with the lung tissue, rendering it non-functional. Again, using the scoring system represented in Tables-1 and 2, an average score of greater or equal 180 represents Stage 3.

Stage of Convalescence: The course and duration of this Stage 3 depends on the intensity of the poisoning and complete removal of the patient from the premises or source of mycotoxin. Therefore, the duration of the recovery period is variable. There is considerable cellular necrosis and scarring to all major organs of the body in which cells will not regenerate, including the brain, spinal cord, eyes, lung, heart, liver, pancreas, kidney, adrenal, and blood vessels. If the disease is diagnosed during the first Stage, hospitalization is usually unnecessary, but allergies and asthma should be monitored closely. If the disease is diagnosed during the second Stage and even at the transition from the second to third Stages, early hospitalization may preserve the patient's life. If however, the disease is only detected during the third Stage, death cannot be prevented in most Cases.

1. Croft, W. A., Jastromski, B. M., Croft, A. L., and Peters, H. A., "Clinical Confirmation of Trichothecene Mycotoxicosis in Patients Urine", In: Journal of Environmental Biology **23**(3), 301-320 (2002)

2. .Forgacs, J., and W. T. Carll : Mycotoxicoses. *In* : Advances in Veterinary Science. Academic Press, New York and London, pp 273-372 (1962).

-

Abstract Information & Notes

Kaye H. Kilburn, M.D.

University of Southern California
Keck School of Medicine
2025 Zonal Ave., CSC-201
Los Angeles, CA 90033

Phone: 323/442-1830
Fax: 323/442-1833
E-mail: kilburn@usc.edu

Date of talk: Thursday, June 19, 2003, 2:30pm

Medical School Attended:	University of Utah College of Medicine
Major and date of Graduation:	1954
Board Certifications:	American Board of Internal Medicine, American Board of Preventive Medicine
Current Faculty Appointments:	Professor of Medicine University of Southern California Keck School
Current Job Description:	Ralph Edgington Professor - Academic Medicine Teaching, Research on Neurotoxicology, Pulmonary Disease
Other Information:	Author 240 peer reviewed papers; Book: Chemical Brain Injury, NY, John Wiley and Sons, 1998; President Neurotest Inc.; Develop test and use of Neurobehavioral methods in evaluated brain damage from chemicals; hydrogen sulfide, PCBs, pesticides, chlorine, ammonia, molds and mycotoxins
Disclosure Statement:	Neuro-test Inc.

SPEECH TITLE: **"How Molds and Mycotoxins Affect Human Brains"**

The speaker has provided the information below.

1.) Goals and objectives: Review the evidence from patients studied that show neurophysiological impairments: balance, vision, reaction time and on problem solving and memory.

2.) Outline of talk/abstract: Sixty-five adults were studied using 26 neurobehavioral tests, pulmonary function measurements and serum and saliva antibody titers

3.) Conclusion of what is to be learned: The human brain is the major target and premature aging is produced. Temporally parallel effects on pulmonary airways cause small airways obstruction and other organs may be involved.

4.) References: see abstract

How Molds and Mycotoxins Affect Human Brains

Kaye H. Kilburn, M.D.

University of Southern California, Keck School of Medicine

Background: Mold spores and mycotoxins produce airway irritation, asthma and bleeding. Neurobehavioral and respiratory symptoms suggested testing.

Methods: Neurobehavioral functions as means of percent predicted were compared in 65 consecutive mold exposed adults and 202 community controls. Measurements included balance, choice reaction time, color discrimination, blink reflex, visual fields, grip, hearing, problem solving, verbal recall, perceptual motor speed, and memory. Check lists surveyed histories, mood states and symptom frequencies (Kilburn 2002a and 2002b).

Findings: Exposed persons had abnormal balance, reaction time, blink reflex latency, color discrimination, visual fields, and grip. Also digit symbol substitution, peg-placement, trail making, verbal recall, and picture completion scores were reduced. Twenty-one of 26 tested functions were abnormal. Airways were obstructed and vital capacities reduced. Mood scores and symptom frequencies were elevated.

Interpretation: Mold exposures indoors were associated with neurobehavioral impairment probably from mycotoxins, such as trichothecenes. Correlation of human impairment with measured mycotoxins is the next step (Johanning et all 1999 and 2002, Nielsen and Thrane 2001).

REFERENCES

1. Kilburn KH. Janus Revisted, Molds Again. *Arch Environ Health* 2002a;57(1):7-8.
2. Kilburn KH. Inhalation of Moulds and Mycotoxins. *Eur J Oncol* 2002b;7(3):____.
3. Johanning E et al. Clinical Experience and Results of a Sentinel Health Investigation Related to Indoor Fungal Exposure. *Environ Health Perspect* 1999;107(3):489-494.
4. Johanning et al. Airborne Mycotoxin Sampling and Screening of Trichothecenes in Fungal Cultures - Using Gas Chromatography - Tandem Mass Spectrometry. *J Chromatography A* 2002;929(1):75-87.

Abstract Information & Notes

Kalpanna D. Patel, M.D.

65 Wehrle Dr.
Buffalo, NY 14225

Date of talk: Thursday, June 19, 2003, 3:30pm

Phone: 716/833-2213
Fax: 716/833-2244
E-mail: aehcwhy@wny.com

Medical School Attended: BJ Medical College, India
Residency: University of Texas Health Science Medical School at San Antonio
Board Certifications: American Board of Pediatrics, American Board of Environmental Medicine
Current Faculty Appointments: Associate Professor of Pediatrics, Suny, Buffalo
Current Job Description: President/Director AEHC-Buffalo
Other Information: Elected Appointments: President of American Board of Environmental Medicine, President of International Board of Environmental Medicine

Disclosure Statement: None

SPEECH TITLE: **"What Is New And Different in The Diagnosis And Management of Different Skin Disorders - Itching Eczema And Urticaria"**

The speaker has provided the information below.

1.) Goals and objectives:
1) To demonstrate importance of good environmental and dietary history of mold and mycotoxin exposure for the diagnosis and management of different skin disorders.

2) To demonstrate importance of Intradermal mold antigen testing and the efficacy of maximum tolerated end point, dose of mold antigen to obtain optimal response to relieve different skin conditions.

2.) Outline of talk/abstract:
5. $ Presentation of different cases having different symptomatology that failed to respond to traditional management.

6. $ To demonstrate effectiveness of Environmental Medicine approach in treatment of these cases.

7. $ Discussion of newer methods for the diagnosis and treatment of mold related skin disorders.

3.) Conclusion of what is to be learned:
1) Exposure to mold has become an enigma in homes as well as newer and older sick buildings.

2) Mold plays a major role in the immune dysfunction and development of inhalant sensitivity.

3) Chronic health problems like intense itching, eczema urticaria fatigue and many others can be effectively treated without drug therapy if the source of the problem is detected and treated effectively.

4.) References:

Abstract Information & Notes

Katherine Warsco, Ph.D.

East Carolina University
152 Rivers Building, ECU
Greenville, NC 27834

Phone: 252/328-6929
Fax: 252/328-4276
E-mail: warscok@mail.ecu.edu

Date of talk: Thursday, June19, 2003, 4:00pm

Medical School Attended: NA (Ph.D. College of Human Ecology, Michigan State University)

Major and date of Graduation: Family and Child Ecology, 1988

Residency: NA (Doctoral Internship B Energy Information Administration, US Department of Energy, Washington D.C.)

Board Certifications: NA

Current Faculty Appointments: Interior Design Program, School of Human Environmental Sciences, East Carolina University, Greenville, NC

Current Job Description: Chair, Department of Apparel Merchandising and Interior Design, School of Human Environmental Sciences, East Carolina University, Greenville, NC

Other Information: Founder of Environmental Quality in Interiors Network, Interior Design Educators Council

Disclosure Statement: None

SPEECH TITLE: **"Teaching Design for Good Indoor Air Quality"**

The speaker has provided the information below.

1.) Goals and objectives: The purpose of this speech is to report on the development of an environmental education curriculum that bridges fields of medicine, building science, and design to address indoor air pollution, environmental illness, and interior design. As a result of participation in this curriculum, students of interior design will be able to apply design criteria to address the following issues:

a) Application of non-toxic and radon-resistant construction practices;
b) Removal, isolation, and/or dilution of chemicals released into indoor air by appliances, cleaning solvents, pesticides, and the off gassing of synthetic materials, adhesives, and finishes;
c) Selection and layout of HVAC and lighting systems to avoid the buildup of moisture and the collection of dust;
d) Selection of furniture, cabinetry, and surface finishes that provide inhospitable conditions for microbial growth; and
e) Accessible design of living quarters to facilitate movement by a chemically sensitized client suffering from episodes of reduced stamina and mobility.

2.) Outline of talk/abstract: An environmental education curriculum was developed to assist students in exploring linkages between micro-climate, architectural shell, interior space plan, building systems, materials specification, indoor air quality, and environmental illness. In the context of a national design competition, students applied a series of programming exercises to develop solutions for a hypothetical residential design problem. Exercise 1 required students to identify effects of chemical and biological contaminants on building occupants based on patient profiles published in Volume III of William Rea's series on chemical sensitivity. Exercise 2 required students to identify sources, paths of entry and design solutions for chemical and biological irritants. Exercise 3 required students to identify the effects of Multiple Chemical Sensitivity on physical dexterity as applied to an anthropometric model developed by Ralph Faste. Exercise 4 required students to outline design solutions for controlling indoor air quality. Students applied a series of schematic diagramming exercises to a hypothetical building and residential site located in a hot, humid climate to explore the following:

a) Orienting the building and fenestration for day lighting and ultraviolet light, passive solar and winter heat movement, and passive cooling and prevailing winds;
b) Radon resistant construction practices;
c) Interior spatial arrangement to facilitate movement of building occupants with low stamina and exhaustion of pollutants in a depressurized building;
d) Interior spatial arrangement to isolate sleeping quarters to provide a pollutant-free sanctuary; and
e) Specification of building materials, and interior finishes, fabrics, and equipment to remove contaminants from the interior-breathing zone.

3.) Conclusion of what is to be learned: This environmental education project represents a first attempt to partner with the Environmental Protection Agency to employ student design competitions as a vehicle for disseminating scientific environmental information to post-secondary schools of interior design in the U.S. Fulfilling sponsor expectations for developing a curriculum inclusive of the major threats to human health by indoor air was complicated by conflicting assumptions within the scientific community as to what constitutes environmental disease. Although this curricular tool was successful in providing a thorough overview of indoor air quality design considerations, deliberately retaining the complexity of issues within a multi disciplinary framework challenged the definition of acceptable boundaries for a problem addressed by interior designers. Future efforts to integrate environmental health issues in interior design curricula must balance the need to avoid unidimensional problem solving with limitations to the scope of training for interior design students. Increasing collaboration among schools of design and health sciences to provide opportunities for multi disciplinary problem solving would ensure design solutions are grounded in current knowledge of the effects to health of biological and chemical indoor contaminants.

4.) References: See attached

Teaching Design for Good Indoor Air Quality

Katherine Warsco
East Carolina University

Goals and objectives

The purpose of this speech is to report on the development of an environmental education curriculum that bridges fields of medicine, building science, and design to address indoor air pollution, environmental illness, and interior design. As a result of participation in this curriculum, students of interior design will be able to apply design criteria to address the following issues:

a. Application of non-toxic and radon-resistant construction practices;
b. Removal, isolation, and/or dilution of chemicals released into indoor air by appliances, cleaning solvents, pesticides, and the off gassing of synthetic materials, adhesives, and finishes;
c. Selection and layout of HVAC and lighting systems to avoid the buildup of moisture and the collection of dust;
d. Selection of furniture, cabinetry, and surface finishes that provide inhospitable conditions for microbial growth; and
e. Accessible design of living quarters to facilitate movement by a chemically sensitized client suffering from episodes of reduced stamina and mobility.

Outline

A curriculum was proposed for improving the quality of interior design instruction for addressing indoor air pollution and environmental illness through residential interior design. The purpose of the project was:

a. To develop, disseminate, and implement materials and methods for assisting post-secondary instructors and students of design to increase their knowledge of indoor air quality issues;
b. To promote the application of this knowledge to develop innovative solutions for radon-resistant, non-toxic, and allergy-free interiors; and
c. To provide opportunities for interdisciplinary problem solving, drawing from the fields of medicine, architecture and engineering to address residential design solutions for medically at-risk populations such as the elderly and the infirm.

This project targeted 460 faculty and their students in two- and four-year university interior design programs in the US. The coastal southeast region was the focus of this environmental illness project because the following factors make indoor air quality concerns critical components of the building design process:

a. The population exceeds the national average for age 65 years and older and percent of elderly both in poverty and in poor health;
b. For coastal states such as Florida, housing stock exists with radon levels exceeding EPA limits; and
c. Hot and humid climate conditions increase microbial growth and the release of chemicals that incite toxic and allergic reactions among the hypersensitive.

The vehicle used to improve the quality of interior design instruction was a national student design competition and teacher's supplement. A competition was developed incorporating requirements for the design of a residential setting for a retired couple who suffered from environmental illness. Included in the competition were the following components linked to principles of environmental design:

a. Site profile. Students were asked to design a retirement residence in Northern Florida in a county where radon gas is prevalent in levels that exceed the EPA limit. The hot, humid micro-climate posed medical problems concerning microbial growth. Lots were selected from a housing development that adheres to tenets of sustainable design in order to integrate principles of enviroscaping (i.e., environmentally safe & energy conscious landscaping practices) and sustainable construction with the residential design problem. Sustainable construction building codes released by the John D. and Catherine T MacArthur Foundation and information from a market survey conducted by the University of Florida Center for Construction and the Environment were made available for use in this competition.
b. Building profile. Students were asked to modify a design given of a Florida Cracker House, a vernacular archetype suitable for hot, humid climates predating mechanized air conditioning and development of synthetic building materials. Focusing on a process of archetypal ideation developed by Ronald Haase,

students explored traditional principles of passive cooling to address good indoor air quality.

c. Client profile. Students were given information concerning the needs and characteristics of a hypothetical client family. The medical case histories were based on actual patient case reports from the Environmental Health Center-Dallas, Texas. Students were introduced to medical profiles as a source of information regarding client responses to toxin and allergen exposures within building interiors.
d. Drawings. Site plans, building elevations, foundation plan, roof plan and floorplan were included in hard copy and made available in electronic format. Information was provided to assure compliance with the Southern Building Code regarding foundation design and break-away construction to address the velocity of hurricane-force winds and coastal flooding of the foundation.
e. Resources. Order forms were included for literature and electronic media pertaining to designing for indoor air quality, hot humid climates, and environmental illness.

A supplement to the competition provided a suggested teaching plan for integrating the competition in university instruction. The teaching plan included learning objectives, suggested readings, discussion questions, and examples of student outcomes. In the context of the national design competition, students applied a series of programming exercises to develop solutions for a hypothetical residential problem:

a. Patient Profile. In Table 1, students were required to identify effects of chemical and biological contaminants on building occupants based on patient profiles published in Volume III of William Rea's Series on chemical sensitivity (Rea, W., 1996). In Table 2, students were required to identify sources, paths of entry and design solutions for chemical and biological irritants based on information provided in the suggested textbook, *Your Home, Your Health and Well-Being* (Rousseau, Rea, and Enwright, 1990). The purpose of these exercises was to gain understanding of the linkages between pollutant sources in the built environment and medical symptom behaviors of building occupants. Students explored translating medical data into design parameters for use in the process of design ideation for a hypothetical client family.
b. Disability Compensation. In Table 3, students were required to identify the effects of chemical sensitivity on physical dexterity as applied to "The Enabler," an anthropometric model developed by Ralph Faste (Rashko, B., 1991). The purpose of this exercise was to identify design requirements associated with selective physical disabilities for use in the process of design ideation for a hypothetical client family.
c. IAQ Solutions. In Table 4, students were required to outline design solutions for controlling indoor air quality in the "sanctuary;" the primary sleeping quarters for their hypothetical client couple. In Table 5, students provide a material assessment for client health and well-being. Building materials, finishes and furnishing materials, and building systems (i.e., plumbing, electrical, heating and appliances) were chosen according to a rating-for-safety system developed by Rousseau and Rea (Rousseau, et. al., 1990). Students intertwined issues of materials science with aesthetic considerations in their development of a residential prototype for benign design.

Students applied a series of schematic diagramming exercises to a hypothetical building and residential site located in a hot, humid climate as a means to explore principles from the following areas of design:

a. Micro-Climate Design. Students explored orientation of openings in the architectural shell for daylighting, passive solar and winter heat movement, and passive cooling and prevailing winds to address building occupant thermal comfort, reduce consumption of fossil fuels for heating and cooling, and dilute airborne contaminants in the near environment.
b. Enviroscaping and Sustainable Development. In the manner of case studies of the Kanapaha Botanical Gardens and the Florida House Learning Center, students were required to develop conceptual plot plans. Specification of drought tolerant plants indigenous to the climate minimized irrigation as a habitat for mold and use of chemical fertilizers that were a source of toxins for their chemically sensitive clients.
c. Radon-Resistant Construction Practices. Students explored radon mitigation techniques for homes with above-grade foundations in terrain prone to coastal flooding.
d. Accessible Design. Students considered human activity patterns influenced by loss of stamina, general muscle weakness, and chronic fatigue characteristic of medical conditions of the age and chemical sensitivity of their clients. Structural and nonstructural design solutions were explored to support daily routines of the non-ambulatory and infirm.
e. Non-Toxic, Allergy-Free Design of the "Sanctuary." Students explored the design of sleeping quarters free of environmental irritants that might interfere with healing and revitalization. Strategies for removing and isolating indoor contaminants were explored with regards to design of mechanical systems, space planning, and specification of furnishings, finishes, equipment, and accessories for residential interiors.

Evaluation of Competition Submissions

Of the 460 competition packets mailed to US members of the Interior Design Educators Council, thirteen universities participated with thirty-nine submissions. Although every region except the Northwest was represented in the competition, three-fourths of the submissions were from universities in hot, humid climates. A three-member jury independently ranked student entries. Criteria used to evaluate submissions included the following:

a. The design solution is appropriate for sustainable development considerations of the climate, terrain, and vernacular archetype (15%)
b. The design solution reflects an accurate and sensitive application of principles of radon-resistant, non-toxic, allergy-free, and accessible design (40%)
c. The solution represents innovative design for the "sanctuary," addressing specific medical conditions of the clients as explained in the patient profiles of the design program (10%)

These criteria constituted 65% of the overall evaluation score. Winning submissions were selected from top scores, ranging from 81-90/100 points. Jurors provided numerical and open-ended responses to student submissions. Faculty and students participating in the competition received written feedback as to the strengths and weaknesses of each submission according to disciplinary perspectives represented by the jury.

Evaluation of Student Contributions to Teacher's Supplement

Student outcomes presented as illustration for exercises in the teacher's supplement received national attention. Conceptual designs prepared by an undergraduate research assistant, Michelle Puckett Jenkins were awarded first place in two national student competitions: a) a call for student design entries sponsored by Affordable Comfort Inc., an interdisciplinary organization that promotes environmentally friendly, energy-efficient, healthy building practices; and b) a national lighting competition sponsored by Cooper/Halo Metalux, Inc. These projects received external validation from juries comprised of educators and practitioners in the fields of building construction, architecture, and mechanical engineering.

Conclusions

This environmental education project demonstrated viable linkages between the arts and sciences in addressing indoor air quality and environmental illness. Employing a student design competition as a vehicle for disseminating scientific environmental information provided a thorough overview of indoor air quality design considerations. Increasing collaboration among schools of design and health sciences to provide opportunities for multidisciplinary problem solving would ensure design solutions would be grounded in current knowledge of the effects to health of biological and chemical indoor contaminants.

References

American Lung Association. (1992). Indoor Air Pollution Fact Sheet: Radon. Atlanta, GA: American Lung Association.

American Lung Association, Environmental Protection Agency, Consumer Products Safety Commission, & American Medical Association. (1994). *Indoor Air Pollution: An Introduction for Health Professionals* (Government document 1994-523-217/81322). Washington, D.C.: American Lung Association

Environmental Protection Agency, Consumer Product Safety Commission, & American Medical Association. (1998). Trends in Cigarette Smoking. *Morbidity & Mortality Weekly Report, 47*(43).

Axelrad, B. (1993). Improving IAQ: EPA's program. *EPA Journal, 4*(October-December), 14-17.

Beecher, M. A., & Davies, B. (2002, March 19-24). *Shades of Green: The Philosophical Challenges of Ecological Responsibility in Interior Design Education and Practice.* Paper presented at the Mesas and the Mysteries: On the Edge of Imagination/Green Design, Santa Fe, NM.

Bode, M., & Munson, D. (1995). *Controlling Mold Growth in the Home*, [Guidance Document]. Kansas State University Agricultural Experiment Station and Cooperative Extension Service. Available: http://www.oznet.ksu.edu/library/hous2/mf2141.pdf [2002, November 11].

Browner, C.M. (1993). Environmental Tobacco Smoke: EPA's report. *EPA Journal, 4*(October-December), 18-22.

Canada Mortgage and Housing Corporation. (1993). *The Clean Air Guide: How to Identify and Correct Indoor Air Problems in Your Home* (NHA 6695; NH15-8311993E). Eobicode, ON: Canada Mortgage and Housing Corporation.

Coleman, C. (1999). Life-cycle design: Leaving future generations a legacy. *Perspective* (Spring/Summer), 27-28, 30.

Danko, S., Eshelman, P., & Hedge, A. (1990). A taxonomy of health, safety, and welfare implications of interior design decisions. *Journal of Interior Design Education and Research, 16*(2), 19-30.

Fernández-Caldas, E., Trudeau, W. L., & Ledford, D. K. (1994). Environmental control of indoor biologic agents. *Journal of Allergy & Clinical Immunology, 94*(2), 404-412.

Guerin, D. A. (1992). Issues facing interior design education in the twenty-first century. *Journal of Interior Design Education and Research, 17*(2), 9-16.

Haase, R. W. (1992). *Classic Cracker: Florida's Wood-Frame Vernacular Architecture*. Sarasota: Pineapple Press.

Hasell, M. J., & Scott, S. C. (1996). Interior Design Visionaries' Explorations of Emerging Trends. *Journal of Interior Design, 22*(2), 1-14.

Human Ecology Action League. (1992). Chemicals Can Effect Your Health . Atlanta, GA: Human Ecology Action League.

Inman, M., & Shea, J. (1993, October, 1993). *Housing problems for older adult households in the southeast.* Paper presented at the Paper presented at the 1993 annual meeting of the American Association of Housing Educators, Columbus, OH.

Kibert, C. J. E. (1995). *Sustainability Rationale: Analysis of Sustainable Construction Aspects of the Abacoa Development* (Unpublished Manuscript). Gainesville: University of Florida Center for Construction and Environment.

Kibert, C. J. E. (1996). *Sustainable Construction Code--Residential: For the Abacoa Development* (Unpublished Manuscript). Gainesville: University of Florida Center for Construction and Environment.

Kloeppel, J. E. (1993, April, 1993). Beware the fungus among us: Emissions from mold & fungus may be culprits in indoor air problems. *Georgia Tech News,* 1-4.

Lechner, N. (1991). *Heating, Cooling, Lighting: Design Methods for Architects*. New York: John Wiley & Sons.

Marcinowski, F., Lucas, R. M., & Yeager, W. M. (1994). National and regional distributions of airborne radon concentrations in U.S. homes. *Health Physics, 66*(6), 699-706.

Mendler, S. (2002). LEED: A Roadmap for Added Value. *Perspective: Journal of the International Interior Design Association*(Winter 2002), 42-49.

Miller, B. R., Miller, P. B., & Bateman, M. S. (2002, March 19-24). *Two's Company, Three's a Good Team: Multidisciplinary Teams are a 21st Century Necessity.* Paper presented at the Mesas and the Mysteries: On the Edge of Imagination/Green Design, Santa Fe, NM.

Miller, J. W. (2002). Green Home Building. *Perspective: Journal of the International Interior Design Association*(Fall 2003), 22-27.

Moussatche, H., King, J., & Rogers, T. S. (2002, March 19-24, 2002). *Material Selection in Interior Design Practice.* Paper presented at the Mesas and the Mysteries: On the Edge of Imagination/Green Design, Santa Fe, NM.

Odom, J. D.,& DuBose, G. (1996). *Preventing Indoor Air Quality Problems in Hot, Humid Climates: Design and Construction Guidelines* (Unpublished manuscript). Orlando: CH2M Hill, Inc., Disney Development Co.,.

Peart, V. (1993). *Indoor air quality in Florida: Formaldehyde* (Fact Sheet He 3205). Gainesville: University of Florida Cooperative Extension Service.

Raschko, B. (1991). *Housing Interiors for the Disabled & Elderly*. New York: Van Nostrand Reinhold.

Rea, W. J. (1996). *Chemical Sensitivity* (Vol. 3). Boca Raton: CRC Press.

Rousseau, D., Rea, W. J., & Enwright, J. (1990). *Your Home, Your Health, & Well-Being*. Berkeley: Ten Speed Press.

Science Advisory Board. (1990). *Reducing risk: Setting priorities and strategies for environmental protection* (Government Report SAB-EC-90-021). Washington, D.C.: The Science Advisory Board.

Seltzer, J. M. (1995). *Creating healthy indoor environments: A road map for the future* (Vol. 10). Philadelphia: Hanley & Belfus.

Sexton, K. (1993). An inside look at air pollution. *EPA Journal, 19*(4), 9-12.

US Environmental Protection Agency. (1993). *Targeting Indoor Air Pollution: EPA's Approach and Progress* (Bulletin EPA 400-R-92-012). Washington D.C.: U.S. Environmental Protection Agency, Air and Radiation.

US Environmental Protection Agency. (2001a, May 28, 2002). *Healthy Buildings, Healthy People: A Vision for the 21st Century*, [Guidance Document]. US Environmental Protection Agency. Available:

http://www.epa.gov/iaq/hbhp/index.html [2002, November 11].

US Environmental Protection Agency. (2001b, December 31, 2001). *Mold Remediation in Schools and Commercial Buildings*, [Guidance Document]. US Environmental Protection Agency. Available: http://www.epa.gov.iaq/pubs [2002, November 11].

Warsco, K. (1997a). Designing for Good Indoor Air Quality in a Hot, Humid Climate: Student Design Competition . Unpublished competition materials prepared for US EPA Environmental Education Grants Program, Washington, D.C.

Warsco, K. (1997b). Teaching Supplement to the Student Design Competition: Designing for Good Indoor Air Quality in a Hot, Humid Climate: Unpublished supplement to competition materials prepared for US EPA Environmental Education Grants Program, Washington, DC.

Waxman, H. A. (1993). The view from congress. *EPA Journal, 19*(4), 38-39.

Wilson, K. P. (2002). The Case for Green Design. *Perspective: Journal of the International Interior Design Association*(Winter 2002), 21-26.

Zummos, S. M., & Karol, M. H. (1996). Indoor air pollution: Acute adverse health effects and host susceptibility. *Environmental Health, January-February*, 25-29.

Abstract Information & Notes

Michael R. Gray, M.D., M.P.H.

300 S. Ocotillo
Benson, AZ 85602

Phone: 520/586-9111
Fax: 520/586-9091
E-mail: DocMike007@aol.com

Date of talk: Thursday, June 19, 2003, 4:30pm

Medical School Attended: University of Cincinnati, College of Medicine; University of Illinois School of Public Health

Major and date of Graduation: M.D., 1974; M.P.H., 1978

Residency: 1975-77 Internal Medicine, Cook County Hospital, Chicago, IL; 1977-78 Chief Resident, Internal Medicine, Cook County Hospital, Chicago, IL

Current Job Description: Medical Director, Progressive Healthcare Group, Benson, AZ, 1988-present; Medical Director and Board Chairman, Benson Ambulance Service, Benson, AZ, 1990-present

Other Information: Published several articles

Disclosure Statement: Cholestyramine

SPEECH TITLE: **"Mold, Mycotoxins & Public Health: a Clinicians Perspective"**

The speaker has provided the information below.

1.) Goals and objectives: To help clinical practitioners recognize and treat systemic illnesses associated with mixed molds and mycotoxins

2.) Outline of talk/abstract: A descriptive epidemiologic survey of the results of clinical evaluations of 250 patients exposed to mixed, toxigenic, structural molds and mycotoxins will be presented

3.) Conclusion of what is to be learned: How to evaluate and manage mycotoxicosis

4.) References: Bibliography provided with handouts.

Mold, Mycotoxins, and Public Health

Michael R. Gray, M.D., Robert C. Crago, Ph.D., Kaye Kilburn, M.D.

Clinical evaluations of 250 patients compiled from 1994-2003, are presented in a clinically based descriptive epidemiologic study with results compared to unexposed controls, general reference ranges, and national databases for multiple parameters measured. Abnormalities of immune function, pulmonary function, and neurocognitive function and impairment, are presented confirming that exposure to mixed toxigenic structural filamentous, terrestrial molds (e.g., *Stachybotrys sp., asperigillius/Penicillium sps., Fusarium, etc)*, and their associated mixed mycotoxins (eg., *aflatoxin, rubrotoxin, sterigmatocystin, vomitoxin, tremulotoxin, zearalanone, trycothecenes, T-2 toxin, etc.)* collectively induce mycotoxicosis, a clinical syndrome resulting from infectious and toxic neuroimmunopathophysiologic effects. Key features include: immune toxicity with hyper activation and simultaneous suppression, small airways obstruction and reactivity, and neurological toxicity involving multiple neurophysiologic processes (e.g., *balance, visual perception, peripheral vision, simple and choice reaction time, blink reflex, etc.).*

21st ANNUAL INTERNATIONAL SYMPOSIUM
ON MAN & HIS ENVIRONMENT

SCHEDULE
Friday, June 20, 2003

7:00 a.m.	**Breakfast with Drucker Labs, Michelangelo Room**
8:00 a.m.	**ANNOUNCEMENTS/MODERATOR: Sherry A. Rogers, M.D.**
8:05	**Eugene A. Shinn,** U.S. Research Geologist, Geological Survey, St. Petersburg, FL: "**Update: Transoceanic Soil Dust Transport and Medical Implications"**
8:25	Q & A
8:35	**Lester Friedlander, B.A., D.V.M.,** Wyalusing, PA: "**Molds and Mycotoxins in the Food Chain"**
8:55	Q& A
9:05	**Katherine Warsco, Ph.D.,** Department of Interior Design, East Carolina University, Greenville, NC: **"Interior Design for a Mold-Free Environment"**
9:25	Q & A
9:35	**Aristo Vojdani, Ph.D., M.T.,** Director, Immunosciences Laboratories, Inc., Beverly Hills, CA: **"Immunotoxicology of Molds and Mycotoxins"**
9:55	Q & A
10:05	**BREAK WITH EXHIBITORS**
10:30	**Bruce Small,** Director of Envirodesic Certification Program, Georgetown, Ontario, Canada: **"Prescription for Preventing Mold and for Mold Remediation"**
10:50	Q & A
11:00	**Martha Stark, M.D.,** Department of Psychiatry, Harvard Medical School, Boston, MA: **"Mysterious Mental Illnesses, Pernicious Poisons"**
11:20	Q & A
11:30	**John H. Boyles, Jr., M.D.,** Dayton Ear, Nose & Throat Surgeons Inc., Centerville, OH: **"Diagnosis & Treatment of Inhalant and Mold Allergy"**
11:50	Q & A
12:00n	**OPEN LUNCH**
	MODERATOR: Kaye H. Kilburn, M.D.
1:00 p.m.	**David C. Straus, Ph.D.,** Department of Microbiology, Texas Tech University Health Science Center, Lubbock, TX: **"The Role of Fungi in Sick Building Syndrome"**
1:20	Q & A
1:30	**Tapani Tuomi,** Laboratory Chief, Finnish Institute of Occupational Health, Helsinki, Finland: "**Mycotoxins in Cigarettes and in Tobacco Smoke"**
1:50	Q & A
2:00	**Mohamed B. Abou-Donia, Ph.D.,** Duke University Medical Center, Durham, NC: **"Acute Exposure to Sarin Increases Blood Brain Barrier Permeability & Induces Neuropathological Changes in the Rat Brain: Dose Response Relationship"**
2:20	Q & A
2:30	**Sherry A. Rogers, M.D.,** Medical Director, Northeast Center for Environmental Medicine, Author, Syracuse, N.Y.: "**Rescuing the Heart as Toxic Target Organ"**
2:50	Q & A
3:00	**BREAK WITH EXHIBITORS**
3:30	**Bruce Jarvis, Ph.D.**, Department of Chemistry & Biochemistry, University of Maryland, College Park, MD: **"History and Toxicology of Mycotoxicoses"**
3:50	Q & A
4:00	**David C. Straus, Ph.D.,** Department of Microbiology, Texas Tech University Health Science Center, Lubbock, TX: **"Recent Research in Sick Building Syndrome"**
4:20	Q & A
4:30	**William J. Rea, M.D.,** Director, Environmental Health Center B Dallas, Dallas, TX: **"Treatment of Mold & Mycotoxin Exposure"**
4:50	Q & A
5:00	**Panel Discussion: "How and when to remodel a moldy building"** Donald P. Dennis, M.D., Larry Foster, Professor Tang G. Lee, AAA, David C. Straus, Ph.D., Bruce Small, and Tapani Tuomi
6:00	**RECEPTION WITH THE EXHIBITORS**

FRIDAY, JUNE 20, 2003

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

Eugene A. Shinn

U.S. Geological Survey
600 4th Street South
St. Petersburg, FL 33701

Phone: 727/803-8747 ext.3030
Fax: 727/803-2032
E-mail: eshinn@usgs.gov

Date of talk: Friday, June 20, 2003, 8:05am

Medical School Attended: University of Miami, Florida - honorary Ph.D., University of South Florida, 1998

Current Faculty Appointments: Adjunct professor University of South Florida and University of Miami

Current Job Description: Research Geologist

Other Information: Through geology/earth surface processes, I developed a theory that pathogens transported in African dust to the Caribbean caused the ongoing demise of coral reefs - this led to human health effects, especially asthma in the Caribbean.

Disclosure Statement:

SPEECH TITLE: **"Update: Transoceanic Soil Dust Transport and Medical Implications"**

The speaker has provided the information below.

1.) Goals and objectives:

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:

4.) References:

Update: Transoceanic Soil Dust Transport and Medical Implications

E.A. Shinn, Christina A. Kellogg, Dale W. Griffin, Carles W. Holmes, Virginia H. Garrison, Douglas B. Seba

Increasing transoceanic dust flux may affect public health, especially among chemically sensitive and medically compromised individuals. Indigenous dust in the western U.S. is known to transport the valley fever pathogen *Coccidioides immitis*, but the effects of African dust, which transports bacteria, viruses, and spores of fungi, including numerous species of *Aspergillus* have not been investigated until recently.

Estimates of annual African dust flux to the Amazon basin, Caribbean, and southeast U.S. range into the hundreds of millions of tons. Flux of African dust to the Caribbean and U.S. has increased dramatically since 1970 because of the ongoing drought in North Africa. The drought is a result of fluctuations in the North Atlantic Oscillation (NAO), and long-term dust monitoring in Barbados and Miami shows a direct correlation with the NAO. The incidence of asthma on Barbados and Trinidad, documented by the Caribbean Allergy and Respiratory Association (CARA), is among the highest in the world and has increased 17- fold since 1973. Recent studies in Trinidad indicate a correlation between dust events and pediatric admissions for respiratory distress.

Of the over 250 microbial isolates identified from African dust, roughly 30% were pathogenic; capable of infecting plants, animals, or humans with compromised immune systems. In addition to viable bacteria, fungi and viruses dust contains organic debris, insects, and various toxic metals including naturally occurring radioactive isotopes Be-7 and Pb-210. Recent studies indicate that iron in African dust triggers red tides that in turn aerosolize toxins that have pronounced effects on humans living near marine shorelines.

Abstract Information & Notes

Lester C. Friedlander, B.A., D.V.M.

P.O. Box 534
Wyalusing, PA 18853

Phone: 570/746-3072
Fax: 570/746-1386
E-mail: iamlfbadvm@aol.com

Date of talk: Friday, June 20, 2003, 8:35am

Undergraduate School Attended: Northland College, Ashland, WI
Araneta University Foundation, College of Veterinary Medicine, Metro Manila, Philippines

Major and date of Graduation: BA, 1971: DVM,1979
Board Certifications: None
Current Faculty Appointments: None
Current Job Description: Independent Researcher, Consultant
Other Information: Chronic Wasting Disease Workshop – Veterinary Professional C.E. UNIV. of Minnesota, College of Veterinary Medicine. Co-Authored: Accumulation 0f 2.8 Dihydroxyadenine in Bovine Liver, Kidneys, and Lymph Nodes. Journal of Veterinary Pathology 28:99-109(1991)

Disclosure Statement: None

SPEECH TITLE: **"Molds and Mycotoxins in the Food Chain"**

The speaker has provided the information below.

1.) Goals and objectives: To inform and educate health professionals that there are molds and mycotoxins in the foods we eat. Furthermore to explain and list the symptoms of mycotoxins in both humans and animals

2.) Outline of talk/abstract: Please refer to page 2

3.) Conclusion of what is to be learned: There are many mycotoxins in our environments with similar symptoms in humans

4.) References:

1. **Black, Kevin.** "Aflatoxins in Corn" (Dec. 1996): 2 pag. Online. Internet. 20, Feb. 1997

2. **Cheeke, Peter R.**, **Lee R. Shull**. ed. Natural Toxicants in Feeds and Poisonous Plants. Westport: AVI Publishing 1985.

3. **Hascheck**, **Wanda M.** "Selected Mycotoxins Affecting Animal and Human Health": Academic Press 2002

4. **GIPSA, Technical Services Division: "** Grain Fungal Diseases & Mycotoxin Reference" USDA 2003

5. http://vm.cfsan.fda.gov/~frf/iupac.html
http://www.btny.purdue.edu/NC129/
http://pasture.ecn.purdue.edu/~grainlab/
http://www.ces.ncsu.edu/drought/dro-29.html
http://www.aces.edu/department/grain/ANR767.htm
http://www.ianr.unl.edu/pubs/pesticides/g790.htm
http://www.ipm.iastate.edu/ipm/icm/1998/1-19-1998/btdiscon.html
http://www.scisoc.org/feature/Btcorn/Top.html

Molds and Mycotoxins in the Food Chain

Lester Friedlander, B.A., D.V.M.

1.) Goals and objectives:

To inform and educate health professionals that there are molds and mycotoxins in the foods we eat. Furthermore, to explain and list the symptoms of mycotoxins in both humans and animals.

2.) Outline of talk/abstract:

I. Introduction
   - A. Condensed History of Mycotoxicology (Early 1960's to Present)

II. Classification of Mycotoxins
   - A. Aflatoxins
   - B. Trichothecenes
   - C. Fumonisins
   - D. Zearalenone
   - E. Ochratoxin A
   - F. Ergot Alkaloids

III. How We Consume Mycotoxins
   - A. How mycotoxins get into your Food
     1. Growth
     2. Storage
     3. Transportation
   - B. Mycotoxins Carried by Animals
     1. General Carriers
     2. Species Specific
     3. Rarities

IV. Conclusion
   - A. Current Mycotoxin Standards
   - B. High risk areas

Abstract Information & Notes

Katherine Warsco, Ph.D.

East Carolina University
152 Rivers Building, ECU
Greenville, NC 27834

Phone: 252/328-6929
Fax: 252/328-4276
E-mail: warscok@mail.ecu.edu

Date of talk: Friday, June 20, 2003, 9:05am

Medical School Attended: NA (Ph.D. College of Human Ecology, Michigan State University)

Major and date of Graduation: Family and Child Ecology, 1988

Residency: NA (Doctoral Internship – Energy Information Administration, US Department of Energy, Washington D.C.)

Current Faculty Appointments: Interior Design Program, School of Human Environmental Sciences, East Carolina University, Greenville, NC

Current Job Description: Chair, Department of Apparel Merchandising and Interior Design, School of Human Environmental Sciences, East Carolina University, Greenville, NC

Other Information: Founder of Environmental Quality in Interiors Network, Interior Design Educators Council

Disclosure Statement: None

SPEECH TITLE: **"Interior Design For A Mold-Free Environment"**

The speaker has provided the information below.

1.) Goals and objectives: This speech will provide an overview of current thinking within the professional community of interior design with regards to methods of proactive environmental design. Objectives include identifying current >best practices= advice of a general and conceptual nature for the types of mold problems experienced in many homes, schools and office facilities. Current thinking is based on a comparison of philosophical positions of the interior design community evident among practitioners and educators of interior design and agricultural experiment station cooperative extension specialists.

2.) Outline of talk/abstract: Presented here are proactive approaches to achieving mold-free building interiors along with strategies for mold remediation in building interiors resulting from water leakage, condensation, or flooding. Interior design decisions impact prevalence of moisture and nutrient matter through space planning and specification of interior finishes, fabrics, furnishings, and equipment. Also, covered are current efforts in the development of certification programs to rate building materials and interior products. This speech concludes with trade-offs between >best practices= advice and other considerations regarding up-front building costs, building maintenance and operation, and building occupant comfort, health and well-being.

3.) Conclusion of what is to be learned: Interior design practice and education contribute to the prevention and remediation of mold growth in indoor environments. The literature points to a need for interior designers to be educated from a perspective of eco-materialism that links indoor material choices to building occupant health.

4.) References: See attached

Interior Design For A Mold-Free Environment

Katherine Warsco, Ph.D.
East Carolina University

Goals and objectives

This speech will provide an overview of current thinking within the professional community of interior design with regards to methods of proactive environmental design. Objectives include identifying current 'best practices' advice of a general and conceptual nature for the types of mold problems experienced in many homes, schools and office facilities. Current thinking is based on a comparison of philosophical positions of the interior design community evident among practitioners and educators of interior design and agricultural experiment station cooperative extension specialists.

Presented here are proactive approaches to achieving mold-free building interiors along with strategies for mold remediation for moisture intrusion due to water leakage, condensation, and flooding. The US Environmental Protection Agency and East Carolina University supported the development of an environmental education curriculum. A design competition and teacher's supplement were developed to assist educators and students of interior design to explore linkages between microclimate, architectural shell, interior space plan, mechanical systems, materials specification, indoor air quality, and environmental illness. Excerpts from student submissions to this competition, shown in this presentation serve to illustrate interior design for a mold-free environment.

Proactive Approach

Interior design decisions impact prevalence of moisture and nutrient matter that support microbial growth through space planning of the building interior and specification of interior finishes, furnishings, fabrics, and equipment. Strategies for removing, isolating, and diluting moisture and nutrient matter in the building interior follow two schools of thought; they typically either focus on the natural environment (i.e., healthy planet), or they focus on the interaction of the building and the building occupant (i.e., healthy people). Architectural and engineering ventilation strategies illustrate these approaches. A 'healthy planet' design approach might use a negative pressurized architectural shell that maximizes airflow through interior breathing zones. Benefits to the planet include reduced fossil fuel consumption for heating, ventilating and air conditioning. A 'healthy people' design approach might use a positive pressurized architectural shell that balances and filters airflow within interior breathing zones. Although ventilation strategies are largely within the domains of architecture and engineering, Interior design decisions intertwine with those of allied building fields to impact the ultimate success of these approaches to address mold-free design.

Ventilation (dilution) strategies are addressed through space planning and design of mechanical systems.

a. Arranging fenestration and interior partitions to facilitate natural ventilation will dry moisture-laden areas and exhaust indoor contaminants.
b. Arranging air inlets and air outlets to facilitate cross-ventilation will dry moisture-laden stored items and exhaust indoor contaminants.
c. Specifying fans will exhaust moisture-laden air prevalent in rooms such as kitchens, bathrooms, hobby rooms, and utility rooms.
d. Specifying air-to-air exchanger units can be used to filter incoming air for interior living spaces and to exhaust contaminants at their source.

The arrangement of interior spatial volumes in conjunction with openings in the architectural shell of a building can impact moisture intrusion and dissipation.

e. Maximizing day lighting and solar gain in the interior will retard microbial growth.
f. Planning for adequate space to ensure airflow around furnishings will dry moisture-laden areas.
g. Providing transitional zones such as vestibules, air lock entries, mudrooms, or breezeways to shed contaminants from outer garments and footwear reduces airborne contaminants entering living areas.

The specification of interior products contributes to prevalence of moisture and nutrient matter within the breathing zones of buildings. Specification of products includes interior finishes, fabrics, furnishings & equipment.

h. Specifying porous wall coverings on perimeter interior walls will allow surfaces with temperature

differences to 'breathe.'

i. Specifying wall materials so that permeability increases from exterior cladding to interior finish will allow moisture within the wall cavity to dissipate.
j. Specifying non-porous finishes for interior walls with low temperature difference between rooms can avoid entrapment of moisture and organic materials due to activities of the building occupant.
k. Specifying nonporous, continuous work surfaces will avoid entrapment of moisture and organic materials.
l. Specifying non-fleecy interior surfaces and designing built-in furniture reduces surface-to-volume ratio for collection of dust and the sink effect of VOCs.
m. Specifying non-fleecy fabrics and designing built-in window treatments reduces surface-to-volume ratio for collection of dust and sink effect of VOCs.
n. Specifying finishes and fabrics that easily can be cleaned (laundered or wiped clean) will prevent build up of contaminants.
o. Specifying non-cellulose materials such as ceramic, glass, and metal decreases hospitable habitats for microbial growth.
p. Specifying open storage containers will allow stored items to 'breathe'.
q. Specifying upholstery with open construction allows for air movement and dissipation of body heat.

Reactive Approach

Strategies to minimize exposure to contaminants in indoor air as a result of moisture intrusion focus on their removal, isolation, and dilution. These strategies are a response to flooding and damage control for water leakage and condensation.

a. Drying contaminated materials with daylight and air and exposing contaminated materials to ultraviolet light will kill mold.
b. Depressurizing the building through exhaust fans and natural ventilation will dry wet materials.
c. Elevating the building to facilitate airflow in the crawl space will retard microbial growth.
d. Encapsulating fungi embedded in organic materials will prevent their exposure to interior breathing zones.
e. Cleaning, drying, sealing, and maintaining the building will prevent damage from water intrusion and microbial growth.
f. Pressurizing the interior with central air systems can prevent infiltration of ambient pollutants and dormant spores within cavities of the architectural shell.
g. Drying, cleaning, and disinfecting nonporous interior products will kill mold and remove dormant fungal spores.
h. Disposing of porous interior products such as bedding, upholstery, thermoplastics draperies, softwoods, and paper products will remove interiors furnishings and accessories with embedded mold.
i. Seeking restoration specialists to remove mold and dormant fungal spores from porous products can preserve valuable belongings.

Caveats to 'best practices' advice

Important trade-offs should be factored into design decisions between 'best practices' advice and other considerations regarding upfront building costs, building maintenance and operation, and building occupant comfort, health and well-being. These considerations are relevant to ventilation strategies and specification of interior products.

Procedures for operating mechanical systems, designing passive convection systems, and tightening the architectural shell to address reductions in energy consumption can be counterproductive to occupant health.

a. Strategies to address energy efficiency can result in high relative humidity for indoor air conducive to growth of molds (e.g., cycling air with no cooling or heating on weekends; bringing in unfiltered ambient air in humid climates via passive convection).
b. Negative air pressure fans used to exhaust moisture also can draw dormant spores out of crawl spaces and wall cavities into interior breathing zones.
c. Negative air pressure caused by inadequate ventilation in tightly sealed buildings may cause back drafts from combustion appliances and equipment, releasing pollutants into indoor air.

Decisions regarding the selection of interior products can be counterproductive to occupant health with regards to building occupants with heightened sensitivities to noise pollution, allergens and toxins.

d. Acoustics - specifying non-fleecy surfaces may increase transmission of unwanted sounds.
e. Chemical sensitivity - using chemicals for cleaning and in finishes that retard microbial growth (e.g., fungicides, bleach, and desiccants) may trigger allergic or toxic response.
f. Maintenance - interior finishes specified in office and hotel facilities for their ease of maintenance often have a low permeability rating. Inability of exterior wall cavities to 'breathe' can result in condensation and growth of mold.

Certification Programs & Consultancies

Certification programs and consultancies have emerged in the fields of building sciences and industrial hygiene. Current efforts in the development of certification programs to rate building materials and interior products include the following:

a. Leadership in Energy and Environmental Design (LEED) program is a voluntary certification program that analyzes content of building materials in new facilities (US Green Building Council, 2002; Mendler, 2002). Beginning with exterior materials, the program is currently focusing on interior materials for commercial buildings and eventually will develop a materials rating system for remodeling of old buildings. Criteria of this program include recycled content, energy efficiency, and VOC emissions.
b. Air Quality Sciences, Inc. has a Greenguard certification program that rates building materials according to biologicals and toxins (Air Quality Sciences, Inc., 2002).
c. Industrial Hygiene association regulates a mold remediation certification program that has been developed along the lines of asbestos removal and radon mitigation (American Industrial Hygiene Association's EMLAP Program).
d. McDonough Braungart Design Chemistry (MBDC) and Environmental Protection Encouragement Agency (EPEA) provide consulting services on chemical composition of interior products from the perspective of 'cradle-to-cradle' impact on the natural environment and wellness of building occupants (McDonough, 2003).

The Interior Design Community

Interior design practice and education contribute to the prevention and remediation of mold growth in indoor environments. The literature points to three prevailing philosophical perspectives with regards to interior design practice that influences approaches to achieving a mold-free environment.

A traditional materialist philosophy, Indicative of mainstream interior design practice values materials for their appearance, durability, and availability (Nielson & Taylor, 2002). Designers subscribing to this philosophy have focused on client considerations to the exclusion of ecological considerations of protecting and preserving the natural environment. If economy is a consideration, the expectation is that designing with ecologically sensitive products will increase the cost for completion of the project (Beecher and Davies, 2002). Historically, knowledge underlying interior design decisions has been held by an elite group and applied on behalf of an elite clientele.

An eco-materialism philosophy, indicative of a growing interest in the green design movement and sustainable design movement values materials for their environmental sensitivity over availability, convenience, or appearance (Beecher and Davies, 2002). This philosophy embraces an ecology-centered approach that expands traditional measures of building performance (e.g., cost per square feet, minimum standards for air quality) to include issues of building occupant comfort, satisfaction, and overall well-being. Health factors affecting material selections would include VOC emissions and susceptibility to allergens and toxins. Long-term environmental impact would be considered in tandem with upfront building costs (Moussatche, King, and Rogers, 2002). Knowledge based on the science of product quality can effect changes to the way interior products are manufactured, the composition of interior products, and the specification of products by the interior designer (McDonough, 2003).

A Human ecology (Home Economics) philosophy, indicative of a cooperative extension approach to design values materials for their durability and for wise use of natural resources within a tradition of supporting health and wellness in the family. This philosophy embraces a holistic approach to design decisions that includes considerations of the environment and human needs (e.g., University of Florida, University of Minnesota, Kansas State University). Knowledge based on science and best practices developed by federal and state government (e.g., programs addressing disaster relief for flooding) is translated to a layman's language to empower the consumer.

Conclusions - Toward a Hybrid Model of Benign Design

The professional community of design education and practice could benefit from the development of a design model that reflects a merger of values indicative of an aesthetic tradition (beautiful design), a science of product quality evolving from the environmental movement (contaminant-free design), and the cooperative extension tradition of empowering individuals and families (user-driven design). Needed to support this model is a more comprehensive metrics to evaluate building performance inclusive of measures of improved occupant health and well-being and life-cycle analyses of interior products that provide measures of long-term impact to the natural environment.

References

AERIAS. (2002a). *Carpet, a Haven for Unwanted Guests*. AERIAS. Available: http://www.aerias.org/sitemap.htm [2002, October 11].

AERIAS. (2002b). *Ceiling Materials and Problems Associated with Indoor Air*. AERIAS. Available: http://www.aerias.org/sitemap.htm [2002, October 11].

AERIAS. (2002c). *Healthy ("Green") Workplaces: The Economical Choice*. AERIAS [2002, October 11].

AERIAS. (2002d). *Indoor Air Problems Associated with Different Types of Flooring*. AERIAS. Available: http://www.aerias.org/sitemap.htm [2002, October 11].

AERIAS. (2002e). *Overview of Furniture in the Indoor Environment*. AERIAS. Available: http://www.aerias.org/sitemap.htm [2002, October 11].

AERIAS. (2002f). *Overview of IAQ Problems in Homes and Apartments*. AERIAS. Available: http://www.aerias.org/home_overview.htm [2002, October 11].

AERIAS. (2002g). *Prevention of Indoor Air Quality (IAQ) Problems in the Home*. AERIAS. Available: http://www.aerias.org/home_prevention.htm [2002, October 11].

Air Quality Sciences, I. (2002). *Greenguard Registry: The World's Leading Guide to Healthy Indoor Products and Materials*. Air Quality Sciences, Inc. [2002, October 11].

American Red Cross, & Agency, F. E. M. (1992, 2001). *Repairing Your Flooded Home*, [Guidance Document]. American Red Cross

Federal Emergency Management Agency. Available: http://www.redcross.org/pubs/dspubs/cdelist.html [2002, November 11].

Beecher, M. A., & Davies, B. (2002, March 19-24). *Shades of Green: The Philosophical Challenges of Ecological Responsibility in Interior Design Education and Practice.* Paper presented at the Mesas and the Mysteries: On the Edge of Imagination/Green Design, Santa Fe, NM.

Bode, M., & Munson, D. (1995). *Controlling Mold Growth in the Home*, [Guidance Document]. Kansas State University Agricultural Experiment Station and Cooperative Extension Service. Available: http://www.oznet.ksu.edu/library/hous2/mf2141.pdf [2002, November 11].

Coleman, C. (1999). Life-cycle design: Leaving future generations a legacy. *Perspective* (Spring/Summer), 27-28, 30.

Dickson, A. W., & White, A. C. (1994). The Polsky Forum: The Creation of a vision for the interior design profession in the year 2010. *Journal of Interior Design, 20*(2), 3-11.

Durst, C. S. (2000a). Assessing the Future of Green Design. *Perspective: Journal of the International Interior Design Association* (Fall/Winter 2000), 30-33.

Durst, C. S. (2000b). Dateline: Design. *Perspective: Journal of the International Interior Design Association* (Fall/Winter 2000), 9-12.

Foundation Interior Design Education Research. (2001). *FIDER Professional Standards 2000* (Standards and Guidelines Document). Grand Rapids, MI: Foundation of Interior Design Education Research.

Gates, G. S., & Yousri, K. A. (1996). Strategic Planning for Interior Design Education: Effective Development in Conditions of Resource Decline. *Journal of Interior Design, 22*(1), 45-50.

Guerin, D. A. (1992). Issues facing interior design education in the twenty-first century. *Journal of Interior Design Education and Research, 17*(2), 9-16.

Hasell, M. J., & Scott, S. C. (1996). Interior Design Visionaries' Explorations of Emerging Trends. *Journal of Interior Design, 22*(2), 1-14.

Heerwagen, J. H. (2000). Design Research. *Perspective: Journal of the International Interior Design Association* (Winter/Spring 2000), 50-53.

McDonough, W. & Braungart, M. Redefining Green: A New Definition of Quality Empowers the Next Wave of

Design. *Perspective: Journal of the International Interior Design Association* (Spring 2003), 20-25.
Mendler, S. (2002). LEED: A Roadmap for Added Value. *Perspective: Journal of the International Interior Design Association* (Winter 2002), 42-49.
Miller, B. R., Miller, P. B., & Bateman, M. S. (2002, March 19-24). *Two's Company, Three's a Good Team: Multidisciplinary Teams are a 21st Century Necessity.* Paper presented at the Mesas and the Mysteries: On the Edge of Imagination/Green Design, Santa Fe, NM.
Miller, J. W. (2002). Green Home Building. *Perspective: Journal of the International Interior Design Association* (Fall 2003), 22-27.
Minnesota Department of Commerce. (2001, November 7, 2002). *Home Moisture*. Minnesota Department of Commerce - Energy Information Center [2002, November 11].
Minnesota Department of Commerce. (2002a, June 24, 2002). *Ice Dams*, [Home Energy Guide]. Minnesota Department of Commerce - Energy Information Center. Available: http://www.commerce.state.mn.us/pages/Energy/InfoCenter/EnergyGuides.htm [2002, November 11,].
Minnesota Department of Commerce. (2002b, June 24, 2002). *Indoor Ventilation*, [Home Energy Guide]. Minnesota Department of Commerce - Energy Information Center. Available: http://www.commerce.state.mn.us/pages/Energy/InfoCenter/energyGuides.htm [2002, November 11].
Minnesota Department of Commerce. (2002c, June 24, 2002). *New Homes*, [Home Energy Guide]. Minnesota Department of Commerce - Energy Information Center. Available: http://www.commerce.state.mn.us/pages/Energy/InfoCenter/EnergyGuides.htm [2002, November 13].
Minnesota Department of Health. (2001, November 7, 2002). *Recommended Best Practices for Mold Investigations in Minnesota Schools*, [Guidance Document]. Minnesota Department of Health - Indoor Air Unit. Available: http:/www.health.state.mn.us/divs/eh/indoorair/mold/index.html [2002, November 11, 2002].
Moussatche, H., King, J., & Rogers, T. S. (2002, March 19-24, 2002). *Material Selection in Interior Design Practice.* Paper presented at the Mesas and the Mysteries: On the Edge of Imagination/Green Design, Santa Fe, NM.
Nielson, K., & Taylor, D. (2002). *Interiors: An Introduction*. Boston: McGraw Hill.
US Environmental Protection Agency. (2001a, May 28, 2002). *Healthy Buildings, Healthy People: A Vision for the 21st Century*, [Guidance Document]. US Environmental Protection Agency. Available: http://www.epa.gov/iaq/hbhp/index.html [2002, November 11].
US Environmental Protection Agency. (2001b, December 31, 2001). *Mold Remediation in Schools and Commercial Buildings*, [Guidance Document]. US Environmental Protection Agency. Available: http://www.epa.gov.iaq/pubs [2002, November 11].
US Environmental Protection Agency. (2002, December 31, 2001). *A Brief Guide to Mold, Moisture, and Your Home*, [Guidance Document]. US Environmental Protection Agency. Available: http://www.epa.gov/iaq/pubs [2002, November 11].
US Green Building Council. (2002). *Leadership in Energy and Environmental Design*. US Green Building Council. Available: http://www.usgbc.org/LEED/publications.asp [November 11, 2002].
Wilson, K. P. (2002). The Case for Green Design. *Perspective: Journal of the International Interior Design Association* (Winter 2002), 21-26.
Ziehe, J. (1994). 25 Principles of Bau-Biologie, *AESCLEPIOUS,* 3(3): 3.

Abstract Information & Notes

Aristo Vojdani, P.H.D., M.T.

Immunosciences Lab., Inc.
8693 Wilshire Blvd., Suite 200
Beverly Hills, CA 90211

Phone: 301/657-1077
Fax: 310/657-1053
E-mail: immunsci@ix.netcom.com

Date of talk: Friday, June 20, 2003, 9:35am

Medical School Attended:	Bar-Ilan University Ramat-Gan Israel
Major and date of Graduation:	Immunology 1976
Residency:	University of California Los Angeles 1979-2001
Board Certifications:	Laboratory Medicine
Current Faculty Appointments:	A Clinical Professor UCLA Dept. Neurobiology
Current Job Description:	Research in the field of Neuroimmunology at Immunosciences Lab. And at UCLA School of Medicine
Disclosure Statement:	Part owner of Immunosciences Lab. Inc., and have financial interest in the company.

SPEECH TITLE: "**Immunotoxicology of Molds and Mycotoxins"**

The speaker has provided the information below.

1.) Goals and objectives: To understand the impacts of molds and mycotoxins on the human immune system

2.) Outline of talk/abstract: 1. Sign and symptoms of patients exposed to toxigenic molds in water-damaged building. 2. Detection of mucosal immune response to molds. 3. Detection of cellular and humoral immune responses to molds and mycotoxins. 4. Neurotoxicity induction by molds and mycotoxins.

3.) Conclusion of what is to be learned: A. Exposure to toxigenic molds and mycotoxins can induce type 1 to type 4 allergic reactions. B. Allergy evaluation by skin test or in vitro tests for detection of IgE mediated allergies are not enough for detection of molds induced toxicity. C. Prolonged and intense exposure to molds and mycotoxins is associated with disorders of mucosal, cellular and humoral immune system.

4.) References:

1. Vojdani et al., Antibodies Against Molds and Mycotoxins after Exposure to Toxigenic Fungi in a Water Damaged Building (submitted)
2. Mahfoud R., et al., The Mycotoxin Patulin Alters the Barrier Function of the Intestinal Epithelium: Mechanism of Action of the Toxin and Protective Effect of Glutathione. Toxicology and Applied Pharmacology 181:209, 2002

Abstract Information & Notes

Bruce Small

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

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

Date of talk: Friday, June 20, 2003, 10:30am

Medical School Attended: University of Toronto, Engineering Science, B.A.Sc., Southern Illinois University, Physics and Design, M.S. 1975

Current Job Description: Director of the Enviorodesic Certification Program, a private sector initiative to identify and foster the development of building materials and other products that will lead to healthier indoor environments.

Other Information: Executive Director of Technology and Health Foundation, a Canadian charity focused on environmental health.

Disclosure Statement: Icynene Inc. and Cogent Environmental Solutions

SPEECH TITLE: **"Prescription for Preventing Mold and for Mold Remediation"**

The speaker has provided the information below.

1.) Goals and objectives: To outline common causes of mold growth and regrowth in buildings. To outline primary means of preventing mold growth and regrowth in buildings.

2.) Outline of talk/abstract: Existing building and maintenance practices predispose many buildings to mold growth. Current mold remediation methods and building reconstruction practices may also lead to mold regrowth. Environmental physicians are in a unique position to specify effective building science principles to promote mold-free buildings and more effective remediation. When designed, built and maintained with sufficient thought and care, there is no reason for buildings to go moldy.

3.) Conclusion of what is to be learned: Proper use of rain screens, air barriers, and vapor barriers can alleviate some of the primary causes of building envelope moisture penetration and mold growth. Timely maintenance is also paramount. Homeowners, school boards and building managers armed with this knowledge are in a better position to avoid occupant mold exposures. Physicians can play a role by advocating the use of good building science to prevent mold growth.

4.) References: To be included in presentation notes.

Useful web sites on building science for avoiding mold growth:

http://www.aiha.org/GovernmentAffairs-PR/html/prmoldsources.htm
http://www.cdc.gov/nceh/airpollution/mold/moldfacts.htm
http://www.epa.gov/iaq/molds/
http://www.buildingscience.com/housesthatwork/default.htm
http://www.buildingscience.com/resources/mold/default.htm
http://www.envirodesic.com/sfl/HomeEnergyArticle.pdf

Abstract Information & Notes

Martha Stark, M.D.

3 Ripley St.
Newton Centre, MA 02459-2209

Date of talk: Friday, June 20, 2003, 11:00am

Phone: 617/244-7188
E-mail: marthastarkmd@hms.harvard.edu

Medical School Attended:	Harvard Medical School
Major and date of Graduation:	M.D., 1974
Residency:	Adult Psychiatry Residency - The Cambridge Hospital/Cambridge, MA Child Psychiatry Fellowship - Massachusetts Mental Health Center/Boston, MA
Board Certifications:	None (Board-Eligible in Adult and Child/Adolescent Psychiatry)
Current Faculty Appointments:	Faculty: Harvard Medical School; Boston Psychoanalytic Institute; Massachusetts Institute for Psychoanalysis; Center for Psychoanalytic Studies, Massachusetts General Hospital; Department of Continuing Education, Massachusetts Mental Health Center; Continuing Education Program, Massachusetts School of Professional Psychology; and Program of Continuing Education, Smith College School for Social Work.
Current Job Description:	Private practice of psychiatry/psychoanalysis; various faculty appointments and teaching responsibilities; teaching/supervising analyst at several local psychoanalytic institutes; speaking engagements at training institutes around the country; completing fourth book and at work on fifth book
Other Information:	Author of four books
Disclosure Statement:	none

SPEECH TITLE: **"Mysterious Mental Illnesses, Pernicious Poisons"**

The speaker has provided the information below.

1.) Goals and objectives:

a. To understand that mycotoxins have the power both to intoxicate and to poison
b. To appreciate all the mysterious ways in which mycotoxins can affect the mind (and the brain)

2.) Outline of talk/abstract: Mycotoxins have figured more prominently in human affairs than most would have suspected B in both good ways and bad. They have been used since earliest times as mind-altering hallucinogens and, as such, have promoted psychedelic/mystical experiences. Furthermore, evidence suggests that these poisons have also been responsible for "bewitching" people (as Happened, for example, in Salem, Massachusetts, during the 17th century). Particularly virulent strains have been known to cause bizarre physical ailments, mystifying mental disorders, and, in some instances, a slow steady decline into deathBwhich is why some mycologically savvy mystery writers have recognized that some mycotoxins are perfect murder weapons! Finally, mycotoxins are now understood to have been responsible for wiping out whole groups of people who had the misfortune of consuming foods contaminated with mold (as happened, for example, during the Irish potato famine).

3.) Conclusion of what is to be learned:

a. To develop a healthy respect for mycotoxins and their potency
b. To recognize that patients with certain mysterious mental illnesses (patients once described as simply "mad" or "deranged") are actually suffering from poisoning by mycotoxins

4.) References:

Hudler, G.W. (2000). **Magical Mushrooms, Mischievous Molds**. Princeton, NJ: Princeton University Press. Princeton and Oxford: Princeton University Press

Matossian, M.K. (1989). **Poisons of the Past: Molds, Epidemics, and History**. New Haven and London: Yale University Press.

Schaechter, E. (1998). **In the Company of Mushrooms: A Biologist=s Tale**. Cambridge, MA: Harvard University Press.

MYSTERIOUS MENTAL ILLNESSES, PERNICIOUS POISONS
Martha Stark. M.D. -- Harvard Medical School

Harvard Health Letter (January 2003) – "The Truth About Mold"
"Most experts say there's more fear than fact to 'toxic mold.'"

neither mental illness nor mental health can be meaningfully understood without reference to the brain and the nervous system
our minds do not exist in a vacuum, they are linked to the chemistry of the brain

molds can have a **negative effect** on human health in three ways:

1. as **pathogens** (whereby they take up residence either in or on the body of those whose immune systems are compromised)
result: **infectious symptoms**

2. as **allergens** (whereby they provoke either immediate or delayed hypersensitivity reactions in those who are sensitized to molds)
result: **allergic symptoms**

3. as **toxins** (whereby they poison otherwise healthy individuals)
result: **toxic symptoms**
neurotoxic symptoms when the brain is involved

moisture + warmth ---> germination of the spore
balloon-like protuberances (hyphae) that **exude powerfully toxic digestive enzymes, which systematically break down whatever they can find to feed upon**
the hyphae then take back in what's left of the ravaged substrate, where it is further degraded in order to nourish the mold's relentless growth

what happens when mycotoxins come into contact with the brain?
although we have yet to understand the exact mechanism of action whereby mold toxins affect how we feel, think, and act, there is **much that we can learn about both mental illness and mental health by studying the impact of various mycotoxins on the brain and, more particularly, on brain neurotransmitters and their receptors**

in fact, **mycotoxins have been found to bear a striking resemblance to specific neurotransmitters in the brain--especially serotonin**

in other words, mycotoxins are in a **prime position to compete with serotonin for (re-)uptake at various postsynaptic 5-HT (5-hydroxytryptamine) receptor sites**

in essence, mycotoxins appear to **affect the central nervous system at the level of the synapse**

Claviceps purpurea - a perniciously poisonous mold that produces a variety of mycotoxins known as **ergot alkaloids**
ingestion of products made from ergot-infected rye (like bread) --->
ergot poisoning, a disease that is thought to have affected the physical and mental health of people throughout much of the Middle Ages

gangrenous ergotism involves an ergot alkaloid that can be such a powerful vasoconstrictor that it can restrict entirely the flow of blood to body parts, particularly the extremities

convulsive, hallucinogenic ergotism involves an ergot alkaloid that has a more direct effect on the brain
symptoms range from (1) vomiting, diarrhea, and general lethargy; to (2) a sensation of ants crawling on the skin (formication), twitching, and grotesque distortion of limbs; to (3) vivid hallucinations and seizures similar to

those associated with epilepsy

the mental effects of ergot poisoning are said to be unremittingly unpleasant: fright, outright panic, and an inability to maintain a sense of distance in the face of such strange experiences

the historian **Mary Matossian** (in her book Poisons of the Past) has hypothesized that **"bewitched" people in central Europe between the 16th and 19th centuries were actually exhibiting symptoms of ergot poisoning** (both gangrenous and convulsive)

furthermore, the **women (and men) in Salem, Massachusetts, accused in 1692 of being witches (and wizards) either were themselves afflicted with ergotism or, interestingly, were wrongly accused by others who had become paranoid and delusional because they were afflicted with ergotism**

from earliest times, **mycotoxins** have been implicated as the cause of several **major human epidemics**

but, from earliest times, mycotoxins have also been found to **produce mind-altering, consciousness-enhancing, mind-expanding, bewitching, intoxicating, inebriating, psychedelic, hallucinogenic experiences** that have afforded the user much pleasure

many of the most respected **Greek philosophers--like Plato and Socrates**--were inspired to formulate some of their greatest thoughts when they were under the influence of certain **"magical mushrooms"** (and their mycotoxins)

hallucinogenic mushrooms (also known as 'shrooms) are still in use today as recreational drugs

chemical analyses of 'shrooms have determined that they contain psilocybin and psilocin, psychoactive substances that closely resemble serotonin (the "feel good" hormone)--which lends further credence to the hypothesis that all the major plant hallucinogens contain substances similar in chemical composition to psychoactive chemicals within the brain

1938 - birth of the psychedelic age

a young German chemist, **Dr. Albert Hofmann**, began experimenting with **lysergic acid, the chemical backbone of the ergot alkaloids**

five years later, he was able to synthesize **lysergic acid diethylamide--LSD**

Dr. Hofmann described his first unintentional use of LSD as a "not unpleasant intoxicated-like condition" involving "interesting imagery" that lasted for several hours

"...all objects appeared in unpleasant, constantly changing colors, the predominant shades being sickly green and blue. When I closed my eyes, an unending series of colorful, very realistic, and fantastic images surged in upon me. A remarkable feature was the manner in which all acoustic perceptions (e.g., the noise of a passing car) were transformed into optical effects, every sound invoking a corresponding colored hallucination constantly changing in shape and color like pictures in a kaleidoscope. At about one o'clock I fell asleep and awoke the next morning feeling perfectly well."

Hofmann noted that some of the more bizarre symptoms he endured on subsequent "trips" were remarkably similar to those accompany schizophrenia

amongst other things, **schizophrenics frequently speak of having out-of-body experiences and "interpret" sights and sounds in distinctly idiosyncratic ways--much as he had when he was "tripping"**

Hofmann's experience certainly lent support to the hypothesis that **certain mental illnesses, initially presumed to be purely psychic in nature, might actually have a biochemical cause**

but actually long before Dr. Hofmann's discovery of LSD, **Indians in southern Mexico** had discovered these same mushrooms growing in the wild--intoxicating mushrooms that proved to be hallucinogenic when ingested

the visions so produced were thought to be **divinely inspired and capable of leading to spiritual enlightenment**

in the **1950s**, **Gordon Wasson** spent time in Mexico studying the effects of these **inebriating mushrooms**

"The sacred mushrooms of Mexico seize hold of you with irresistible power. They lead to a temporary schizophrenia in which your body lies heavy as lead on the mat ... while your soul flies off to the ends of the world and indeed to other planes of existence."

“I experienced hallucinations...visions of palaces, gardens, seascapes, and mountains... With the speed of thought, you are translated wherever you desire to be, and you are there, a disembodied eye, poised in space, seeing but not seen, invisible, incorporeal. ...All of <your> senses are equally affected, and the human organism as a whole is lifted to a place of intense experience. A drink of water ... is transformed, leaving you breathless with wonder and delight. The emotions and intellect are similarly stepped up. Your whole being is aquiver with life.”

“What is happening to you seems freighted with significance, beside which the humdrum events of every day are trivial. All these things you see with an immediacy of vision that leads you to say to yourself: 'Now I am seeing for the first time, seeing direct without the intervention of mortal eyes.’”

and in **1960**, at the **Center for Research in Personality at Harvard University**, **Dr. Timothy Leary (with his colleague Dr. Richard Alpert)** was doing his own experimenting with magical mushrooms

“I was whirled through an experience which could be described ... as above all and without question the deepest religious experience of my life.”

more generally, it would seem that **magical mushrooms (and their mycotoxins)** are appealing in large part because they **transport the user into other realms where knowledge is intuitive, non-linear, dynamic, rather than reasoned, linear, ordered**

intense experiences of vivid colors and abstract patterns alternate with visions of mystical union, cosmic connectedness, and transcendence of the usual boundaries of time and place

later still, none other than the **CIA** began its own experimentation with the hallucinogenic drugs produced by magical mushrooms

they were **searching for a mind-control agent that would allow them more effectively to interrogate suspected spies, preferably without the spy's knowledge of this interrogation**

but the magical mushrooms were found to have **many untoward and some potentially lethal side effects**

so by the late 1960s, early 1970s, all scientific experimentation (whether in the laboratories of academic institutions or governmental agencies) had ground to a halt

the psychoactive ingredients in magical mushrooms appear to alter a person's state of consciousness, affecting how she feels, thinks, and acts

overall, **her senses are heightened, her feelings are exaggerated, and her perceptions are intensified**

emotional changes may occur **suddenly, dramatically, unpredictably**

the **distinction between past, present, and future may become blurred**

cognitive impairment may take the form of fluctuating attention, a short attention span, memory loss, word finding difficulties

there is often **a profusion of vague ideas and a preoccupation with philosophical and existential issues**--but because of impaired judgment and illogical thought processes, the person may imagine that she has “discovered” new truths when, in fact, she is creating “word salads” (akin to those produced by schizophrenics) that usually do not stand up to the light of day

as noted earlier, although the exact mode of action of mycotoxins in the brain has not yet been established, **presumably both LSD and psilocybin are psychoactive chemicals that alter how we feel, think, and act by disrupting the action of the neurotransmitter serotonin--a monoamine chemical messenger known to play a major role in the regulation of mood, energy, appetite, libido, sleep, and cognitive functioning**

many of the effects of LSD are through serotonin-mediated pathways (particularly those involving the dorsal raphe nucleus, which lies along the midline of the brainstem and projects to most parts of the brain, including the cerebral cortex and the limbic system)

the **hallucinogenic effects of LSD** have **mainly been attributed to the interaction of this drug with the serotonergic system, but it seems more likely that there are complex interactions of LSD with dopaminergic and noradrenergic target sites as well**

studies suggest that **LSD** not only **penetrates the blood-brain barrier but slips slyly into the transmission site inside the nerve cells themselves**

it can mimic serotonin to the point where **the brain thinks it is serotonin and consequently shoots it across the synaptic cleft**

when LSD reaches the other side, it is accepted by the serotonin receptor sites (particularly the 5-HT2A receptor sites)--but now the jig is up, because the LSD cannot carry the message any further

in essence, **the LSD blocks or "antagonizes" the normal activity of the serotonergic relay system**

instead, the electrical impulse (the action potential) generated by the LSD at the receptor site is redirected more or less randomly down either new or less familiar pathways--pathways that may not have been highly conditioned, pathways that may not have been frequently traveled

it could be said that **consciousness is "redirected" to unimprinted areas of the cortex, thus the variety of "interpretations" of sensations, emotions, and perceptions that accompany hallucinogenic experiences**

in fact, **sensory perceptions may blend in a phenomenon known as synesthesia, a cross-circuiting of brain information in which a person sees sound, smells color, or hears motion--evidence that there is a problem with sensory integration**

in sum, the **work of Hofmann and other researchers** who have devoted their lives to studying mycotoxins **points not to a separation but to an association between the brain and the mind**

more specifically, biochemical events that take place in the brain with respect to neurotransmitters and their receptors are the physical correlates of psychic experiences that take place in the mind with respect to how a person feels, thinks, and acts

indeed, the mind does not exist in a vacuum; it is linked to the biochemistry of the brain

References:

Cook, Robin (1994). **Acceptable Risk**. New York: Berkley Books.

Grafton, Sue (1992). **"I" is for Innocent**. New York: Fawcett Crest.

Greene, Graham (1978). **The Human Factor**. New York: Simon and Schuster.

Harvard Health Letter - Volume 28 - Number 3 - January 2003.

Hudler, George W. (1998). **Magical Mushrooms, Mischievous Molds**. Princeton, NJ: Princeton University Press.

Kavaler, Lucy (1965). **Mushrooms, Molds, and Miracles**. New York: A Signet Book.

Masters, Robert, & Houston, Jean (2000). **The Varieties of Psychedelic Experience**. Rochester, VT: Park Street Press.

Matossian, Mary K. (1989). **Poisons of the Past**. New Haven: Yale University Press.

Ramachandran, Vilayanur, & Hubbard, Edward. **Hearing Colors, Tasting Shapes**. In Scientific American, May 2003.

Restak, Richard M. (1994). **Receptors**. New York: Bantam.

Schaechter, Elio (1997). **In the Company of Mushrooms**. Cambridge, MA: Harvard University Press.

Stahl, Stephen M. (2000). **Essential Psychopharmacology**, 2nd edition. London: Cambridge University Press.

Abstract Information & Notes

John H. Boyles, M.D.

Dayton Ear, Nose & Throat Surgeons Inc.
7076 Corporate Way
Centerville, OH 45459

Phone: 937/434-0555
Fax: 937/434-7413
E-mail: N/A

Date of talk: Friday, June 20, 2003, 11:30am

Medical School Attended:	Northwestern University Medical School, Chicago, Illinois
Major and date of Graduation:	M.D., 1960
Internship:	Latter-Day Saints Hospital, Salt Lake City, Utah; Northwestern University Medical School, Chicago, Illinois (1964-1967)
Board Certifications:	American Board of Otolaryngology and American Board of Environmental Medicine
Current Faculty Appointments:	Assistant Clinical Professor, Wright State Medical School
Current Job Description:	Private Practice - Dayton Ear, Nose and Throat Surgeons, Inc.
Other Information:	Published several articles
Disclosure Statement:	None

SPEECH TITLE: **"Diagnosis & Treatment of Inhalant and Mold Allergy"**

The speaker has provided the information below.

1.) Goals and objectives: How to properly diagnose and treat mold sensitivities through allergic desensitization using SET and traditional build-up therapy.

2.) Outline of talk/abstract: Practitioners must know the prevalent molds in their geographic location, and be able to test the individual sensitivity for each mold. In addition, they should be able to test and properly treat TOE sensitivity.

3.) Conclusion of what is to be learned: Treatment of mold sensitivity is extremely important in an allergic patient, and must be controlled before one goes on to testing of food and chemical sensitivity.

4.) References:

Diagnosis & Treatment of Inhalant and Mold Allergy

John H. Boyles, Jr., M.D.

For centuries, we have known that mold exposure and infestation is injurious to human health. In this presentation, we want to emphasize the importance of recognizing fungus infections, and treating them effectively. This includes the controversial concept of Candidiasis. We also seek to explain the importance of fungus allergy, and how to diagnose and treat it.

We will also show the importance of fungal contamination of buildings, how it relates to human health, and how it may be eradicated.

Abstract Information & Notes

David C. Straus, Ph.D.

Texas Tech University
Department of Microbiology
Texas Tech University Health Sciences Center
Lubbock, TX 79430

Phone: 806/743-2523
Fax: 806/743-2334
E-mail: david.straus@hmc.huhsc.edu

Date of talk: Friday, June 20, 2003, 1:00pm

Graduate School Attended: Loyola University of Chicago
Major and date of Graduation: Microbiology - May 1974
Current Faculty Appointments: Professor of Microbiology & Immunology
Current Job Description: Teaching and Research
Other Information: Member Sigma Xi, American Society for Microbiology, American Academy of Microbiology

Disclosure Statement: Assured IAQ7 of Dallas, TX

SPEECH TITLE: **"The Role of Fungi in Sick Building Syndrome"**

The speaker has provided the information below.

1.) Goals and objectives: To educate the audience on how fungi produce the phenomenon known as sick building syndrome

2.) Outline of talk/abstract: I will describe what organisms are important in sick building syndrome and how they produce the associated symptoms.

3.) Conclusion of what is to be learned: Fungi cause health problems inside buildings by the production of spores and mycotoxins

4.) References: Cooley et al. 1998. Correlation between the prevalence of certain fungi and sick building syndrome. Occup. Environ. Med 55:579-584. McGrath et al. 1999. Continually measured fungal profiles in Sick Building Syndrome. Curr. Microbiol. 38:33-36.

The Role of Fungi in Sick Building Syndrome

By David C. Straus, Ph.D.
Professor of Microbiology and Immunology
Texas Tech University
Health Sciences Center
Lubbock, Texas 79430

Man has known about the consequences of having mold (a form of fungi) grow inside his buildings for over 3,300 years. Verses in the Old Testament of the Bible (Leviticus 14:33-45) describe how mold infested houses should not be occupied, and could potentially lead to the destruction of the house. Therefore, it should not be surprising that mold-infestation of our buildings still remains a problem. What is different now than in the time of Leviticus, is we now know what the problematic fungi are and we know what products they produce that we should be concerned with.

Sick Building Syndrome (SBS) is a lay term, which is characterized by a constellation of symptoms. These symptoms include itchy eyes, congestion, scratchy throat, nausea, headaches, fatigue, and decreased attention span. This phenomenon was first described in 1982 and published in 1984 (1). It literally means that there is something inside a particular building that can make people sick. It does not usually refer to a bacterial pneumonia like Legionairre's Disease that can be a consequence of being in a building with a certain bacterium in the air conditioning system. Legionairre's Disease is caused by the bacterium *Legionella pneumophila* and is an actual infection. In this case, when the person leaves the building, the organism leaves with him. In the case of SBS, the fungi (for the most part) stay inside the building. However, we do know that fungal spores travel with us on and in our clothing (2).

We are constantly breathing microorganisms, both inside and outside, and the immune system of a healthy individual normally has no problem clearing the lungs of these living particles. However, immunocompromised individuals (like those with Acquired Immunodeficiency Syndrome or AIDS) are at an increased risk of a fungal pneumonia due to the inhalation of living fungal spores. Fungi can cause human disease by at least four different mechanisms. They are 1) allergic rhinitis (hay fever), 2) hypersensitivity pneumonitis (which is due to the sensitization and recurrent exposure to inhaled fungal spores), 3) toxicity (certain fungi produce toxic substances called mycotoxins), and 4) infection. The latter is not usually an important part of SBS, but the possibility exists for it to occur.

We get into trouble with fungi or mold when we allow them to grow inside our homes, schools, or commercial buildings. Fungi essentially need four things to grow: food, water, oxygen, and the appropriate temperature. If water enters a building where it should not be and cellulose containing building structures becomes wet, mold can use the building material as a food source. Fungi particularly like the paper on sheetrock, ceiling tiles and pressed particleboard. They use these materials as a food source because they are made of cellulose, which is the most abundant organic material on the planet. It is the job of fungi to break down organic material. So we should not be surprised when they begin to grow in our wet buildings. In fact, we should be surprised if it did not happen.

We recently published several studies where we examined the role of mold on building materials, in animals, and in SBS. The first study was a two-year investigation of 48 U.S. public schools (3). In that study we showed that spores of *Penicillium* species and the presence of *Stachybotrys* species inside schools correlated with the prevalence of SBS, and when these organisms were removed, and the conditions that allowed them to grow in the first place were remediated, the SBS-associated symptoms were significantly reduced.

In another study (4), examining a contaminated hotel in the Southwestern United States, we showed that even though the outside fungal profile is continually changing, the indoor fungal profile in a "sick building" tends to remain unchanged. Because it appeared from the above studies that the inhalation of *Penicillium chrysogenum* spores played a role in SBS, we undertook studies to determine the consequences of long-term intranasal instillation of *Penicillium chrysogenum* spores in a mouse model. We discovered that when mice inhale *Penicillium chrysogenum* spores, the spores remain viable for up to 36 hours in the lungs of these animals (5). We later found that the long-term inhalation of viable (but not nonviable) *Penicillium chrysogenum* spores induced type 2 T-helper cell mediated (Th2) inflammatory responses such as an increase in total and spore specific serum IgE and IgG_1, together with bronchial alveolar lavage fluid levels of interleukins 4 and 5 and peripheral and

airway eosinophilia, which are mediators of allergic reactions (6). Finally, in an effort to elucidate ways to prevent fungal mediated SBS, we evaluated fungal growth on cellulose-containing and inorganic ceiling tile (7). We showed that inorganic ceiling tile did not support the growth of *Cladosporium, Penicillium* and *Stachybotrys* while the cellulose-containing ceiling tile did. These results demonstrate that it will be possible to develop mold-resistant building materials and possibly decrease the occurrence of SBS.

What then are the health effects of exposure to mold inside our buildings? We believe that *Penicillium* and *Stachybotrys* are two of the most important genera in SBS and we believe that these two organisms affect people in different ways. Other workers have shown that *Penicillium* species spores were good potentiators of asthma (8). As stated above, we have shown that the long-term inhalation of viable *Penicillium chrysogenum* spores by mice results in an inflammatory reaction in their lungs (6). As regards *Stachybotrys* exposures, we believe that the reported health effects are due to the mycotoxins and its role in SBS is probably due to the production of these compounds. However, much more work needs to be done in this area before this can be conclusively shown. Some of the most potent mycotoxins produced by *Stachybotrys chartarum* are called trichothecenes. They are potent inhibitors of protein synthesis (9). The symptoms most commonly reported by individuals who have been exposed to *S. chartarum* include loss of balance, hair loss, hearing loss, mucosal bleeding, rashes, and vomiting (personal communications). The above symptoms are consistent with those observed when trichothecene mycotoxins were injected intravenously (0.077 mg/kg) daily for 5 days in patients with hepatic metastases. The well-known effect of the trichothecene mycotoxins on rapidly dividing cells was the impetus for their evaluation as antitumor drugs in the late 1970's. The trichothecene examined in these studies was 4,15-Diacetoxyscripenol or DAS (anquidine) (10). DAS in these patients demonstrated no antitumor activity and was so toxic its use was discontinued. The signs and symptoms of toxicity reported by these patients were hair loss, vomiting, burning erythema, diarrhea, confusion, ataxia, fever, chills, and hypotension.

In conclusion it appears that fungi are important in SBS but the exact roles of their spores and mycotoxins remains to be elucidated. However, it appears quite clear that the inhalation of high concentrations of fungal spores is responsible for the respiratory complications (allergies, hypersensitivity pneumonitis and asthma) associated with SBS. A high number of peer-reviewed scientific publications are available to support this hypothesis (8, 11, 12, 13, 14, 15, 16, 17, 18). Although the data seem to indicate the importance of *Penicillium* and *Stachybotrys* species in this phenomenon, we believe that other fungi such as *Chaetomium* species (19), *Alternaria* species (8), *Memnoniella* species (9), and *Aspergillus* species (20) also play a role.

REFERENCE

1. Finnigan, M.S., Pickering, C.A.C., and Burge, P.S. (1984). The sick building syndrome: prevalence studies. *British Medical Journal*, 289,1573-1575.

2. Dart, B.L., and Obendorf, S.K. (2000) Retention of *Aspergillus Niger* spores on textiles. *In* Nelson, C.N. and N.W. Henry (ed). Performance of Protective Clothing: Issues and Priorities for the 21st Century: Seventh Volume, ASTM STP 1386. American Society for Testing and Materials, West Conshohocken, PA, pg 251-268.

3. Cooley, J.D., Wong, W.C., Jumper, C.A., and Straus, D.C. (1998). Correlation between the prevalence of certain fungi and sick building syndrome. *Occupational and Environmental Medicine*, 55, 579-584.

4. McGrath, J.J., Wong, W.C., Cooley, J.D., and Straus, D.C. (1999). Continually measured fungal profiles in sick building syndrome. *Current Microbiology*, 38, 33-36.

5. Cooley, J.D., W.C. Wong, C.A. Jumper, and D.C. Straus. (1999). Cellular and humoral response in an animal model inhaling *Penicillium chrysogenum* spores. Proceedings of the Third International Conference on Bioaerosols, Fungi and Mycotoxins, pp 403-410.

6. Cooley, J.D., Wong, W.C., Jumper, C.A., Huston, J.C., Williams, H.J., Schwab, C.J., and Straus, D.C. (2000). An animal model for allergic penicilliosis induced by the intranasal instillation of viable *Penicillium chrysogenum* conidia. *Thorax,* 55, 489-496.

7. Karunasena, E., Markham, N., Brasel, T., Cooley, J.D., and Straus, D.C., (2000). Evaluation of fungal growth on cellulose-containing and inorganic ceiling tile. *Mycopathologica* 150: 91-95.

8. Licorish, K., Novey, H.S., Kozak, P., Firshter, R.D., and Wilson, A.F., (1985). Role of *Alternaria* and *Penicillium* spores in the pathogenesis of asthma. *Journal of Allergy and Clinical Immunology*, 76, 819-825.

9. Jarvis, B.B., Sorenson, W.G., Hintikka, E.L., Nikulin, M., Zhou, Y., Jiang, J., Wang, S., Hinkley, S., Etzel, R.A., and Dearborn, D. (1998), Study of toxin production by isolates of *Stachybotrys chartarum* and *Memnoniella echinata* isolated during a study of pulmonary hemosiderosis in infants. *Applied and Environmental Microbiology,* 64, 3620-3625.

10. Wannemacker, R.W., and Wiener, S.L. (1997). Trichothecene Mycotoxins. *In* Textbook of Military Medicine, Part I. Warfare, Weaponry, and the Casualty, Medical Aspects of Chemical and Biological Warfare, (eds). Zajtchuk, R, and Bellamy, R.F. Published by the Office of the Surgeon General, Chapter 34, pp 655-676.

11. Apostolakos, M.J., Rossmore, H., and Beckett, W.S. (2001). Hypersensitivity pneumonitis from ordinary residential exposures. *Environmental Health Perspectives,* 109, 979-981.

12. Jaakkola, M.S., Nordman, H., Piipari, R., Uitti, J., Laitinen, J., Karjalainen, A., Hahtola, P., and Jaakkola, J. (2002). Indoor dampness and molds and development of adult-onset asthma: A population-based incident case-control study. *Environmental Health Perspectives*, 110, 543-547.

13. Jacob, B., Ritz, B., Gehring, U., Koch, A., Bischof, W., Wichman, H.E., and Heinrich, J. (2002) Indoor exposure to molds and allergic sensitization. *Environmental Health Perspectives*, 110, 647-653.

14. Dales, R.E., Burnett, R., and Zwanenburg, H. (1991). Adverse health effects among adults exposed to home dampness and molds. *American Review of Respiratory Disease*, 143, 505-509.

15. Dhasmage, S., Bailey, M., Roven, J., Abeyawickrama, K., Cao, D., Guest, D., Rolland, J., Forbes, A., Thien, F., Abramsch, M., and Walters, E.H. (2002). Mouldy houses influence symptoms of asthma among atopic individuals. *Clinical and Experimental Allergy*, 32, 714-720.

16. Downs, S.H., Mitakakis, T.L., Marks, G.B., Car, N.G., Belousova, E.G., Leuppi, J.D., Xuah, W., Downie, J.R., Tobias, A., and Peat, J.K. (2001). Clinical importance of *Alternaria* exposure in children. *Respiratory and Critical Care Medicine*, 164, 455-459.

17. Fung, F., Tappen, D., and Wood, G. 2000. *Alternaria*-associated asthma. *Applied Occupational and Environmental Hygiene*, 150, 924-927.

18. Fergusson, R.J., Milne, L.J.R., and Crompton, G.K. (1984.) *Penicillium* allergic alveolitis: faculty installation of central heating. *Thorax*, 39, 294-298.

19. Abbott, S.P., Sigler, L., McAleer, R., McGough, D.A., Rinaldi, M.G., and Mizell, G. (1995). Fatal cerebral mycoses cause by the ascomycete *Chaetomium strumarium. Journal of Clinical Microbiology,* 33, 2692-2698.

20. Hodgson, M.J., Morey, P., Leung, W.Y., Morrow, L., Miller, D., Jarvis, B.B., Robbins, H., Halsey, J.F., and Storey, E. (1998). Building-associated pulmonary disease from exposure to *Stachybotrys chartarum* and *Aspergillus versicolor*. *Journal for Environmental Medicine*, 40, 241-249.

Abstract Information & Notes

Tapani Tuomi

Finnish Institute of Occupational Health (FIOH)
Arinatie 3 A
Helsinki, Finland FIN-00370

Phone: 358-9-47472926
Fax: 358-9-5061087
E-mail: tapani.tuomi@occuphealth.fi

Date of talk: Friday, June 20, 2003, 1:30pm

School Attended: Helsinki University of Technology
Major and date of Graduation: DR, Chemical Engineering (Applied Microbiology), 1995
Current Faculty Appointments: Docent in Environmental Chemistry and Microbiology, Helsinki Univ. of Technology
Current Job Description: Laboratory Chief, Laboratory of Chemistry and Microbiology, Finnish Inst. Of Occupational Health, Helsinki, Finland

Disclosure Statement: None

SPEECH TITLE: **"Mycotoxins in Cigarettes and in Tobacco Smoke"**

The speaker has provided the information below.

1.) Goals and objectives: To present current literature on the mycotoxin content of tobacco products as well as research data on the carry-over of mycotoxins to mainstream and side stream tobacco smoke

2.) Outline of talk/abstract: The harvesting and production of tobacco products is vulnerable to contamination by mycotoxin producing fungi. There is evidence that tobacco products may contain aflatoxins and aflatoxins have been suggested to contribute to the cancer outcome of smokers. The talk will review current literature on the mycotoxin content of tobacco products. Potential exposure routes are discussed and research data presented on the mycotoxin content of cigarettes as well as data of an attempt to estimate the carry-over of mycotoxins to mainstream and side stream tobacco smoke.

3.) Conclusion of what is to be learned: Tobacco products are potential sources of mycotoxins - as are many other agricultural products. Tobacco stored in curing barns may become contaminated with aflatoxin and it is becoming recognized that aflatoxins should be monitored during the production of tobacco products. Recently, techniques have been developed to inhibit mycotoxin production in refined agricultural products, including tobacco. The talk examines the evidence for the carry-over of mycotoxins to mainstream and side stream smoke.

4.) References: Tuomi et al., 2001, Analyst, 126:1545-1550, El-Maghraby and Abdel_Sater, Zentralbl Mikrobiol, 1993, 148:253-64; Subbiah V., USPO, 1997, U.S. Patent 5,698,599; Georggiett et al., Rev Fac Cien Med Univ Nac Cordoba 2000, 57:95-107, Welty and Lucas, Appl Microbiol, 1969: 17:360-365

Mycotoxins in Cigarettes and in Tobacco Smoke

Tapani Tuomi

Cigarettes are natural products produced from the leaves of *Nicotina tabacum*. Before or after harvesting, tobacco leaves may become infected by common sapphrophytic and/or pathogenic fungi. Particularly fungi belonging to the following genus or genera, that have been established in different studies, on a variety of tobacco products, raise concern that tobacco products including cigarettes may contain mycotoxins: *Aspergillus flavus*, *Aspergillus fumigatus*, *Fusarium* spp. (*F. solani, F. moniliforme* and *F oxysporum*), *Stachybotrys chartarum*, *Trichoderma viride*[1,2,3]. As of present, the aflatoxins B_{1-2} and G_{1-2}, T-2 toxin and zearalenone have been found in cigarettes or in chewing tobacco[1,2,4]. Mycotoxins have not, however, been detected in either side-stream or mainstream tobacco smoke. In one study, aflatoxins artificially added to cigarettes were upon smoking detected in the cigarette filter but not in tobacco smoke[1].

During the smoking of cigarettes, gas-phase temperatures reach 850°C at the core of the firecone[5]. Solid phase temperatures reach 800°C at the core and 900 or greater at the char line[5]. Core temperatures are high enough to carbonize the tobacco and produce an oxygen deficient region where smoke constituents are formed through reductive processes. Temperatures behind the char line drop rapidly to the range 200-400 °C within 2 mm of the char line, enabling steam distillation of tobacco components[5]. Some 1200 chemical components, including nicotine, n-parafines and terpenes are distilled into mainstream smoke in the region behind the char line[5]. Side stream smoke is produced mainly between puffs when the firecone temperatures are 200-300°C lower than during puffs, and the smoke is convectively driven in the reverse direction of airflow accompanying a puff[5]. This results in quantitative and qualitative differences of individual components between mainstream and side stream smoke. Side stream smoke is richer than mainstream smoke in cancerogenous nitrosamines and in tar, as well as in nicotine[5]. Environmental tobacco smoke is composed mainly of diluted side stream smoke, which as a result of emission and distribution into ambient air differs from side stream smoke in particle size and mass -distribution, as well as in the distribution of compounds between particle and gas -phases[5].

As a result of these dynamic processes, resulting in the production of mainstream smoke inhaled by the smoker and environmental tobacco smoke inhaled both by the smoker and passive smokers, it is impossible to define the faith of mycotoxins potentially present in cigarettes without performing chemical analysis of side stream and mainstream smoke from native cigarettes and from cigarettes spiked with mycotoxins already present in native cigarettes. Such tests cannot, as of yet, be found in the literature. It is possible that aflatoxins or other toxins originally present in raw or stored tobacco are neutralized by ammoniation[6]. Different curing processes might also affect the distribution of mycotoxins potentially present in tobacco.

In conclusion, tobacco products are potential sources of mycotoxins - as are many other agricultural products. Tobacco stored in curing barns may become contaminated with aflatoxin produced by *Aspergillus flavus* as well as other mycotoxins, including fumitremorgins, produced by *Aspergillus fumigatus.* It is becoming recognized that mycotoxins should be monitored during the production of tobacco products. Recently, techniques have been developed to inhibit mycotoxin production in refined agricultural products, including tobacco. It has not been established as of yet, however, that mycotoxins are carried-over to mainstream and side stream smoke.

REFERENCES: [1]El-Maghraby and Abdel-Sater, Zentralbl Mikrobiol, 1993, 148: 253-64; [2]Warke et al., J. Food Prot., 1999, 62: 678-686; 3Verweij et al., JAMA, 2000, 284: 2875; [3]Welty and Lucas, App Micro, 1969, 17: 360-365; [4]Georgiett et al., Rev Fac Cien Med Univ Nac Cordoba, 2000, 57: 95-107; [5]Jenkins et al., Mainstream and side stream tobacco smoke, In *The chemistry of environmental tobacco smoke: composition and measurement*, M. Eisenberg, Ed, Lewis Publishers, Boca Raton, Florida, 2000, p. 49-75; [6]Subbiah V., USPO, 1997, U.S. Patent 5,698,599.

Abstract Information & Notes

Mohamed B. Abou-Donia, Ph.D.

Duke University Medical Center
Laboratory of Neurotoxicology
Dept. of Pharmacology and Cancer Biology
Box 3813
Durham, NC 27710

Phone: 919/684-2221
Fax: 919/681-8224
E-mail: donia@acpub.duke.edu

Date of talk: Friday, June 20, 2003, 2:00pm

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: **"Acute Exposure to Sarin Increases Blood Brain Barrier Permeability and Induces Neuropathological Changes in the Rat Brain: Dose Response Relationship"**

The speaker has provided the information below.

1.) Goals and objectives:

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:

4.) References:

ACUTE EXPOSURE TO SARIN INCREASES BLOOD BRAIN BARRIER PERMEABILITY AND INDUCES NEUROPATHOLOGICAL CHANGES IN THE RAT BRAIN: DOSE RESPONSE RELATIONSHIP.

MB Abou-Donia[1], AA Abdel-Rahman[1], and AK Shetty[2,3]. Departments of [1]Pharmacology and Cancer Biology, [2]Neurobiology, and [3]Surgery (Neurosurgery), Duke University Medical Center, Durham NC 27710

In this study, we evaluated the early changes in the adult rat brain after a single exposure to different doses of sarin. Adult male rats were exposed to sarin by a single intramuscular injection at a doses of 1, 0.5, 0.1, and 0.01 x LD50 corresponding to 100, 50, 10 and 1 μg sarin /kg body weight of animals. Twenty-four hours after this treatment, both sarin-treated and vehicle-treated (controls) animals were processed for: (i) plasma butyrylcholinesterase (BChE) activity; (ii) brain acetylcholinesterase (AChE) and m2 muscarinic acetylcholine receptor (m2 mAChR) ligand binding assays; (iii) analysis of blood brain barrier (BBB) permeability using [H^3] hexamethonium iodide uptake assay and immunohistochemistry for BBB protein; and (iv) histopathological analyses of the brain using H&E staining, and microtubule-associated protein (MAP-2) and glial fibrillary acidic protein (GFAP) immunohistochemistry. Animals treated with 1 and 0.5 x LD50 sarin exhibited a significant decrease in plasma BChE (58-70% of control). Animals treated with 1 x LD50 also exhibited a significant inhibition of AChE in the cerebrum, brainstem, midbrain, and the cerebellum (54-69% of control) associated with a decrease in m2 mAchR ligand binding in the cerebrum (39% of control). m2 mAChR ligand binding however, showed a significant increase in the brainstem (250-300% of control) following exposure to all doses of sarin. Analysis of [H^3] hexamethonium iodide uptake demonstrated a significant increase in BBB permeability in animals treated with 1xLD50 sarin. Where as, animals treated with 0.5XLD50 exhibited significant increased in BBB permeability only in brainstem and midbrain. Quantitative histopathological analysis in the latter group of animals also revealed a significant decrease in the expression of BBB protein, a diffuse neuronal cell death and a decrease in MAP-2 positive elements within the cerebral cortex and the hippocampus, and degeneration of Purkinje cells in the cerebellum. While animals treated with 0.5 and 0.1 x LD50 did not exhibit the above changes, animals treated with 0.5 x LD50 showed Purkinje neuron loss in the cerebellum. Results of this study indicate that, the early brain damage after acute exposure to sarin is dose-dependent, and that exposure to 1 x LD50 sarin induces significant damage in many regions of the adult rat brain by 24 hours after exposure. The early neuropathological changes observed after a single dose of 1 x LD50 sarin could lead to a profound long-term neuronal degeneration in many regions of the brain, and resulting behavioral abnormalities. Supported, in part, by the U.S. Army Medical Research and Materiel Command Contract: DAMD 17-98-C-8027.

Abstract Information & Notes

Sherry A. Rogers, M.D.

Northeast Center for Environmental Medicine
2800 W. Genesee St.
Syracuse, NY 13219

Phone: 315/488-2856
Fax: 315/488-7518
E-mail: N/A

Date of talk: Friday, June 20, 2003, 2:30pm

Medical School Attended: S.U.N.Y Health Sciences Center at Syracuse formerly Upstate Medical Center, State University of New York

Major and date of Graduation: 1969

Board Certifications: (1) Family Practice, (2) Environmental Medicine Fellowship American College Nutrition, Fellowship American College Asthma, Allergy & Immunology

Current Job Description: Clinician 33 years, lecturer, author 14 books monthly referenced newsletter 15 years

Other Information: Latest book: Detoxify or Die, with over 700 references showing reversibility of nearly all disease (Prestigepublishing.com or detoxifyordie.com)

Disclosure Statement: None

SPEECH TITLE: **"Rescuing the Heart as Toxic Target Organ"**

The speaker has provided the information below.

1.) Goals and objectives: Learn of the evidence that environmental chemicals are unavoidably ubiquitous, bioaccumulate, and cause disease which can be reversed.

2.) Outline of talk/abstract: The heart slowly bioaccumulate ubiquitous environmental toxins and nutrient deficiencies which cause all symptoms. Reversing even the most severe heart disease is possible when molecular biochemical principles are appreciated, versus masking symptoms with drugs.

3.) Conclusion of what is to be learned: No cardiovascular problem is hopeless until the true causes have been found.

4.) References: Rea, WJ, Chemical Sensitivity, Vol. I-IV, 1992-97, CRC Press, Boca Raton
Bralla, JA, Lord RS, Laboratory Evaluations, Molecular Medicine, Meta Metrix, Norcross, GA, 2002
Beasley, VR, Trichothecane Mycotoxicosis, CRC Press, Boca Raton, 1989

Rescuing the heart as toxic organ: Reversing the damage from mycotoxins, Solanaceae, heavy metals, pesticides, phthalates, styrene, PCBs, hydrocarbons and other xenobiotics

Sherry A. Rogers M.D., ABFP, ABEM, FACAAI, FACN
prestigepublishing.com

Objectives: to appreciate the enormous amount of evidence showing that environmental toxins are

- **ubiquitous and unavoidable**
- **bioaccumulate**
- **cause nearly every symptom** and disease imaginable
- Using the heart as an example of one target organ, we will show how medical science has now proven that removal of toxins from the body **allows healing**, leaving the prognostic value of the diagnosis or medical label meaningless

Abstract:

The heart and cardiovascular system have a finite number of recognizable symptoms to express imbalance and disease. Cardiac arrhythmias, angina, infarct, congestive heart failure, mycarditis, hypertension, hypercoagulability, hypercholesterolemia and vasculitis are among the most common. Scientists agree 95% of cancer is caused by only two things: diet and environment. And the same formula turns out to be true for a multitude of diseases especially those of the cardiovascular system.

With this in mind, a logical first step would be to identify nutrient deficiencies as well as toxic accumulations, and then proceed to correct these. As an example, multiple nutrient deficiencies are commonly observed to by the underlying defects causing cardiac arrhythmia. Instead it is treated as a deficiency of calcium channel blockers. This robs the patient of the opportunity to identify the extremely common fatty acid and mineral deficiencies that lead to damaged calcium channels. Added to the nutritional deficiencies are commonly PCBs and mercury, as simple examples of ubiqutously unavoidable xenobiotics, that also damage proper function of calcium channels. Clearly the patient has only one course to follow with medications, and that is to slowly get worse, because no one has identified and fixed what is broken. Meanwhile calcium channel blockers cause MRI-proven shrinking and deterioration of the brain and cognition.

On the other hand, damaged calcium channels have been repaired, improving drug-dependent/resistant cardiac symptoms while reducing medications. Clearly we are proceeding in the wrong direction by seeing all heart disease as a deficiency of multiple drugs, and scientific evidence will be presented for the rationale for exchanging this approach for that of evidence-based molecular medicine.

Conclusions: regardless of the medical label applied to any malfunction in the heart, there are usually identifiable nutrient deficiencies and toxic overloads which when corrected, allow for reversal of disease with reduction of symptoms and medications.

References:

Rea, WJ, *Chemical Sensitivity, Volumes I-IV*, CRC Press, Boca Raton FL, or www.aehf.com, 1992-97

Bralley JA, Lord RS, *Laboratory Evaluations in Molecular Medicine*, Institute for Advanced Molecular Medicine, 1-800-221-4640, Norcross GA, 2002

Beasley VR, *Trichothecene Mycotoxicosis: Pathophysiologic Effects, Volume II*, CRC Press, Boca Raton FL, 1989

AACME rules prohibit listing a source of over 700 references for this presentation

Abstract Information & Notes

Bruce Jarvis, Ph.D.

Department of Chemistry & Biochemistry
University of Maryland
College Park, MD 20742

Phone: 301/405-1843
Fax: 301/314-9121
E-mail: bj6@umail.umd.edu

Date of talk: Friday, June 20, 2003, 3:30pm

Education: B.A., Ohio Wesleyan University, Delaware, Ohio, 1963; Ph.D. University of Colorado, Boulder, Colorado, 1966; Postdoctoral Research Associate, Northwester University, Evanston, Illinois, 1966-67

Other: Instructor of Chemistry (Part-time), Northwestern University, Evanston, Illinois, 1966; Assistant Professor, University of Maryland, 1967-71; Associate Professor, University of Maryland, 1971-79; Visiting Scholar in Residence, University of Virginia, 1975-76; Professor of Chemistry, University of Maryland, 1979-Present; Program Officer, Organic Synthesis, NSF 1987-88; Associate Chair 1988-89, 1992-1993, 1998-1999; Acting Chair 1989-90; Chair, Department of Chemistry and Biochemistry, 1993-1998; Visiting Professor, Dept. of Biotechnology, Danish Technical Univ., 1999-2000.

Disclosure Statement: None

SPEECH TITLE: **"History and Toxicology of Mycotoxicoses"**

The speaker has provided the information below.

1.) Goals and objectives: Present history and overview of animal and human toxicoses associated with exposure to fungal toxins

2.) Outline of talk/abstract: Animals and humans have a long history of exposure, mainly through ingestion, to toxins produced by filamentous fungi, e. g. *Aspergillus, Fusarium, Penicillium, Alternaria*, etc. The mode of exposure is most commonly through contamination of food and feed, and symptoms range from mild discomfort to death. Many fungal toxins (mycotoxins) are immunosuppressants; some are among the most potent carcinogens known.
Although most of our experience with exposure to mycotoxins has been gained in an agricultural setting, recent concern has been centered on inhalation exposure to mold spores generated in damp buildings and homes. The problems associated with extrapolating data from the agricultural settings to those found in buildings will be discussed.

3.) Conclusion of what is to be learned: Exposure to mycotoxins is not a new problem, but it has many, many variables that often make it difficult to relate symptoms in individuals to exposure to specific toxigenic fungi. There are certainly many notorious cases of mycotoxicoses (mainly in animals, but some in humans) where this linkage between exposure to mycotoxins and adverse health outcome (e. g. death) is clear. However, such cases in indoor environments, where exposure is through relatively low levels of toxigenic fungi, are very often problematic, and the case for cause and effect much more difficult to establish.

4.) References: 1. B. B. Jarvis, "Chemistry and Toxicology of Molds Isolated from Water-damaged Buildings," in Mycotoxins and Food Safety: *Adv. Exp. Med. Biol.* Vol 504: 42-53. J. W. DeVries, M. Trucksess, and L. Jackson, eds., Kluwer Academic/Plenum (2002).

Abstract Information & Notes

David C. Straus, Ph.D.

Texas Tech University
Department of Microbiology
Texas Tech University Health Sciences Center
Lubbock, TX 79430

Phone: 806/743-2523
Fax: 806/743-2334
E-mail: david.straus@hmc.huhsc.edu

Date of talk: Friday, June 20, 2003, 4:00pm

Graduate School Attended: Loyola University of Chicago
Major and date of Graduation: Microbiology - May 1974
Current Faculty Appointments: Professor of Microbiology & Immunology
Current Job Description: Teaching and Research
Other Information: Member Sigma Xi, American Society for Microbiology, American Academy of Microbiology

Disclosure Statement: Assured IAQ7 of Dallas, TX

SPEECH TITLE: **"Recent Research in Sick Building Syndrome"**

The speaker has provided the information below.

1.) Goals and objectives: To acquaint the audience in recent research examining the role of fungi in sick building syndrome

2.) Outline of talk/abstract: Recent research on this topic will be presented.

3.) Conclusion of what is to be learned: That fungi growing inside building is what is primarily responsible for the phenomenon known as sick building syndrome.

4.) References: Cooley et al. 1998. Occupat. Environ. Med. 55:579-584. McGrath et al. 1999. Curr. Microbiol. 38:33-36. Cooley et al. 2000. Thorax 55:489-496. Karunasena et al. 3001. Mycopathologica 150:91-95.

Abstract Information & Notes

William J. Rea, M.D.

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

Phone: 214/368-4132
Fax: 214/691-8432
E-mail: wjr@ehcd.com

Date of talk: Friday, June 20, 2003, 4:30pm

Medical School Attended:	Ohio State University College of Medicine
Major and date of Graduation:	M.D., 1962
Residency:	UTSWS
Board Certifications:	American Board of Surgery, American Board of Thoracic Surgery, American Board of Environmental Medicine
Current Faculty Appointments:	Professor of Medicine, Capital University of Integrative Medicine, Washington, DC
Current Job Description:	President, Environmental Health Center - Dallas
Disclosure Statement:	None

SPEECH TITLE: **"Treatment of Mold and Mycotoxin Exposure"**

The speaker has provided the information below.

1.) Goals and objectives: To tech the clinician how to treat mold and mycotxin exposure.

2.) Outline of talk/abstract: Avoidance of molds and toxic chemicals; injection therapy; nutrition; heat therapy; and massage; immune modalities

3.) Conclusion of what is to be learned: How to treat mycotoxins and mold exposure.

4.) References: Chemical Sensitivity, Volume IV

21st ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT

SCHEDULE

Saturday, June 21, 2003

7:45 a.m. **ANNOUNCEMENTS/MODERATOR: Kalpana Patel, M.D.**

8:00 **Chris Rea,** Environmental Consultant, R.H. of Texas, Dallas, TX: "**The Identification and Remediation of Mold and Mycotoxin Problems in a House"**

8:20 Q & A

8:30 **Wallace Rubin, M.D.,** Private Practice, Metaire, LA: "**Mold and Chemical Sensitivity Related Inner Ear Disease"**

8:50 Q& A

9:00 **Donald P. Dennis, M.D., F.A.C.S.**, Atlanta, GA, **"Treatment of Allergic Fungal Sinusitis by Decreasing the Environmental Air Fungal Load and Anti-Microbial Nasal Sprays Based on 14 Years Clinical Observation in 639 Patients"**

9:20 Q & A

9:30 **Geoffrey Hutton,** Architect, Hutton & Rostron Environmental Investigations, Guildford, Surrey, England: "**The Way We Build Now"**

9:50 Q & A

10:00 **BREAK WITH EXHIBITORS**

10:30 **William J. Meggs, M.D.,** Professor of Toxicology, Department of Emergency Medicine, E. Carolina University School of Medicine, Greenville, NC: **"The Need for a National Environmental Medical Unit"**

10:50 Q & A

11:00 **Allan D. Lieberman, M.D.,** Medical Director, Center for Occupational Environmental Medicine, North Charleston, SC: **"Explosion of Mold Cases in Homes, Work Places and in Occupational Medical Practices"**

11:20 Q & A

11:30 **Nancy A. Didriksen, Ph.D.,** Environmental Health Psychologists, Richardson, TX: "**Neurocognitive Deficits in Individuals Exposed to Toxigenic Molds"**

11:50 Q & A

12:00n **BUFFET LUNCH WITH THE EXHIBITORS**

MODERATOR: William J. Meggs, M.D.

1:30 **Jean A. Monro, M.D.,** Medical Director, Breakspear Hospital, Hertsfordshire, England: **"Treatment of Cancer with Mushroom Products"**

1:50 Q & A

2:00 **Larry Foster**, Environmental Consultant, Enviro-Cure, Atlanta, GA**: "General Reasons and Causes of Sick Buildings that are a Result of Airborne Biohazards"**

2:20 Q & A

2:30 **Theodore R. Simon, M.D.,** Private Nuclear Medicine Practice, Nuclear Medicine, Dallas, TX: "**Neurotoxicity: Mold Exposure Versus All Causes"**

2:50 Q & A

3:00 **BREAK WITH EXHIBITORS**

3:30 **Donald P. Dennis, M.D., F.A.C.S.,** Atlanta, GA: **"Guidelines and Theory for Treatment of Chronic Fungal Sinusitis with Reduction of Environmental Air Mold Loan and Anti-microbial Nasal Sprays based on 14 Years Clinical Observation in 639 Patients"**

3:50 Q & A

4:00 **Larry Foster,** Environmental Consultant, Enviro-Cure, Atlanta, GA**: "Toxic Molds: The XXI Century Mold Rush"**

4:20 Q & A

4:30 **CASE STUDIES**: Richard Jaeckle, Ph.D., Andrew Campbell, M.D., Donald P. Dennis, M.D., William J. Rea, M.D., John H. Boyles, Jr., M.D., Kaye H. Kilburn, M.D.

6:00 **AJOURN**

SATURDAY, JUNE 21, 2003

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

Chris Rea

P.O. Box 780392
Dallas, TX 75378

Date of talk: Saturday, June 21, 2003, 8:00am

Phone: 214/351-6681
Fax: 214/358-2177
E-mail: N/A

Other Information: In business for 18 years. Examined over 1,000 houses and buildings for problems that relate to or cause environmental illness. This includes construction and renovations flaws, mold and bacteria problems, and chemical and pesticide problems. Completed several hundred renovation and construction projects, all done to environmentally safe standards. Worked on projects all over the United States, Mexico, Canada and Germany.

Disclosure Statement:

SPEECH TITLE: **"The Identification and Remediation of Mold and Mycotoxin Problems in a House"**

The speaker has provided the information below.

1.) Goals and objectives:

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:

4.) References:

The Identification and Remediation of Mold and Mycotoxin Problems in a House

The problem of mold and fungus growth in homes and buildings has received a great amount of press over the last 3 years. The majority of stories have centered around lawsuits against insurance companies, builders, landlords and sometimes municipal authorities. Less often, but probably more significant for the health care professional, are the sensationalized stories about the health effects that some individuals have suffered as a result of living in a moldy home. However, there have been relatively few stories about the best way to determine if a house is moldy, why it is moldy, and if it does have a problem with mold growth, how this should be corrected.

Fungal growth and the resulting mycotoxins contamination that goes along with it can cause many different types of health problems. This is well documented and will be discussed by other speakers in this conference. Further, the specific problem of fungal contamination in a home is a very old problem that has probably existed since humans began dwelling indoors.

When people started living indoors, and in particular began to construct closed in homes they inevitably set up the conditions for fungal growth. The factors that cause a home to be moldy are varied and they can change drastically depending on the climate in the area where a home is constructed, they type of construction, materials used, building techniques employed, etc. Knowledge of these variables is crucial in diagnosing why a house has developed a mold problem. This answer to this question is THE MOST IMPORTANT piece of information necessary in determining how to clean up a moldy house.

This talk will present a basic overview, with a case study, in the techniques used to diagnose fungal problems in a house. The factors and variables that cause fungal growth in homes will also be discussed. Finally, some of the techniques used to solve the problems and clean up the house will also be used.

Abstract Information & Notes

Wallace Rubin, M.D.

3434 Houma Boulevard, Suite 201
Metairie, LA 70006

Phone: 504/888-8800
Fax: 504/455-6796
E-mail: wrubinmd@bellsouth.net

Date of talk: Saturday, June 21, 2003, 8:30am

Medical School Attended: University of Illinois College of Medicine
Major and date of Graduation: 1946
Residency: Tulane University, Department of Otolaryngology 1949-1951

Board Certifications: American Board of Otolaryngology 1953
Current Faculty Appointments: Clinical Professor of Otolaryngology - LSU
Current Job Description: Solo Practitioner

Disclosure Statement: None

SPEECH TITLE: **"Mold, Chemical Sensitivity Related Inner Ear Disease"**

The speaker has provided the information below.

1.) Goals and objectives: The mold and chemical sensitivity immunologic mechanisms as they relate to inner ear disease.

2.) Outline of talk/abstract: Patient presentations that will objectively document these causative mechanisms as they relate to the diagnosis and treatment of inner ear disease.

3.) Conclusion of what is to be learned: The methodologies for diagnostic documentation of the causative mechanisms and the approaches to etiologic treatment will be specifically presented.

4.) References:

8. 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. Neurol Clin 1990; 8:225-234.
9. 2. Rubin, W: Site of lesion vestibular function testing. Laryngoscope 1985; 95:386-390
10. 3. Rubin, w: Biochemical evaluation of the patient with dizziness. Semin Heal 1989; 10:151-159.

Mold and Chemical Sensitivity Related Inner Ear Disease

Wallace Rubin, M.D.

Metairie, LA

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 than logically culminate in a systematic etiological investigation. I will present a patient example of this type of difficulty with mold and chemical sensitivity and show how the proper evaluation and treatment are significant.

The Questions to be answered by the neurotological evaluation are:

1. What neurotological tests can be used for site of lesion confirmation?

2. Which biochemical, immunologic, metabolic, and hormonal tests are indicated?

3. What modalities of therapy can then be efficacious?

References

Brookes GB. Circulating immune complexes in Meniere's disease.
Arch Otolaryngol 1986; 112:536-40.

Clemis JD. Allergy of the inner ear.
Ann Allergy 1967; 25:370-6.

Clemis JD. Allergic cochleovestibular disturbances.
Tans AM Acad Opthalmol Otol 1972; 76:59-65

Clemis JD. Cochleovestibular disorders and allergy.
Otol Clin North AM 1974; 7:757-80.

Criep HL. Allergic vertigo.
Penn Med J 1939; 43:258-282

Derebery MJ, Rao V, Siglock TH, et al. Meniere's disease. An immune complex mediated illness?
Laryngoscope 1991; 101:225-229

Derebery MJ, Valenzuela S. Meniere's Syndrome and Allergy.
Otolaryngologic Clinics of North America 1992;Vol 25#1:213-23.

Duke WW. Meniere's syndrome caused by allergy.
JAMA 1923; 81:2179-81

Endicott JN, Stucker FJ. Allergy in Meniere's disease related fluctuating hearing loss primarily findings in a double blind cross over clinical study.
Laryngoscope 1971; 87:1650.

Futaki T, Yamane M, Shirahata A. Immunologic analysis of IgG and other protein fractions in endolymph obtained from endolymphatic sac of Meniere patients and a control.
Acta Otolaryngol 1985; 4191:71-8.

Harris JP. Immunology of the inner ear: response of the inner ear to antigen challenge.
Otolaryngol Head Neck Sug 1983; 91:18-23

Powers WH. Allergic factors in Meniere's disease.
Trans Am Acad Ophtalmol Otol 1973; 77:22-9.

Stahle J, Deuschl H, Johansson SG. Meniere's disease and allergy, with special reference to immunoglobulin E and IgE (regain) antibody in serum.
Int J.Equil Res 1974; 4:22-27.

Williams HL. Allergy of the inner ear – Meniere's disease.
Arch Otol 1952; 16:24-44.

Abstract Information & Notes

Donald P. Dennis, M.D., F.A.C.S.

ENT & Facial Plastic Surgery, L.L.C.
3193 Howell Mill Rd., Suite 215
Atlanta, GA 30327

Phone: 404/355-1312
Fax: 404/352-2798
E-mail: ddennis@mindspring.com

Date of talk: Saturday, June 21, 2003, 9:00am

Medical School Attended:	Medical College of Georgia, Augusta, Georgia
Major and date of Graduation:	MD, 1974
Residency:	Otolaryngology - Head & Neck Surgery, John's Hopkins Hospital
Board Certifications:	American Board of Otolaryngology & Head & Neck Surgery
Current Faculty Appointments:	ENT Department, Northside Hospital
Current Job Description:	Private Practice, Atlanta
Disclosure Statement:	None

SPEECH TITLE: **"Treatment of Allergic Fungal Sinusitis by Decreasing the Environmental Air Fungal Load and Anti-Microbial Nasal Sprays Based on 14 Years Clinical Observation in 639Patients"**

The speaker has provided the information below.

1.) Goals and objectives:

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:

4.) References:

Treatment of Allergic Fungal Sinusitis by Decreasing the Environmental Air Fungal Load and Anti-Microbial Nasal Sprays Based on 14 Years Clinical Observation in 639 Patients

By
Donald P. Dennis, M.D., F.A.C.S.
Atlanta, Georgia

Treatment Strategy:

In September 1999 Mayo Clinic published an article that pegs 93% of all chronic sinusitis as being caused by mold. The most likely mechanism of formation of sinusitis is, a Type 2 Gell Coombs reaction. As mold is breathed into the nasal mucosa intact eosinophylls migrate through the mucosa and bind to IgG antibodies in the nasal mucus. The mold antibody complex binds to the mold and the eosinophyll lyses, releasing major basic protein, which pits the mucosa. These pits trap mucous so that the mucous cannot drain. The stagnant mucous gets infected which causes nasal polyps and thickening of the lining, which obstructs the outflow of mucous. The polyps cause more infection and the infection causes more polyps and then there is a viscous cycle, which perpetuates itself. This reaction is most likely a type 2 hypersensitivity reaction. All type 2-hypersensitivity reactions stop when the antigen (mold in this case) is removed. We knew from 20 years of clinical experience that when patients cleaned their environmental air their sinusitis improved. The air mold level required for health was discovered by testing the one hour gravity plate exposure inside each chronic sinusitis patient=s home and following them by endoscopic photographs as remediation of mold was done. Over 600 patients homes were tested for mold before and after mold remediation. It was discovered that a mold count of 0-4 colonies with a one-hour gravity plate exposure was required for the sinus mucosa to clear by endoscopic photography. Normal saline nasal irrigation with a combination of antifungal-antibacterial-anti-inflammatory nasal sprays were found to markedly improve mucosal recovery. A ASystemic Fungal Syndrome@ was observed in some patients consisting of memory loss, hearing loss, dizziness, arthritis, fibromyalgia, dermatitis, ataxia, muscle weakness, disorientation, and GI disturbance. These patients likely hypersensitivity mechanism was a Gell-Coombs Type 3 and 4 reactions. Many different ways of mold remediation and nasal sprays were tested and a protocol was developed that was easy for the patient to implement and cost effective. An effective environmental and nasal treatment protocol was developed. The environmental treatment consists of a HEPA air filter combined either with either botanical oil evaporation, or non-ozone ionization, with vent covers sprayed with botanical extract. Whole house fogging was also developed to bring the colony counts down to zero in 1 hour.

Attached are 3 photos.

Fig. 1 is an endoscopic photo showing the mold count in the patient=s room air before and after room air mold remediation with the endoscopic photographs showing the purulent infection clearing after the mold count drops to 4 colonies.

Fig. 2 shows the colony count dropping from TNTC to 0 in six days using the Wein 2500 room unit.

Fig. 3 shows the mold colony count going from TNTC to 0 in 1 hr. and 1 colony in 2 hrs.

Note

EPA "Introduction to Indoor air Quality: A Reference Manual" uses 50 CFU/m3 as a beginning concern and 10,000 as a problem amount using an Anderson sampler. The equivalents of Gravity feed to Anderson Sampler for 3 minutes @ 28.3 L/min are:

CFU Comparison

Gravity Fed	Anderson Sampler Range	Anderson avg.
1	40-400	345
2	1100-1200	1198
3	2100-2500	2401
4	3400-3500	3496
5	4800-5000	4913
6	6200-6300	6263
7	7500-7600	7531
8	8700-8900	8745
9	9900-10,100	9978
10	11,000-11,300	11,254
11	12,500-12,600	12,513
12	13,700-14,000	13,749

Therefore in reality the value for mold safe air using an Anderson sampler is 3400-3500 cfu (colony forming units).

Abstract Information & Notes

Geoffrey Hutton

Hutton & Rostron Environmental Investigations
Gomshall, Guildford
Surrey, UK GU5-9QA

Date of talk: Saturday, June 21, 2003, 9:30am

Phone: 011/44-1483-203221
Fax: 011/44-1483-202911
E-mail: ei@handr.co.uk

Current Job Description: Partner in private, specialist architectural practice and Chairman of a firm of environmental; investigators

Other Information: Hutton & Rostron (H&R) is a firm of architects, health and legal professionals with scientific support specializing in building pathology. Investigation of building defects and loss prevention is an important part of its activities. Although maintenance is often reactive, a proactive policy is preferred and for this reason reliable and time-related data is acquired for diagnosis and warning

Disclosure Statement: None

SPEECH TITLE: **"The Way We Build Now"**

The speaker has provided the information below.

1.) Goals and objectives: To introduce the delegates to the nature of building construction, the materials and the consequences for the environment and health.

2.) Outline of talk/abstract: The background of construction as a fundamental activity to fill the many needs for shelter and working facilities, and the impact these have on the environment, disease and perceptions of health, fashion and the technology involved.

3.) Conclusion of what is to be learned: Intricacy of construction and engineering services may no necessarily be beneficial and the increasing use of products depending on volatile components for manufacturer or maintenance is a hazard.

4.) References: None

THE WAY WE BUILD NOW

Geoffrey H Hutton, ARIBA, DipArch (Dist)

Building design is a multi-factorial equation of thousands of unknowns and few knowns with interactions with health at every level, this has been resolved in the evolution of vernacular building, but not so successfully in conceptually based design

Birds are great builders. They exploit local resources in competition with others while optimizing the loads and traveling distance. They have to get the location and structure right; too high in the tree and the wind will destroy the nest and too low and a predator will get your eggs. Too strong or elaborate construction will take longer and waste otherwise valuable breeding or feeding time. The nest must be defensible, inconspicuous, protect the eggs from breakage, not harbor pests or support fungal growth, must retain sufficient heat for insulation, be well drained and ventilated. The initial owner may decide to clear out after a year and build a new hygienic model next season or invest in a family home to be refurbished each year or even to be a squatter. Every climate and context has residents and tourists who have solved their accommodation problems sufficiently well to have survived and prospered over many generations with no insurance. We have much to learn from the blind watchmaker

Man too has solved the problems of climate and context in the building methods and materials available to him either as a nomad or in a settled community with as much convenience and defense that could be afforded. The possible solutions, if not the result of self-help, were understood by the users and the technology was within the grasp of the individuals concerned and shared within the community. Differentiation between building functions was slight, the essentials of shelter and enclosure could be met by common methods for providing spanning, load bearing and enclosing structures relying on the inherent properties of the materials for weather resistance, load bearing and insulation or thermal capacity. The interior was modified by the combustion of the limited fuel available, which was of primary importance for cooking. Such buildings continued to serve satisfactorily for millennia and can still be found throughout the world providing the ultimate barrier to the environment after the primary barrier provided by clothing for the climate and season. The technology evolved incorporates the accumulated experience acquired for practical survival and prosperity in the conditions experienced over many generations. Such empirical knowledge includes such issues as ventilation, temperature and evaporation from surfaces with an elegance, which may not be perceived, and thus overlooked in analysis

Building from the earliest times has, of course, also served symbolic purposes demonstrating power, identity or spiritual values. These monumental characteristics are usually achieved either at some otherwise unjustified expense and inconvenience to the practical performance of the building, or they are ignored altogether

Unfortunately, the empirical has been overlaid by the monumental which is further complicated by the introduction of codes and standards which try to capture the multi-factorial performance of buildings by setting out criteria for aspects of structural strength, fire resistance, thermal characteristics, ventilation, lighting etc further defined by methods of test limited by the laboratory methods available. Such aspects of performance are inherent in the overall construction by evolutionary optimization. Important to both birds and man is the health of the occupants as fundamental to survival, whereas this is not of great significance in monumental buildings (which may well be tombs)

The gradual incorporation of imagery and construction from monumental building into the mainstream of the vernacular has led to the dominance of conceptual design over the utilitarian product of evolution and the local environment. Professions developed to match, each with a visual or engineering idea to promote, but without an overarching experience of the interactions between the diverse needs and performance required. In turn, they served building owners and users with less personal appreciation of the issues

Much of the performance required is now specific to particular components, which, in turn, are used in assemblies and bought as a catalogue items to achieve theoretically defined objectives. The performance of items may be known from tests, but the consequences for the whole assembly, and particularly at the interfaces, may not be fully understood, defined or even measurable. Further, the overall design may not be repeated or the result of

experience acquired over many projects. Consequently, failures occur due to unanticipated effects or events, at changes of material or section, or in voids concealed from inspection

Historically, buildings were either heavyweight, usually masonry, or assembled from frames and infilling panels normally of timber. Each has advantages for particular climates and occupancies. The masonry building will have a smaller exposed surface area and a higher thermal capacity than the frame building, which will also contain more cavities. Current building is generally of frame and fill construction, even if faced with masonry, as modern construction tries to limit the activities on site. Typically, such buildings have suspended ceilings, raised floors and paneled or cavity wall constructions creating voids which are not easily accessible and can be contaminated and difficult to clean. Builders' dirt and votive offerings are frequently found in such places, which also provide the accommodation for engineering plant and distribution services, pipes and ductwork

Since the early 19th century, the exploitation of fossil fuels has been the cheap and, for the time being, pervasive source of thermal process energy in the manufacture of products such as glass, metals, cement and bricks; and power for cutting, forming, handling and distribution of products. Fossil fuels now provide raw materials for plastics, fibers, dyes, paints and adhesives used in construction, building services, furniture, soft furnishing, household utensils and clothing, and a host of treatments in the form of cleaning agents, polishes, insecticides, and life-style support generally. The processes involved generate persistent wastes, and the products can have undesirable emissions and result in toxic waste. Most of all, fossil fuel has been used to power environmental services in buildings for heating, ventilation and air-conditioning without which (and lifts and escalators) much of the world would be virtually uninhabitable at the present densities. The management of such services is not precise and the fuel is still cheap and generally available. Consequently, there is little incentive to economize, thus environmental services consume some 60 per cent of all fuel. In historic terms, this relatively short-term cost-free resource has changed lifestyles to reduce the thermal importance of clothing, increase the size of individual spaces, reduce the need for natural light or ventilation, and medicated our surroundings. The availability of hot water, for the dispersal of activities, such as bathing, laundry and cooking results in higher levels of humidity. The engineering required to provide for such conditions, over that possible by the passive qualities of the building enclosure, becomes increasingly complex while occupying a large proportion of the available space and requires regular and diligent maintenance to achieve its theoretical performance

The environmental standards achievable by the use of engineering services inside buildings, particularly if sealed, subjects the external envelope to greater stresses in providing what has come to be regarded as the primary barrier to external conditions. It must now accommodate greater thermal movement, vapor pressure and rates of heat transfer, as well as continuing to offer weather resistance, and in some conditions condensation will occur within the structure

The nature of frame and fill buildings favors the use of space heating, rather than structural heating and high thermal capacity which results in slow adjustment to ambient conditions and can normally be expected to give comfort with lower air temperatures and thus less likely to suffer from condensation. Space heating (or cooling) depends on the processing of large volumes of air, much of which is re-circulated to economize in fuel. Such air carries the waste products of respiration and activities in the space, which must be processed, filtered and mixed with 'fresh' air for re-use. Such a system is liable to contamination and condensation at a number of points even if the filters are routinely changed and well maintained. Biological material will form part of the contaminants and with the appropriate humidity fungal growth can be anticipated

Depending on building use and standards of housekeeping, the various unobserved voids may become home to insects and other pests, which may be encouraged by the ambient conditions. They may be sustained by human litter and food droppings, and initiate further contamination and damage to structure and furnishings. Particular activities such as sleeping and cooking bring particular infestations such as house mites and cockroaches with consequences for health. Major destruction can result from fungi such as the wet and dry rots, and wood-boring insects all of which are indicators of poor environments and housekeeping

Very heavily serviced buildings such as hospitals could become hygienic liabilities; the continuing cost of maintenance and the difficulty of decontamination may make simpler disposable accommodation viable. Ultimately, the properties will be removed for reasons of fashion or return on investment and it seems reasonable that this should be foreseen as part of the brief. Contamination and hazards to health should be factors in this

decision

Buildings of historic, technical, literary or national interest should be preserved. This helps understanding of how people lived and adapted to the climate and context in which they lived in the thousands of years before the profligate use of energy and how we may have to live again. These buildings often suffer from fungal decay and insect attack due to neglect, misuse, the introduction of modern heating, new materials and lifestyles; but rarely due to the work of the original builders. The fungal and insect infestations can almost invariably be dealt with by reversing the conditions causing decay and ensuring ventilation in future

Health must become of central concern in the equation

END

Hutton + Rostron Environmental Investigations Limited
Netley House, Gomshall, Surrey GU5 9QA
Tel 01483 203221 Fax 01483 202911 Email ei@handr.co.uk Web: www.handr.co.uk

Abstract Information & Notes

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

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

Phone: 252/744-2954
Fax: 252/744-3589
E-mail: meggsw@mail.ecu.edu

Date of talk: Saturday, June 21, 2003, 10:30am

Medical School Attended:	University of Miami
Major and date of Graduation:	M.D., 1979
Residency:	University of Rochester
Board Certifications:	Medical Toxicology, Allergy & Immunology, Internal Medicine, Emergency Medicine
Current Faculty Appointments:	Professor & Chief of Toxicology
Current Job Description:	Physician
Other Information:	Author of "The Inflammation Cure" to be published in Sept. 2003. Editor of "Health & Safety in Agriculture, Forestry, & Fisheries." Author of numerous research articles and textbook chapters.
Disclosure Statement:	None

SPEECH TITLE: "**The Need for a National Environmental Medical Unit"**

The speaker has provided the information below.

1.) Goals and objectives:
$ To know what an environmental control unit [environmental medical unit] is.
$ To know the history of Environmental Medical Units in this country and abroad.
$ To know the clinical and research uses of Environmental Medical Units.
$ To know the need for a National Environmental Medical Units for research studies.

2.) Outline of talk/abstract: The concept of an Environmental Control Unit [Environmental Medical Unit] was developed by Dr. Theron Randolph as a clinical tool to evaluate environmental factors such as air pollutants, natural and additive dietary elements, and water contaminants on specific disease processes. From 1966 to 1974,a number of EMUs were opened in this country and for the evaluation of environmental factors contributing to specific disease processes in individual patients. Though tens of thousands of patients were treated in these units with a high degree of patient and physician satisfaction, these units were abandoned due to economic and political factors, the changing healthcare environment, and the refusal of third party carriers to pay for this service. Environmental Medical Units are an important research tool to evaluation environmental factors in a number of syndromes and diseases. The Japanese have taken a leadership role in establishing EMUs for medical research, in particular in evaluating effects of sick building gases and volatile organic chemicals on cerebral function. The case for a national EMU in the United States is presented, and the use of such a unit to study environmental factors in specific diseases is discussed.

3.) Conclusion of what is to be learned: Environmental Medical Units have been very valuable as a clinical tool and will play a prominent role in environmental medicine research in the future. The United States should build a National Environmental Control Unit devoted to research in environmental medicine.

4.) References: Proceedings of the 2003 International Symposium on Indoor Air Pollution and Health Hazards. Tokyo, Japan. January 8 to 11, 2003.

Abstract Information & Notes

Allan D. Lieberman, M.D.

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

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

Date of talk: Saturday, June 21, 2003, 11:00am

Medical School Attended:	Chicago Medical School
Major and date of Graduation:	1960
Residency:	Children=s Memorial Hospital - Chicago
Board Certifications:	Fellowship - American Academy of Environmental Medicine American Board Environmental Medicine
Current Job Description:	Medical Director of Center for Occupational & Environmental Medicine
Disclosure Statement:	None

SPEECH TITLE: **"Explosion of Mold Cases in Homes, Work Places and in Occupational Medical Practices"**

The speaker has provided the information below.

1.) Goals and objectives: To explore the different questions of toxic mold exposures.

2.) Outline of talk/abstract: A clinical presentation of the effects and sequelae of 3 separate sick building exposures to mold.

3.) Conclusion of what is to be learned: Patterns of Reactivity to mold, their similarities, differences and their chronicity.

4.) References:

Explosion of Mold Cases in Homes, Workplaces and in Occupational Medical Practices

Allan D. Lieberman, M.D.

In practices all over the country, there has been an explosion of patients seeking help from alleged exposure to molds both in their homes and workplaces. The severity of their symptoms and the multi-system spectrum of their complaints demands that physicians seeing these patients become more knowledgeable about the serious health effects of mold exposure.

Yet, we are told in a position paper[1] published by the American College of Occupational and Environmental Medicine and peer reviewed by the Council on Scientific Affairs, that "mold growth indoors is undesirable but does not warrant the fear that is too often associated with it. A careful review of the science suggests that irrational fear of indoor mold threatens responsible public policy more than indoor mold threatens public health."[2]

On what clinical evidence is this opinion based? Objective analysis requires you to believe in what you see and not see what you believe.

The case report is the gold standard in identifying the adverse effects of environmental exposures and it is the obligation of physicians to report these cases. This presentation will do just that, summarizing the findings in 48 cases of mold exposure.

The case reports presented derive from workers in a bank, industrial plants, teachers in schools, and people in their homes. All were knowingly exposed to molds that were professionally evaluated, identified, and quantified. Most exposures were long-term lasting weeks to months. Moisture was the universal cause precipitating the growth of the indoor mold. The mold species identified varied but the most common were:

Aspergillus
Penicillium
Cladosporium
Stachybotrys

Multiple systems were affected confirming the multi-system injury that mold exposure can produce. The spectrum of signs and symptoms in descending order of frequency included:

Muscle and/or joint pain	71%	
Fatigue/weakness	70%	
Neurocognitive dysfunction	67%	
Sinusitis	65%	
Headache	65%	
Gastrointestinal problems	58%	
Shortness of breath	54%	
Anxiety/depression/irritability	54%	
Vision problems		42%
Chest tightness	42%	
Insomnia	40%	
Dizziness	38%	
Numbness and tingling	35%	
Laryngitis/hoarseness	35%	
Nausea	33%	
Rashes	27%	
Tremors	25%	
Heart palpitations	21%	
Bronchitis/pneumonia	21%	

Nose bleeds	13%
Nasal Septal Perforation	2%

Mold and mycotoxin antibody titers:

23 out of 29 or 80% of patients tested showed positive antibodies to molds and mycotoxins.

Trichothecene	38%
Aspergillus	34%
Cladosporium	31%
Penicillium	28%
Stachybotrys	28%

Conclusions:

The findings of 48 cases of serious health effects from mold exposure suggests that mold is a significant cause of illness, impairment and disability.

References:

1. Hardin, B.D., Kelman, B.J., Saxon, A., ACOEM's evidence based statement on the Adverse Health Effects Associated With Molds In The Indoor Environment. ACOEM's report. Oct/Nov/Dec 2002.
2. Brunekreff, B., 1992. Damp Housing And Adult Respiratory Symptoms. Allergy 47:498-502.
3. Brunkreff, B., D.W. Dockery, F.E. Speizer, J.H. Ware, J.D. Spengler, and B.G. Ferris. 1989. Home Dampness And Respiratory Morbidity In Children. Am. Rev. Respir. Dis. 140: 1363-1367.
4. Dales, R.E., H. Zwanenburg, R. Burnett, and C.A. Franklin. 1991. Respiratory Health Effects Of Home Dampness And Molds Among Canadian Children. Am. J. Epidemiol. 134:196-203.
5. Packer, C.N., Stewart-Brown, and S.E. Fowle. 1994. Damp Housing And Adult Health: Results From A Lifestyle Study In Worcester, England. J. Epidemiol. Community Health 48:555-559.
6. Firhonen, I., A. Nevalainen, T. Husman, and J. Pekkanen. 1996. Home Dampness, Molds And Their Influence On Respiratory Infections And Symptoms In Adults In Finland. Eur. Respir. J. 9:2618-2622.
7. Platt, S.D., C.J. Martin, S.M. Hunt, and C.W. Lewis. 1989. Damp Housing, Mold Growth, And Symptomatic Health State. Br. Med. J. 298:1673-1678.
8. Engelhart, S. et al, Applied and Environmental Microbiology, August 2002, P. 3886-3890.
9. Bornchag, CG. et al. Indoor Air, 2001 June 1 (2): 71.
10. Beebe, Glenn. Toxic Carpet Three. 1971.
11. Andreissen, J.W., B. Brunekreff, and W. Roemer. 1998. Home Dampness And Respiratory Health Status In European Children. Clin. Exp. Allergy 28:1991-1200.

Abstract Information & Notes

Nancy A. Didriksen, Ph.D.

100 North Cottonwood Drive
Ste. 106
Richardson, TX 75080

Phone: 972/889-9933
Fax: 972/889-9935
E-mail: N/A

Date of talk: Saturday, June 21, 2003, 11:30am

Medical School Attended:	University of North Texas
Major and date of Graduation:	1986 - Health Psychology/Behavioral Medicine
Residency:	N/A
Current Faculty Appointments:	Adjunct Professor of Psychology - University of North Texas
Current Job Description:	Private Practice, primarily evaluating and treating patients with chemical/environmental sensitivity, Chronic Fatigue Immune Deficiency Syndrome, and Fibromyalgia.
Other Information:	Internship - Environmental Control Unit, Northeast Community Hospital, Bedford, TX
Disclosure Statement:	None

SPEECH TITLE: **"Neurocognitive Deficits in Individuals Exposed to Toxigenic Molds"**

The speaker has provided the information below.

1.) Goals and objectives: Describe the neurocognitive profiles of mold-exposed patients and implications of findings as to the neurotoxic properties of toxigenic fungi.

2.) Outline of talk/abstract: 1. General effects of neurotoxic exposure. 2. Description of the neurocognitive deficits observed in mold-exposed patients. 3. Caveats of data interpretation. 4. Implications of data for litigation and/or disability issues. 5. Implications for future research.

3.) Conclusion of what is to be learned: Extent and kind of neurocognitive dysfunction observed in patients exposed to toxigenic molds, implications for litigation and disability issues, and caveats regarding data interpretation.

4.) References:

Auger, P.L. (1994). Mycotoxins and Neurotoxicity. In Johanning, E. and Yang, C.S. (Eds.), Fungi and bacteria in indoor air environments (Proceedings of the International Conference, Saratoga Springs, New York). Eastern New York Occupational Health Program.

Mandell, M. (1976). Mold allergy as a major cause of bio-ecologic mental illness. In L. Dickey (Ed.), Clinical ecology (pp.

Johanning, E. (Ed.). (2001). Bioaerosols, fungi and mycotoxins: Health effects, assessment, prevention and control. Alb

Neurocognitive Deficits in Individuals Exposed to Toxigenic Molds

Nancy A. Didriksen, Ph.D.

Goals and Objectives:

1. Describe the neuropsychological test results of patients reporting primary exposures to toxigenic molds.

2. Describe the types of neurocognitive deficits found most frequently in mold-exposed individuals.

3. Discuss the physical, psychological, and neurocognitive symptoms endorsed most frequently on the checklists and whether the reported neurocognitive symptoms are consistent with neuropsychological findings.

4. Discuss confounding variables in data analysis and their importance in disability and litigation issues.

5. Implications for future research.

Relatively few studies are available regarding the neurocognitive deficits resulting from exposure to toxigenic molds. Dr. Marshall Mandell (1976) reported cerebral reactions including inability to concentrate and confusion during provocative testing with various mold extracts and reviewed the biological sources of antibiotics with proven neurotoxic properties. More recently, cognitive impairment associated with exposure to toxigenic fungi has been reported by Gordon, Johanning, and Haddad (2001). Auger, Pépin, Miller, Gareis, et. al. (2001) have reported chronic toxic encephalopathies apparently related to exposure to toxigenic fungi. Baldo, Ahmad, and Ruff (2002) have described the neuropsychological performance of patients following mold exposure with impairment observed in visuospatial learning and memory, verbal learning and psychomotor speed.

The neurocognitive data of 41 patients (mean age 47.95, mean educational level 15.32) reporting primary mold exposures were examined in the present study. Neuropsychological test batteries of varying comprehensiveness (including the Halstead-Reitan Neuropsychological Test Battery) were administered. Test results were compared with normative data. Deficits were found primarily on measures of executive functions, psychomotor problem-solving, and incidental memory. Scores on the Wechsler Memory Scale-III fell within normal limits, overall, with greatest impairment on measures of visual memory. Fifty-seven percent of patients demonstrated mild to moderate impairment, overall, on the Halstead-Reitan Battery. Forty-two percent demonstrated mild to severe impairment on the Comprehensive Neuropsychological Screen with an additional 17 percent scoring in a low-normal range. Scores on measures of specific neuropsychological abilities showed some impairment of sensory and motor functions. IQ scores fell generally within expected ranges, overall.

Physical Symptoms reported most frequently on the Physical Symptom Checklist included fatigue, low energy, headaches, difficulty remaining asleep, weakness, sinus discomfort, skin problems, decreased balance and coordination, mucus, difficulty falling asleep, and "sick all over." Psychological symptoms most frequently reported on the Psychological Symptom Checklist included present performance inferior to prior performance or level of functioning, overwhelming exhaustion, fatigue, or weariness, "cloudy, foggy, spacey," "This is not me," difficulty getting started in the morning, worry about bodily dysfunction, tense, difficulty setting and reaching goals, inability to cope well with daily or other stressors, irritability, decreased libido, and feels like "insides are racing." Frequently endorsed neurocognitive symptoms on the Neurocognitive Symptom Checklist included decreased immediate and short-term memory, decreased concentration, decreased attention, difficulty remembering the names of things or people, intellectual inefficiency, word-finding problems, decreased comprehension, decreased long-term memory, easily distracted, loses train of thought, and poorly organized.

Age, sex, and educational norms were utilized in addition to the standardization group norms to determine whether a decrement in functioning had occurred. Additionally, prior levels of educational and occupational performance were considered in the interpretation of test results, particularly important for disability determination. All patients to whom a malingering test had been administered scored within normal limits.

Many factors, including time elapsed from exposure to evaluation (0-67 months), duration of exposure (less than 1 month to 12 years), other environmental exposures/sensitivities, motivation of the individual, reactions to environmental incitants at the time of evaluation, head trauma and/or other neurological conditions, medications, metabolic disorders, and past or present drug and alcohol abuse were considered in the interpretation of individual test data and to determine whether the observed deficits were due primarily to mold exposure. These factors are particularly important for litigation issues.

Conclusions:

The neuropsychological test data of mold-exposed individuals present a pattern of deficits quite similar to those found in patients exposed to other neurotoxins including solvents, pesticides, and metals. Results suggest that the molds to which these patients were exposed have neurotoxic properties. Deficits do not appear as severe as those observed with other types of neurotoxic exposures. Possible explanations include (1) a greater awareness of physical discomfort alerting the individual to the toxic environment and decreasing the duration of exposure, (2) neurotoxic effects of molds are less severe, (3) small sample size, (4) sensitivity of tests administered, (5) individual susceptibility.

Further research is necessary to determine the neurotoxic effects of specific molds, types of individuals with greater susceptibility to neurotoxic effects, factors which appear to enhance susceptibility to adverse effects, and interventions, which promote healing after exposure. A larger database and collaboration among various healthcare providers are necessary.

References:

American Psychiatric Association (1990). Diagnostic and statistical manual of mental disorders (4th Ed.) (pp. 733-34). Washington, D.C: American Psychiatric Association.

Auger, P. L. (1994). Mycotoxins and neurotoxicity. In Johanning, E. and Yang, C. S. (Eds.), Fungi and bacteria in indoor air environments (Proceedings of the International Conference, Saratoga Springs, New York). Eastern New York Occupational Health Program.

Baldo, J.V., Ahmad, L. & Ruff, R. (2002). Neuropsychological performance of patients following mold exposure. Applied Neuropsychology, 9(4), 193-202.

Harrell, E.H., Butler, J.R., & Didriksen, N.A. (1986). Comprehensive neuropsychological screen. Unpublished assessment instrument. Health Psychology/Behavioral Medicine Associates, 100 N. Cottonwood Dr., Suite 106, Richardson, TX 75080.

Heaton, R. K., Grant, I. & Matthews, C. G. (1991). Comprehensive norms for an expanded Halstead-Reitan battery. Odessa, FL: Psychological Assessment Resources, Inc.

Jarvis, P.E. & Barth, J.T. (1994). The Halstead-Reitan neuropsychological battery: a guide to interpretation and clinical applications. Odessa, FL: Psychological Assessment Resources, Inc.

Johanning, E. (Ed.). (2001). Bioaerosols, fungi and mycotoxins: Health effects, assessment, prevention and control. Albany, NY: Fungal Research Group (FRG), Inc. Boyd Printing Company, Inc.

Lezak, M.D. (1995). Neuropsychological assessment (3rd Ed.) New York: Oxford.

Mandell, M. (1976). Mold allergy as a major cause of bio-ecologic mental illness. In L. Dickey (Ed.), Clinical ecology (pp. 259-261). Springfield, Il: Charles C. Thomas.

Reitan, R.M. & Wolfson, D. (1993, 2nd Ed.). The Halstead-Reitan neuropsychological test battery: theory and clinical interpretation. Tucson, AZ: Neuropsychology Press.

The Psychological Corporation. (1997). WAIS-III WMS-III technical manual. San Antonio: The Psychological Corporation.

Wechsler, D. (1981). Wechsler adult intelligence scale - revised. New York: Psychological Corporation.

Abstract Information & Notes

Jean A. Monro, M.D.

Breakspear Hospital
Hertfordshire House
Wood Lane, Paradise Estate
Hemel Hempstead, Herts, HP2 4FD
U.K.

Phone: 011442261333
Fax: 011442266388
E-mail: info@breakspearmedical.com

Date of talk: Saturday, June 21, 2003, 1:30pm

Medical School Attended: London Hospital Medical College
Major and date of Graduation: Medicine MB, BS 1960
Current Job Description: Medical Director of Breakspear Hospital, Hemel Hempstead, UK

Disclosure Statement: None

SPEECH TITLE: **"Treatment of Cancer with Mushroom Products"**

The speaker has provided the information below.

1.) Goals and objectives: To provide information on the use of natural resources in the treatment of cancer. To highlight that these can be a protective measure through life.

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned: Prevention of disease and treatment can overlap. Mushroom products can be therapeutically beneficial in prevention. There are toxic mushroom products which have been used in chemotherapy.

4.) References:

4. Ikekawa T. Beneficial effects of edible and medicinal mushrooms on health care. Int J Med Mushrooms 2000;3:291-8.
5. Monro JA. Coriolus: the use of the medicinal mushroom Coriolus MRL as an immunotherapeutic agent in the treatment of patients with chronic fatigue syndrome. To be published 2003.
6. Smith JE, Rowan NJ, Sullivan R. Medicinal mushrooms; their therapeutic properties and current medical usage with special emphasis on cancer treatments. London: Cancer Research UK; 2002.

Dr. Jean A. Monro,
Breakspear Hospital,
Hemel Hempstead, U.K.

ABSTRACT

In the treatment of cancer, most treatments currently used are designed to be cytotoxic to the neoplastic cells. One of the most promising methods to be considered is an immunological method of control. No agent other than mushrooms can be used both for prevention and treatment. In both, mushroom species have the properties for immune modulation and prevention of disease, which certainly is not the case with those forms of treatment, which are cytotoxic. Some mushroom products have other medicinal purposes; for example antibiotics and ergot alkaloids, Penicillin, Griseofulvin and also of course some cytotoxic agents such as Cyclosporin which are fungus-derived.

However, mushroom-derived polysaccharides can modulate animal and human responses and inhibit tumour growth. They influence cells through cytotoxic macrophage activity, monocytes, neutrophils, natural killer cells, dendritic cells, and alter chemical messengers, cytokines, which trigger complement and acute phase responses such as the interleukins, interferons and colony-stimulating factors. Multi-cytokine inducers can induce gene expression of immunomodulatory cytokines and cytokine receptors. They can stimulate
T-cells with cell mediated cytotoxicity and B-cells with antibody production.

In Japan, Korea and China Phase I, II and III clinical trials have been performed and mushrooms are now used as adjuncts to chemotherapy. Some of the main important polysaccharide compounds that have undergone clinical trials include :

- Lentinan from *Lentinula edodes*
- Schizophyllum from *Schizophyllum commune*
- PSK Krestin and PSP from *Trametes versicolor*
- Grifon D from *Grifola frondosa*

Letinan and Schizophyllum are T-cell oriented immuno-potentiators and require a functional T-cell component for biological activity by way of increasing helper T-cell production, increased macrophage production leading to a stimulation of acute phase proteins and colony stimulating factors, which in turn affect proliferation of macrophages, neutrophils and lymphocytes and activation of the complement system.

PSK Krestin and PSP are potent immuno-stimulators with specific activity for T-cells and for antigen presenting cells such as monocytes and macrophages. Their biological activity is characterised by their ability to increase white blood cell counts, interferon gamma, interleukin 2 production and delayed hypersensitivity reactions.

Epidemiologically there have been large-scale studies of the effects of mushrooms. The anti-tumour activities of *Basidiomycetes* have been studied by the National Cancer Centre Research Institute of Japan since 1966. This has included an epidemiological investigation over 15 years from 1972 to 1986. 174,505 people were studied. A group of people in the Nagano Prefecture in Japan was examined and those farmers who were producing mushroom species *Flammulina velutipes* were compared with the rest of the population. The average cancer death rate of farmers was hugely different from those in the rest of the population. The cancer death rate in general was 160.1 per 100,000 and of the farmers 97.1 per 100,000. This edible mushroom is now popularly consumed as a functional food, particularly in the Nagano Prefecture.

In our own work we have shown that natural killer cell numbers can be doubled within two months of taking therapeutic doses of *Coriolus* which contains Krestin PSk and PSP. We have embarked on programs of assessment of treatment of patients with cancer with immune-modulating mushroom products.

Why do mushroom polysaccharides have such an amazing array of biopharmacological activities? Polysaccharides, unlike proteins and nucleic acids, contain repetitive structural features that are polymers of monosaccharide residues joined to each other by glycosidic linkage. These polysaccharides offer a high capacity for carrying biological information because of their increased potential for structural variability. The amino acids in proteins and the nucleotides in nucleic acids can only interconnect in one way, while the monosaccharide units in the polysaccharides can interconnect at several points to provide a wide range of branched molecules. The number of possible permutations from four different sugar monosaccharides could be up to 35,560, whereas four amino acids can only

form 24 different permutations. This allows an enormous flexibility for regulatory mechanisms of cell interactions. The responses that changes in this highly branched structure can allow information transmission. Most of the matrix between cells is constructed of glycoproteins in a similar way and it is the matrix, which allows the transfer of information through the organism at an incredibly rapid rate, it is said at the speed of sound. The incorporation of biological structures which are proteoglycans throughout the matrix of the body can be likened to the equivalent of fungal structures. Biologically this can be said to be equivalent to the incorporation within cells of mitochondria which we know of from plant species. It could be that this biological role that we have for proteoglycans in our structures is being augmented and mimicked by the polysaccharide and proteoglycans from mushroom species.

Natural killer cells are increased in numbers by mushroom treatment. In my paper on *Coriolus* I quoted the work of Roitt who showed that "NK cells are thought to recognise structures on high molecular weight glycoproteins which appear on the surface of virally infected cells and which allow them to be differentiated from normal cells. This recognition probably occurs through lectin-like [i.e. carbohydrate binding] receptors on the NK cell surface which bring killer and target into close opposition.

Activation of the NK cell ensues and leads to polarisation of granules between nucleus and target within minutes and extra-cellular release of their contents into the space between the two cells, often utilising the cytolysin perforin.

NK cells kill by activating **apoptosis.**

In addition to perforin, the granules contain tumour necrosis factor β and a family of serine proteases termed **granzymes**, one of which, granzyme B, can function as an NK cytotoxic factor. Also fully ionised ATP which can cause apoptosis in many different cell types; the effectors themselves are resistant probably due to a lack of ATP receptors on their surface. These factors sequentially induce NK-mediated lysis.

A current view is that granzyme B kills by directly activating an endogenous family of ICE [IL-1β converting enzyme] proteases which subsequently degrade other molecules including the repair enzyme poly [ADP-ribose] polymerase. Chondroitin sulphate A, a protease-resistant highly negatively charged proteo-glycan, is present in the granules and may protect the NK cell from autolysis.

The various interferons augment NK cytotoxicity and since interferons are produced by virally infected cells, there is an integrated feedback defence system.

Virally infected cells can be killed by cytotoxic T-cells and ADCC

Viral antibodies can bring the NK cell very close to the target virally infected cell by forming a bridge and the NK cell being activated by the complexed antibody molecules is able to kill the virally infected cell by its extra-cellular mechanisms. This system, termed **antibody-dependent cell-mediated cytotoxicity [ADCC]**, has been demonstrated *in vitro*." Natural killer cells scavenge cancer cells similarly.

Conclusion

Of the 10 million new cancers diagnosed worldwide annually, 1.5 million are said to have been associated with infections, especially viruses, 3 million due to toxic exposure and 3 million due to dietary causes. The remainder have an unknown aetiology.

Mushrooms may be able to address virally induced cancers as well as those due to dietary and lifestyle causes.

References :

1. Ikekawa T. Beneficial effects of edible and medicinal mushrooms on health care. Int J Med Mushrooms 2000;3:291-8.

2. Monro JA. Coriolus: the use of the medicinal mushroom Coriolus MRL as an immunotherapeutic agent in the treatment of patients with chronic fatigue syndrome. To be published 2003.

3. Smith JE, Rowan NJ, Sullivan R. Medicinal mushrooms; their therapeutic properties and current medical usage with special emphasis on cancer treatments. London: Cancer Research UK; 2002.

Abstract Information & Notes

Larry Foster

Enviro Cure
1280 West Peachtree Street, Suite 1606
Atlanta, GA 30309

Phone: 404/876-3680
Fax: 404/685-0971
E-mail: N/A

Date of talk: Saturday, June 21, 2003, 2:00pm

Current Job Description: Senior Environmental Consultant
Other Information: Discovered that species of mold in the environment (indoors) were causative factors in human sickness including allergies, arthritis, asthma, headaches, learning disabilities, sinus infections, sleep apnea and more.

Disclosure Statement: None

SPEECH TITLE: **"General Reasons and Causes of Sick Buildings that are a Result of Airborne Biohazards"**

The speaker has provided the information below.

1.) Goals and objectives: To provide information to the Physician that is a necessity for an optimum diagnosis.

2.) Outline of talk/abstract:

I. How a Source of Disease Begins
II. How it Grows and Spreads
III. How it Enters the Human System and Causes Disease

3.) Conclusion of what is to be learned: A "New@ Broader view to Health Care (Treatment), viewing a "womb to the tomb@ approach.

4.) References:

E.P.A. Manual on Schools and Commercial Buildings

The Fungus Link by Doug Kaufmann

General Reasons and Causes of Sick Buildings that are a result of airborne biohazards.

Larry Foster

Enviro-CURE Services, Atlanta, Georgia

Do not buy a house (or building) that is in a gully, a food plain, or is downhill from the path of rainwater.

Do not by a house (or building) that has had water leaks that have not been corrected.

Do not by a house that has a flooded crawlspace, a sump pump (a great indications of previous water problems). ***(See A)***

Do not buy a house that has had watermarked walls or ceilings. In just a short time the house can be overrun by visible colonies. ***(See A1)***

Things to inspect (concerns) before you buy: Attic Area – Be sure that this area is not infested by birds, insects, rodents or varmints.

Check for visible signs of water intrusion. ***(See B)***

If there is blown-in fibrous insulation, it will disintegrate and particulate. The minute fibers become airborne and can be visually observed on the topside of ductwork and beams and on the A.C. system. ***(See C)*** These particles are able to infiltrate ***(See D)*** into the living areas as they are sucked into the A.C. and circulated through the air ducts and into the living areas. ***(See E)***

In many instances the attic openings that the ceiling (below) lights and ducting pass through in many cases are not properly caulked. ***(See F)***

This can be a source of airborne fibers, molds, bacteria, animal and insect by products that will infiltrate into the bedrooms (below the attic) and appear to the homeowner as just acceptable "house dust." ***(See G)*** It does not long before belongings are covered with mold and must be discarded. This "house dust" causes allergies, asthma, sleep apnea, snoring, rashes, congestion, infections and a long list of health complaints. ***(See H)***

To avoid these problems when building a new home, use insulation wrapped in plastic to prevent long-term degradation or aerolization of any fibrous particulate. These products are readily available at Lowe's and Home Depot Stores.

Attic A.C. systems are a source of contamination that not only sucks in fibers but minute mold and bacterial and insect fragments (feces) that locate in this dark rarely used area. ***(See I)***

HVAC Systems (In General)

For some reason the architects and builders have not come up with the concept that people live in the houses they design and build.

They have taken no time at all to consider that the houses that they have completed are in fact contaminated boxes that ultimately will cause sickness and death.

Picture a tightly insulated building that has had water leaks or rain or high humidity during the building process. When that house (or building) is "boxed" in, the A.C. units are being used by the contractor, which are sucking in debris, mold, bacteria, etc. ***(See J)***

The air is contaminated with sheetrock dust, fibers, cigarette smoke, chemicals and even food remnants left by laborers. The cabinets and carpets are off-gassing formaldehyde. ***(See K)*** All this is being circulated by a contaminated A.C. system.

You look out a window and it is raining. The roof begins to leak and the rainwater soaks the blown-in insulation. Insects and rodents find wet insulation ideal nesting areas. ***(See L)***

The small fragments of fibers are settling through holes around the lighting fixtures and down between two-by-fours, down the light sockets, in the walls. ***(See M)***

The rain subsides and a foul odor emanates from under the house. You walk around the house and find a small door that enters into a two or three-foot high dirt crawlspace ***(See N)*** that you can just about crawl around in and find it is muddy, foul smelling and there are no drains to alleviate any standing water ***(See O)***, which will infest the under floor beams and flooring.

And to make things worse the one air conditioner is located in this mud and the other one is in the damp attic along side the wet fiberglass insulation.

It is a wonder why the A.C. systems, which are designed fro our comfort and health, are always installed in the dirtiest, foulest, most contaminated locations in our homes. It is as if the homes were designed by the pharmaceutical companies. After living in a home of this description for less than one year, your children will be diagnosed as asthmatic. Someone will experience respiratory ailments, sleep apnea, and chronic illness and even worse your children's life is being programmed with anti-biotic. Isn't there something criminally wrong with this picture?

Humidifiers are helpful at times. They are a source of diseases at other times. When they are installed in the plenum of a furnace they are designed to spray water into the ducts. This will add moisture into the house and mold and bacteria into your lungs. ***(See P)*** The biohazards are spread by the air ducts ***(See P1)*** and eventually appear coming out of the air supply. ***(See P2)***

Humidity (over 50%) is necessary for the growth and habitat for dust mites. If you can control your humidity levels to below 40%, dust mites cannot live in your environment. If you don't the dust mites and their feces become airborne and can cause allergies and respiratory complaints and aid in the spread of diseases. They are one of the top allergens of note.

HEPA Vacuums (Not a Brand Name), a rating of 99.997 @ 0.3 microns, are very effective in reducing airborne particulates and germs that are causes of many allergies and diseases. An average vacuum cleaner produces more dust into the air than it picks up, it's just that you can't see it. Many times the large particles of dust are just pulverized by the vacuum and become minute microscopic airborne contaminants that may remain airborne for months.

People with allergies and those who are hypersensitive to dust and mold are safer in their homes if a HEPA or retrofitted HEPA is attached to your current vacuum cleaner.

Abstract Information & Notes

Theodore R. Simon, M.D.

Functional Imaging of Texas, PA
4429 Southern Avenue
Dallas, TX 75202

Phone: 214/528-2482
Fax: 972/566-4762
E-mail: ted@aya.yale.edu

Date of talk: Saturday, June 21, 2003, 2:30pm

Medical School Attended:	Yale University
Major and date of Graduation:	M.D., 1975
Residency:	Nuclear Medicine
Board Certifications:	Nuclear Medicine
Current Job Description:	Private Practice
Disclosure Statement:	None

SPEECH TITLE: **"Neurotoxicity: Mold Exposure versus All Causes"**

The speaker has provided the information below.

1.) Goals and objectives: Demonstrate the utility of scintigraphy in diagnosing frequently encountered environmental illnesses.

2.) Outline of talk/abstract: Available nuclear medicine techniques will be shown as they relate to specific illnesses with particular emphasis on the brain.

3.) Conclusion of what is to be learned: Appropriate, cost-effective use of nuclear medicine procedures in environmental illnesses can be achieved using a systematic approach.

4.) References:

Neurotoxicity: Mold Exposure versus All Causes
Theodore R. Simon, M.D.
June 21, 2003

Goals and Objectives: We have developed an interest in neurotoxicity as demonstrated by single photon emission computed tomography using a tracer for glutathione function. Recently, we have attempted to focus this interest on patients with putative neurotoxicity from mold exposure. That focus will be explored and used as a basis for explaining the clinical utility of scintigraphy of the brain as a clinical tool for addressing the care of environmentally challenged patients.

The audience will learn how to determine whether this technique is appropriate in individual patients that may require management for environmental disease exposure.

Outline: We have performed single photon emission computed tomography (SPECT) with [technetium-99m]HMPaO on 30 patients taken from a sample of 153 consecutive patients who presented with neurotoxicity and a history of mold exposure. These data will be presented and compared with our normal sample and with previous patients who have been examined for neurotoxicity from all causes.

Our usual technique of early and late single photon emission computed tomography using a triple head SPECT gamma camera and an intravenous injection, under microprocessor control, of [technetium-99m]HMPaO showed at least one of the findings that we expect in neurotoxicity was present in every patient. These findings are: mismatch between the early and late phases, shunting of the tracer to the soft tissues, temporal lobe asymmetry, and a “salt and pepper pattern". The salt and pepper pattern is defined as multiple hot and cold foci distributed throughout the cortex without regard to lobar distribution.

The sample of thirty subjects to be presented was acquired between November 22, 2000 and December 5, 2002. It consisted of 20 (67%) females and 10 (33%) males aged 5 to 67 years (average±sem= 43.3±2.18). Overall, the population showed no severe cases with the neurotoxic pattern; 11(37%), moderate; 15(50%), mild; and 4(13%), without the pattern. This overall distribution differs from the usual population of clinically neurotoxic patients. Mold exposure was associated with an absence of the pattern to a severe degree and by a higher incidence of moderate disease. Other, more subtle, differences will be explained in the presentation.

Conclusions: Triple head single photon emission computed tomography of the brain could be a useful adjunct to the clinical management of patients with putative neurotoxic exposure from mold.

Abstract Information & Notes

Donald P. Dennis, M.D., F.A.C.S.

ENT & Facial Plastic Surgery, L.L.C.
3193 Howell Mill Rd., Suite 215
Atlanta, GA 30327

Phone: 404/355-1312
Fax: 404/352-2798
E-mail: ddennis@mindspring.com

Date of talk: Saturday, June 21, 2003, 3:30pm

Medical School Attended:	Medical College of Georgia, Augusta, Georgia
Major and date of Graduation:	MD, 1974
Residency:	Otolaryngology - Head & Neck Surgery, John's Hopkins Hospital
Board Certifications:	American Board of Otolaryngology & Head & Neck Surgery
Current Faculty Appointments:	ENT Department, Northside Hospital
Current Job Description:	Private Practice, Atlanta
Disclosure Statement:	None

SPEECH TITLE: **"Guidelines and Theory for Treatment of Chronic Fungal Sinusitis with Reduction of Environmental Air Mold Load and Anti-microbial Nasal Sprays Based on 14 Years Clinical Observation in 639 Patients"**

The speaker has provided the information below.

1.) Goals and objectives:

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:

4.) References:

Title:

Guidelines and Theory for Treatment of Chronic Fungal Sinusitis with Reduction of Environmental Air Mold Load and Anti-microbial Nasal Sprays Based on14 Years Clinical Observation in 639 Patients

Introduction:
Mayo Clinic study of 9-99 found 93% of all chronic rhinosinusitis (CRS) was due to Allergic fungal sinusitis (AFS). AFS is caused by an immune response to the fungal antigen on the nasal mucosa. **The purpose of the study was to determine if antigen (fungus) removal in the air and nasal mucosa would reverse the disease and normalize the mucosa.**

Methods

639 patients with AFS were studied. One-hour gravity SDA agar plate exposures and Endoscopic nasal photographs were accomplished in the patient's environment before and after environmental remediation. Nasal fungal cultures were accomplished initially with nasal swabs directly on SDA agar. A protocol was developed to reduce mold in the environmental air and to reduce mold in the nasal mucosa. Environmental air mold reductions was accomplished with a combination of room HEPA air filtration, evaporation of Grapefruit seed extract (GSE), vent covers with GSE spray, or non-ozone ionization in each room the patients frequent. Nasal fungal reduction was accomplished by normal saline nasal irrigations, and antimicrobial nasal sprays using a combination of antibiotics- antifungals-steroids.

Results

639 patients were studied. 365 of 639 were able to achieve a mold count of less than 4 per one-hour plate exposure. 343 of 365 or 94% showed normal nasal mucosa without infection. Of the 22 who failed to normalize the nasal mucosa, 3 had lymphoma and 19 had positive nasal fungal cultures. 219 did not reduce the mold count below 4 colonies and had various degrees of mucosal disease remaining.

Conclusion

AFS is caused by an immune response to fungal antigen. When the antigen is removed from the nose and air, the immune reaction stops and the mucosa normalizes. Exceptions to this are other underlying diseases or failure to find the location of mold exposure.

References

1. Schubert, M.S. asuperantigen hypothesis for the pathogenesis of chronic hypertrophic rhinosonusitris, allergic fungal sinusitis, and related disorders. Ann Allergy, Asthsma, Immun.20014; 87:181.

2. Ponikau JU, Sherris, Kern EB, et al. The diagnosis and incidence of allergic fungal sinusitis. Mayo Clin Proc 1999; 74:877-884.

3. Shin SH, Kita H. Abnormal immunologic responses to fungal antigens in patients with chronic rhinosinusitis. J Allergy Clin Immunol 2001; 107:S163.

4. Schubert MS, Goetz DW. Evaluation and treatment of allergic fungal sinusitis. I. Demographics and diagnosis. J Allergy Clin Immunol 1998; 102:387-394.

Abstract Information & Notes

Larry Foster

Enviro Cure
1280 West Peachtree Street, Suite 1606
Atlanta, GA 30309

Phone: 404/876-3680
Fax: 404/685-0971
E-mail: N/A

Date of talk: Saturday, June 21, 2003, 4:00pm

Current Job Description: Senior Environmental Consultant
Other Information: Discovered that species of mold in the environment (indoors) were causative factors in human sickness including allergies, arthritis, asthma, headaches, learning disabilities, sinus infections, sleep apnea and more.

Disclosure Statement: None

SPEECH TITLE: **"The Mold Rush 21st Century"**

The speaker has provided the information below.

1.) Goals and objectives: To Clarify the Truth and Fiction of Mold

2.) Outline of talk/abstract:
I. Warning: Fly By Nights (Get Rich Quick Companies) of the future liabilities
II. Potential Criminal Charges

3.) Conclusion of what is to be learned: Information that we have discovered and disseminated has reached the medical community who will be fighting a battle that must not and cannot be lost.

4.) References:
Crane vs. Bank of America (MoldUpdate.Com)

Reber vs. ServiceMaster (MoldUpdate.Com)

The Mold Rush 21st Century

Larry Foster

Enviro-CURE Services, Atlanta, Georgia

Since the beginning of time no business or industry has as many people with little or no knowledge of the business, enter into what is known as the " Mold Rush."

Many who are now in this "new" mold remediation business were a carpet cleaner, duct cleaner, restoration or A.C. Company just a short time ago and those are the better ones.

In fact, there are mold instructional companies on the internet that can teach you all you need to know about mold remediation, negotiating with insurance companies, figuring prices, and all that in only three days, you can be a certified mold "expert" overnight. ***(See Lawsuits)***

Many of these companies will wind up in lawsuits because they do not understand the overall scope of the problem. Most will never learn. The insurance companies are now having to deal with the new "mold problems" and they try to sort out who knows what they are talking about.

These problems, as I have previously stated, are not new. As you are all aware, Toxic Molds have been on this planet, for thousands of years, maybe millions.

The Bible makes mentions of mold and mildew in Leviticus. It took four thousand or so years for scientists to discover that the curse of the Pharaoh tomb was caused by Aspergillus mold. Scrapings on the walls of the tomb indicated that the molds that were isolated and I.D. were in fact alive and proved to be fatal to those who had comprised immune systems.

The insurance companies have known for years about the health problems that have caused sickness and death to their insured, but were in denial.

It was cheaper that way and they had more money for lawyers than the victims of mold had. In this era the tide has tuned and karma has finally gotten to the insurance companies who fit into that category.

The "mold remediation" business is compromised of misinformation, a lack of genuine knowledge, get-rich-quick specialists and those with all kinds of products and gimmicks. To top it off mold protocols, are being written by ex-asbestos specialists who have just gotten back to work.

I bring this negativity to the forefront at this meeting to share information with other individuals who want to share their knowledge to others who know the serious nature of the "mold remediation business."

I have some comments that I have learned from my experience that I believe will help the medical profession if they consider my advice.

The medical profession and its skilled practitioners must re-adjust it's thinking when diagnosing a patient with chronic cold or flu symptoms prior to prescribing anti-biotic or free samples from pharmaceutical companies. Doctors who want to cure their patients must first ask a few pertinent questions about their home and workplace environments. If either place has had water or mold damages, they must take this into consideration. This is the tip of the proverbial iceberg. Symptoms that appear as Fibromyalgia, Lupus, Epstein Bar, Chronic Fatigue, and multiple chemical sensitivities may start with just "minor infections or allergies."

I can tell you that I have interviewed thousands of people in my lifetime working with doctors, hospitals, and health departments for over thirty years and the aforementioned "minor" sniffles and congestion have ONE common denominator. ALL of these people lived in (or their merry-go-round of medication and anti-biotic treatments) began in a wet, damp, moldy environment. At this time I will share with you secrets that I shared with the CDC.

The main reason, that the mold remediation business will become a deadly gamble and not a get rich scheme that enables everybody with a pick-up truck to be a mold expert in three days. To begin with, mold is a moving target; molds multiply by the production of microscopic spores. The spores are activated by water, which is the catalyst for mold growth. Their process is similar to watering grass seeds. The nutrients for their rapid growth are provided by building materials, which enables mold to spread by forming other colonies. ***(See Note)***

The airborne spores range in size between 0.3 to 0.10 microns. Once the spores become airborne they are circulated by people traffic, drafts, air conditioning systems and ceiling fans. ***(See Note)***

The spores continue to float in the air until they adhere to other airborne particulates and fall by gravity to ledges, sills, furniture, or worse, in the bedding and carpeting. ***(See Note)***

Once mold forms visible colonies they continue to produce new spores by the billions. This procedure can literally transform a two thousand square foot house into a mold garden in just a couple of months (much sooner in humid areas).

Earlier, I referred to allergic symptoms as the tip of the iceberg. That is to say "there is something unhealthy that is happening to me. I don't feel good, etc."

Now I would like to refer to visible mold colonies as to being the "iceberg." As the colony grows it is extremely visible and it shouldn't take much to wash it with Clorox or disguise it by painting over it with Kilz, or remove a few pieces of sheetrock. Anybody can do that. Wrong! And I'll tell you why. ***(See Notes)*** This is the breaking point in the mold remediation business and a boon to the medical profession. Ant-biotics will not prove to be an effective cure if a patient has been toxified by mold. The anti-biotics are only effective for what they were meant for, killing bacteria.

21st ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT

SCHEDULE

Sunday, June 22, 2003

8:15 a.m.	**ANNOUNCEMENTS/MODERATOR: Douglas B. Seba, Ph.D.**
8:30	**William A. Croft, D.V.M., Ph.D.**, Private Practice, Mycotoxins, Environmental Diagnostic Group Inc., Madison, WI: "**Clinical Confirmation of Trichothecene Mycotoxicosis in Patients"**
8:50	Q & A
9:00	**Bruce Jarvis, Ph.D.,** Department of Chemistry & Biochemistry, University of Maryland, College Park, MD: **"Limitations in the Chemical Analyses for Mycotoxins"**
9:20	Q & A
9:30	**Kou Sakabe, M.D.,** Chief of Clinical Environmental Health Center, Kitasato Hospital, Kitasato Institute, Tokyo, Japan: "**Effects of Estrogenic Mycotoxins on the Brain and Immune Systems."**
9:50	Q & A
10:00	**BREAK WITH EXHIBITORS**
10:30	**Kaye H. Kilburn, M.D.,** Director of Environmental Sciences Lab, Ralph Edgington Professor of Medicine, University of S. California Medical Center**,** Keck School of Medicine, Los Angeles, CA: "**Chemical Agents and Mechanism of Mold Disorders"**
10:50	Q & A
11:00	**Professor Tang G. Lee, AAA,** Professor**,** Faculty of Environmental Design, University of Calgary, Calgary, Alberta, Canada: "**Courthouse Molds Caused by Air Migration"**
11:20	Q & A
11:30	**Bertie Griffiths, Ph.D.,** Director of Environmental Health Center B Dallas Laboratory, Environmental Health Center B Dallas: "**Mycotoxins as Vaccine Stimulants"**
11:50	Q & A
12:30	**SUMMARY AND CLOSE Douglas Seba, Ph.D.**

SUNDAY, JUNE 22, 2003

ABSTRACTS

AND

HANDOUTS

Abstract Information & Notes

William A. Croft, DVM, Ph.D.

Environmental Diagnostic Group Inc.
521 Hilltop Dr.
Madison, WI 53711

Phone: 715/757-3756
Fax: 715/757-9302
E-mail: doccroft@hotmail.com

Date of talk: Sunday, June 22, 2003, 8:30am

Veterinary School Attended:	University of Minnesota
Medical School Attended:	University of Wisconsin, Madison, Wisconsin
Major and date of Graduation:	Ph.D. in Medical Pathology from the University of Wisconsin, Madison, Wisconsin.
Current Job Description:	Study Human diseases within the environment from an outbreak of human disease as a Medical Pathologist.
Other Information:	Was on Faculty of the University of Wisconsin as Medical Pathologist, was accepted by the National Institute of Health as a Medical Pathologist, qualified to research human diseases, obtained over $900,000 of highly competitive research grants from the National Institute of Health while at the University of Wisconsin
Disclosure Statement:	None

SPEECH TITLE: **"Clinical Confirmation of Trichothecene Mycotoxicosis in Patients"**

The speaker has provided the information below.

1.) Goals and objectives: To explain the usefulness of the Urine Test in the Diagnosis of Trichothecene Mycotoxicosis.

2.) Outline of talk/abstract: The investigations of four Cases involving mold-contaminated buildings and human reaction to exposure, documents tests of extracted urine continuing trichothecene mycotoxins confirming exposure and the diagnosis of Mycotoxicosis in humans. In each of four Cases, the urine demonstrated antibiotic activity, sulfuric acid charring, and protein release. Urine was extracted using ethyl acetate 40V/60V[EA]. Extracted mycotoxin spotted on (TLC) displayed color and a range of (rf) between 0.2-0.6 using various solvents. Extract was re-suspended using 50% ethanol V/V to inject mycotoxins into weanling female Sprague-Dawley rats. Degeneration and necrosis of the rat=s tissue followed. Koch=s Postulates conditions were fulfilled by isolation of the causative agent, the trichothecene mycotoxins and the reproduction of disease. Examination of human tissue within the urine extraction group confirms Koch=s Postulates and comparative pathology confirms inhalation Mycotoxicosis, with severe necrosis of the central nervous system and severe scarring within the lungs. Extraction of mycotoxins from human patent urine is a very useful confirmatory test to demonstrate exposure and identify Mycotoxicosis. Low concentrations (6%) of sodium hypochlorite were ineffective against the activity of trichothecene mycotoxin. The severity or stages of disease directly correlates the level of exposure or poisoning (Patent Pending).

3.) Conclusion of what is to be learned: That the Urine test does confirm, the extraction of Trichothecene Mycotoxin does confirm the diagnosis of Mycotoxicosis. It clearly establishes exposure to this very poisonous mycotoxin.

4.) References:

a. Croft, W.A., Jarvis, B.B., and Yatawara, C.S.: Airborne Outbreak of Trichothecene Toxicosis, In: Atmospheric Environ, 20(3), 549-552 (1986).
b. Croft, W.A., Jastromski, B.M., Croft, A.L., and Peters, H.A., "Clinical Confirmation of Trichothecene Mycotoxicosis In Patient Urine," In: Journal of Environmental Biology 23(3), 301-320 (2002).
c. Pathre, S.V., and C.J. Mirocha: Assay Methods For Trichothecenes and Review of their Natural Occurrence, Mycotoxins in Human and Animals Health, Eds.: Roderick J.V., C.W. Hesseltine, and M.A. Mehlman, Pathotox Publishers, Park Forest South, Ill. Pages 229-253 (1977)

-

-

Clinical Confirmation of Trichothecene Mycotoxicosis in Patient Urine.

William A. Croft, Bonnie M. Jastromski, Amanda L. Croft and Henry A. Peters
Environmental Diagnostic Group Inc.,
Department of Environmental Pathology and Toxicology,

Abstract: The investigations of four Cases involving mold-contaminated buildings and human reaction to exposure, documents tests of extracted urine containing trichothecene mycotoxins confirming exposure and the diagnosis of Mycotoxicosis in humans. In each of four Cases, the urine demonstrated antibiotic activity, sulfuric acid charring, and protein release. Urine was extracted using ethyl acetate 40V/60V[EA]. Extracted mycotoxin spotted on (TLC) displayed color and a range of (rf) between 0.2-0.6 using various solvents. Extract was re-suspended using 50% ethanol V/V to inject mycotoxins into weanling female Sprague-Dawley rats. Degeneration and necrosis of the rat's tissue followed. Koch's Postulates conditions were fulfilled by isolation of the causative agent, the trichothecene mycotoxins and the reproduction of disease. Examination of human tissue within the urine extraction group confirms Koch's Postulates and comparative pathology confirms inhalation Mycotoxicosis, with severe necrosis of the central nervous system and severe scarring within the lungs. Extraction of mycotoxins from human patient urine is a very useful confirmatory test to demonstrate exposure and identify Mycotoxicosis. Low concentrations (6%) of sodium hypochlorite were ineffective against the activity of trichothecene mycotoxin. The severity or stages of disease directly correlates the level of exposure or poisoning (Patent Pending).

Key Words: Trichothecene, Mycotoxicosis, Urine confirmation test, Human pathology

Stages of Mycotoxicosis: For Inhalation of Mycotoxin

The three Stages (1-3) ranging from lower to higher severity of poisoning were modified according to exposure via the air as opposed to ingestion already established (Forgacs *et al.,* 1962; Joffe, 1971). A separate Stage of convalescence occurs when a patient is completely removed from the contaminated premises and the source of mycotoxin or mold spores.

Stage 1: The primary changes are in the brain, respiratory and immune systems, mucus membranes and gastrointestinal tract. Signs and symptoms may include burning sensation in the mouth, tongue, throat, palate, esophagus, and stomach, which is a result of the action of the toxin on the mucous membranes and skin in the exposed areas. Moist areas of the body armpits, under breasts, belt line and groin are more sensitive or first affected. Patients may report burning within the eyes, ears and nose. Patients also reported that their tongues felt swollen and stiff. Mucosa of the oral cavity may be hyperemic. Mild gingivitis, stomatitis, glositis, and esophagitis developed. Inflammation, in addition to gastric and (small and large) intestinal mucosal, resulted in vomiting, diarrhea and abdominal pain. Excessive salivation, headache, dizziness, weakness, fatigue and tachycardia were also present.

There may be fever and sweating. The respiratory system develops burning sensations and congestion. Severe exposure to mycotoxin within the lungs may lead to congestion, edema and failure, due to caustic action. Body temperature remains normal and controllable by the patient. The poisoning appears and disappears relatively quickly in this Stage with the exception of, lungs and central nervous system. Initially (Stage 1), the patient's symptoms are very uncomfortable or painful. As the poisoning continues and the patient progress toward Stage 2, he or she becomes accustomed to the presence of the mycotoxin and a quiescent period follows due to lack of nerve sensation. Depending on exposure levels, the first Stage may last from 3 - 9 days. In scoring the 50 signs and symptoms listed in Tables-1 and 2, an average score range of 20-45 represents Stage 1.

Stage 2: This Stage is often called the latent Stage or incubation period because the patient feels apprehensive, but is capable of normal activity in the beginning of this Stage. Every organ of the body is affected by degeneration and necrosis with continued exposure. The primary target organs for an individual become evident over time, due to biological variation. These are disturbances in the central and autonomic nervous systems resulting in headaches, mental depression, loss of short-term memory, loss of problem-solving ability, various neuropsychiatric manifestations, meningism, severe malaise and fatigue, narcolepsy, loss of temperature control, hyperesthesia or numbness of body areas, and cerebellar dysfunction including hypotonia, attitude and gait,

dysmetria, asthenia, vertigo, disturbances of speech, and loss of balance (Best, 1961). Spinal cord degeneration may also be observed in gait and reflex abnormalities, such as the ability to drive vehicles, ride bicycles or pass sobriety tests (inability to tolerate ethyl alcohol). Attention deficient disorder may be observed in children. Various systems may include: **Eyes**: visual disturbances, floating objects, light sensitive, lack of tears, burning and itching. **Ears**: burning, itching, and loss of hearing. **Immune and hematopoietic**: progressive loss of white and red cells including a decrease of platelets and hemoglobin, and high susceptibility to bacterial, mycotic and viral infections, debilitating chemical and allergies. **Gastrointestinal**: metallic taste in mouth, tooth loss, gum problems, stomatitis, sores in gums and throat, nausea, vomiting, diarrhea or constipation, excessive flatulence, abdominal distention, hepatitis, pancreatitis, and diabetes mellitus. **Respiratory:** burning and bleeding from nasal membranes, respiratory difficulty, asthma, extreme susceptibility to cold, flu and pneumonia. **Skin:** thinning of hair on head, burning on face, rashes, irritation, and edema. **Renal**: proteinuria, possible hematuria. **Reproductive:** irregular ovarian cycles, increased menstrual flow, fibroid growths in uterus, cystic development in mammary glands, and tumors of mammary and prostate glands. **Musculoskeletal**: somatitis, muscle weakness, spasms, cramps, joint pain, enlargement of joints in hand, and clubbing of fingers. **Cardiovascular:** chest pain, palpitations, ruptures of atrial walls, myocardial infection and aneurysm of arteries.

The skin and mucous membranes may be icteric, pupils dilated, the pulse soft and labile, and blood pressure may decrease or increase. The body temperature does not exceed 38 degree C and the patient may be afebrile, or chilled. Visible hemorrhagic spots may appear on the skin. Thoughts of suicide may be prominent in the person's mind at this time or anytime in Stage 2. Human bonding is very important for survival.

Degeneration and hemorrhages of the vessels marks the transition from the second to the third Stage of the disease and may not be consistently observed. The degeneration of the vital organs including serious respiratory insufficiency or asthma and CNS degeneration will take the patient into Stage three along with development of necrotic angina. If exposure continues, depending on exposure levels, Stage 2 may continue from weeks to months or even years until the symptoms of the third Stage develop. Evaluating the 50 signs and symptoms (Table-1 and 2) by assigning a score (0-least intense to 5-most intense or severe) to each symptom, we have determined that an average score range of 45-180 represents Stage 2.

Stage 3: Severe degeneration of the vital organs. The transition from the second to the third Stage is sudden. In this Stage, the patient's resistance is already low, and violent severe symptoms are present, especially under the influence of stress, or associated with physical exertion and fatigue. The first visible sign of this Stage may be lung, brain or heart failure (heart attack), with or without the appearance of petechial hemorrhage on the skin of the trunk, the axillary and inguinal areas, the lateral surfaces of the arms and thighs, the face and head, and in serious Cases, the chest. The petechial hemorrhages vary from a few millimeters to a few centimeters in diameter. There is increased capillary fragility and any slight trauma may cause the hemorrhages to increase in size.

Aneurysms of the brain or aorta may be observed by angiography. Hemorrhages may also be found on the mucous membranes of the mouth and tongue, and on the soft palate and tonsils. There may be severe interstitial thickening or scarring of the lungs, or respiratory failure. Nasal, gastric and intestinal hemorrhages and hemorrhagic diathesis may occur. Necrotic angina begins in the form of catarrhal symptoms and necrotic changes soon appear in the mouth, throat, and esophagus with difficulty and pain on swallowing. Severe degeneration of the skin on the face, eyelids, and loss of lashes is also often present.

Necrotic lesions may extend to the uvula, gums, buccal mucosa, larynx, vocal cords, lungs, stomach, and intestines and other internal organs such as the liver and kidneys and are usually contaminated with a variety of avirulent bacteria. Bacteria infection causes an unpleasant odor from the mouth due to the enzymatic activity of bacteria on proteins. Areas of necrosis may also appear on the lips and on the skin of the fingers, nose, jaws, and eyes. Regional lymph nodes are frequently enlarged. Esophageal lesions may occur and involvement of the epiglottis may cause laryngeal edema and aphonia (loss of voice). Death may occur by strangulation.

Patients may suffer an acute parenchymatous hepatitis accompanied by jaundice. Bronchopneumonia, pulmonary hemorrhages, and lung abscesses are frequent complications. Tumors may develop of various organs, including skin, urinary bladder, brain, mammary gland, bone, immune, liver, prostate, possibly resulting in death. The most common cause of death is brain failure due to both direct effects of the mycotoxin on the central nervous system and indirect effects due to respiratory failure or lack of oxygen to the brain caused by the severe

caustic inflammation (fibrinous exudation) reaction with the lung tissue, rendering it non-functional. Again, using the scoring system represented in Tables-1 and 2, an average score of greater or equal 180 represents Stage 3.

Stage of Convalescence: The course and duration of this Stage 3 depends on the intensity of the poisoning and complete removal of the patient from the premises or source of mycotoxin. Therefore, the duration of the recovery period is variable. There is considerable cellular necrosis and scarring to all major organs of the body in which cells will not regenerate, including the brain, spinal cord, eyes, lung, heart, liver, pancreas, kidney, adrenal, and blood vessels. If the disease is diagnosed during the first Stage, hospitalization is usually unnecessary, but allergies and asthma should be monitored closely. If the disease is diagnosed during the second Stage and even at the transition from the second to third Stages, early hospitalization may preserve the patient's life. If however, the disease is only detected during the third Stage, death cannot be prevented in most Cases.

1. Croft, W. A., Jastromski, B. M., Croft, A. L., and Peters, H. A., "Clinical Confirmation of Trichothecene Mycotoxicosis in Patients Urine", In: Journal of Environmental Biology **23**(3), 301-320 (2002)

2. Forgacs, J., and W. T. Carll : Mycotoxicoses. *In* : Advances in Veterinary Science. Academic Press, New York and London, pp 273-372 (1962).

Summary:

1. Direct Method to establish exposure to Mycotoxins.
2. Useful to establish diagnosis obvious and non-obvious exposure to Poisonous molds.
3. Useful method to date exposure to trichothecene mycotoxins, with 36 hours of exposure.
4. The amount of Protein observed in urine is associated with level of exposure and duration.

Abstract Information & Notes

Bruce Jarvis, Ph.D.

Department of Chemistry & Biochemistry
University of Maryland
College Park, MD 20742

Phone: 301/405-1843
Fax: 301/314-9121
E-mail: bj6@umail.umd.edu

Date of talk: Sunday, June 22, 2003, 9:00am

Education: B.A., Ohio Wesleyan University, Delaware, Ohio, 1963; Ph.D. University of Colorado, Boulder, Colorado, 1966; Postdoctoral Research Associate, Northwester University, Evanston, Illinois, 1966-67

Experience in Higher Education: Instructor of Chemistry (Part-time), Northwestern University, Evanston, Illinois, 1966; Assistant Professor, University of Maryland, 1967-71; Associate Professor, University of Maryland, 1971-79; Visiting Scholar in Residence, University of Virginia, 1975-76; Professor of Chemistry, University of Maryland, 1979-Present; Program Officer, Organic Synthesis, NSF 1987-88; Associate Chair 1988-89, 1992-1993, 1998-1999; Acting Chair 1989-90; Chair, Department of Chemistry and Biochemistry, 1993-1998; Visiting Professor, Dept. of Biotechnology, Danish Technical Univ., 1999-2000.

Disclosure Statement: None

SPEECH TITLE: "**Limitations in the Chemical Analyses for Mycotoxins"**

The speaker has provided the information below.

1.) Goals and objectives: To understand the limitations and problems associated with interpreting chemical and microbiological data obtained from damp, water-damaged, and moldy buildings, wherein those working or living are experiencing heath-related problems.

2.) Outline of talk/abstract: Although there are a number of excellent analytical methods for measuring levels of mycotoxins, applying these to real-life environmental situations can be very challenging. In addition to the inherent problems associated with measuring low levels of mycotoxins in the presence of many often interfering materials, there are many variables associated with the production of mycotoxins including, but not limited to, the species of the fungus, the particular isolate of a species, the substrate upon which the fungus is growing, and environmental conditions. Perhaps most vexing, is relating these indirect measurements to actual exposure experienced by people living or working in mold-contaminated buildings.
The fungus *Stachybotrys chartarum* has been much in the news as late for being a threat to those exposed to airborne spores of this toxigenic fungus. Various aspects of the chemistry and toxicology of this fungus will be presented, focusing on the above problems.

3.) Conclusion of what is to be learned: Life's not simple.

4.) References: 1. B. Andersen, K. F. Nielsen, and B. B. Jarvis, "Characterization of morphologically, chemically and physiologically different *Stachybotrys* species from water- damaged buildings," *Mycologia*, **94**, 392-403 (2002).
2. S. F. Hinkley and B. B. Jarvis, "Method for *Stachybotrys* Toxins," in Methods in Molecular Biology: The Mycotoxin Protocols, Vol. 157, M. W. Trucksess and A. E. Pohland, eds., Humana Press, pp. 173-194 (2000).

Abstract Information & Notes

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

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

Phone: 81-3-5490-2366
Fax: 81-3-5490-2366
E-mail: sakabe1@attglobal.net

Date of talk: Sunday, June 22, 2003, 9:30am

Medical School Attended:	Tokai University School of Medicine
Major and date of Graduation:	March 1982
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, Professor of Clinical Ecology
Current Job Description:	Clinical Ecologist, Environmental Toxicologist
Disclosure Statement:	None

SPEECH TITLE: "**Effects of Estrogenic Mycotoxins on the Brain and Immune Systems"**

The speaker has provided the information below.

1.) Goals and objectives: Elucidation of the brain and immune disrupting mechanism of estrogenic mycotoxins for health risk assessment.

2.) Outline of talk/abstract: a) strong inhibitory effect of estrogenic mycotoxins on the PKC activity of cytokine-stimulated $CD8^+$ lymphocytes was observed; b) estrogenic mycotoxins studied all significantly reduced p34cdc2 kinase activities; c) mycotoxin-treated animals showed a significant enhancement of the uptake by most of the preoptic-hypothalamic nuclei (POM, POL, SCH, SO, PV, VM, ARC, SPH and CC).

3.) Conclusion of what is to be learned: a) the cytoplasmic signal-generating system in developing or cytokine-treated lymphocytes are inhibited by environmental signals; b) and the defect occurs at all stages in the sequence of events leading to DNA synthesis, cell proliferation and cell differentiation; c) moreover, estrogenic mycotoxins influence protein synthesis in most of the brain regions including the preoptic and hypothalamic areas; d) the results of the present study suggest that we must recognize the possibility that estrogenic mycotoxins can affect the various physiological responses in the capacity of brain- and immune-disrupting chemicals.

4.) References:

1) Int. J. Immunopharmacol. 20(4-5), 205-212, 1998.
2) Int. J. Immunopharmacol. 21(12), 861-868, 1999.
3) Pathophysiol. 6(1), 231-236, 2000.
4) Environmental Endocrine Disrupters, edited by L.J. Guillette and D.A. Crain, Taylor & Francis, 1999.
5) Chemical brain injury, K.H. Kilburn, Van Nostrand Reinhold, 1998.

Effects of Estrogenic Mycotoxins on the Brain and Immune Systems

Kou Sakabe, M.D.
Chef, Division of Clinical Ecology, Environmental Medical Center-Tokyo, The Kitasato Institute,
Professor, Graduate School of Medical Sciences, Kitasato University
5-9-1 Shirokane, Minatoku, Tokyo 108-8642, Japan
E-mail: sakabe1@attglobal.net

Part 1: The present study was an attempt to elucidate the effect of estrogenic mycotoxins on the proliferation of mitogen-stimulated human peripheral blood lymphocyte (PBL). Our findings follow: (a) the proliferation of PBL in response to Interleukin-2 (IL-2) was mediated by protein kinase C (PKC), but estrogenic mycotoxins had a strong inhibitory effect on PKC activity of IL-2-stimulated PBL; (b) cytoplasmic extracts from IL-2-stimulated PBL greatly activated DNA replication, but estrogenic mycotoxins had a strong inhibitory effect on these activities. The results suggest that the cytoplasmic signal-generating system in cytokine-treated PBL is inhibited by estrogenic mycotoxins, and that the defect occurs at all stages in the sequence of events leading to DNA synthesis and cell proliferation.

Part 2: The Effect of estrogenic mycotoxins on the uptake of amino acids by the brain nuclei of ovariectomized (OVX) mice was examined. Two hr after the last treatment they were given a single s.c. injection of a mixture of ^{3}H-leucine and ^{3}H-methionine, and then sacrificed 2 hr later. Intensity of the uptake of radioactivity was measured on autoradiograms of the stained tissue sections. Control animals showed a relatively high uptake of radioactivity by the SO, PV and SPH, compared with that by the remaining brain. A low dosage mycotoxin-treated animals showed a significant enhancement of the uptake by most of the preoptic-hypothalamic nuclei except for VM, DM and PM, compared with that in control. On the other hand, high dosage mycotoxin-treated animals had slightly enhanced uptake by the POM, POL, SCH, SO, PV, VM, ARC, SPH and CC, compared with that in control. Uptake by the PM and EC remained unchanged in all groups. The present results suggest that estrogenic mycotoxins influence protein synthesis in most of the brain regions including the preoptic and hypothalamic areas.

Abstract Information & Notes

Kaye H. Kilburn, M.D.

University of Southern California
Keck School of Medicine
2025 Zonal Ave., CSC-201
Los Angeles, CA 90033

Phone: 323/442-1830
Fax: 323/442-1833
E-mail: kilburn@usc.edu

Date of talk: Sunday, June 22, 2003, 10:30am

Medical School Attended:	University of Utah College of Medicine
Major and date of Graduation:	1954
Board Certifications:	American Board of Internal Medicine, American Board of Preventive Medicine
Current Faculty Appointments:	Professor of Medicine University of Southern California Keck School
Current Job Description:	Ralph Edgington Professor - Academic Medicine Teaching, Research on Neurotoxicology, Pulmonary Disease
Other Information:	Author 240 peer reviewed papers; Book: Chemical Brain Injury, NY, John Wiley and Sons, 1998; President Neurotest Inc.; Develop test and use of Neurobehavioral methods in evaluated brain damage from chemicals; hydrogen sulfide, PCBs, pesticides, chlorine, ammonia, molds and mycotoxins
Disclosure Statement:	Neuro-test Inc.

SPEECH TITLE: **"Chemical Agents and Mechanisms of Mold Disorders"**

The speaker has provided the information below.

1.) Goals and objectives: Consider the evidence that mycotoxin poisoning of synapses and mitochondria are potent mechanisms of mold disorders.

2.) Outline of talk/abstract: Evidence for mycotoxins roles in neurological, pulmonary and immunologic impairment will be tied to quantitative methods

3.) Conclusion of what is to be learned: Methods to quantify mycotoxins emphasize nanogram amounts and biological activity potentially on synapses and mitochondria that would explain loss of memory, neurological slowing and chronic fatigue.

4.) References: see abstract

Chemical Agents and Mechanisms of Mold Disorders

Kaye H. Kilburn, M.D.

University of Southern California Keck School of Medicine

The new epidemic of mold disorders focuses upon airborne toxic products of molds, spores, fungal fragments and mycotoxins with high biological activity resembling the bacterial endotoxin derived lipopolysaccaride (LPS). Bioassays have been used to characterize cytotoxic activity of extracts of water damaged building material (Anderson et al 1997). Fragments of several fungal species were shown to be more numerous than spores, share their antigens and have immunological activity (Gorny et al 2002). In 1993 Smoragiewiez, Quebec, found trichothecene mycotoxin in dust samples from building ventilation analyzed by TLC a 4-(p-nitrobenzyl) pyridine and confirmed by HP-LC with a detection limit of 0.4-9 nanogram/mg of dust. A purified Stachyrase A (proteinase) from *Stachybotrys chartarus* cleaves protein inhibitors, collagen and lung peptides (Kordula et al 2002). Trichothecene mycotoxin can be detected with a firefly luciferase in rabbit reticulocytes with 400-fold sensitivity of the tetrazolium assay for this T. mycotoxin (Yike I et al 1999). In light of the impairments of the brain in exposed human subjects, effects on synapses should be sought in amyloid B protein aggregation and synaptophysin, a pre-synaptic vesicle protein. Release of these at low (nano-molar) levels slow synaptic neurotransmitter transport and correlate with memory loss in Alzheimer=s disease (Selkoe DJ 2002). Because fatigue is prominent in mold disorders, MCS and CFS, similar effects on electron transport in mitochondria should be explored.

REFERENCES

1. Anderson MA et al. Bacteria, Molds, and Toxins in Water-Damaged Building-Materials. *Appl Environ Microbiol* 1997;63(2):387-393.
2. Gorny RL et al. Fungal Fragments as Indoor Air Biocontaminants. *Appl Environ Microbiol* 2002;68(7):3522-3531.
3. Smoragiewiez W et al. Trichothecene Mycotoxins in the Dust of Ventilation Systems in Office Buildings. *Int Arch Occup Environ Health* 1993;65:113-117.
4. Kordula T et al. Isolation and Properties of Stachyrase A, a Chymotrypsin-Like Serine Proteinase from *Stachybotrys chartarum. Infection and Immunity* 2002;70:419-421.
5. Yike I et al. Highly Sensitive Protein Translation Assay for Trichothecene Toxicity in Airborne Particulates: Comparison with Cytotoxicity Assays. *Appl Environ Microbiol* 1999;65(1)88-94.
6. Selkoe DJ. Alzheimer=s Disease is a Synaptic Failure. *Science* 2002;298:789-791.

Abstract Information & Notes

Professor Tang G. Lee, AAA

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

Phone: 403/220-6608
Fax: 402/284-4399
E-mail: lee@ucalgary.ca

Date of talk: Sunday, June 22, 2003, 11:00pm

Major and date of Graduation:	Site planning and architecture, 1975
Current Faculty Appointments:	Professor of Architecture (Building Science and Environmental Health). Also Adjunct Professor at the University of Manitoba, and visiting scholar at the Lyle center for Regenerative Studies, California State Polytechnic University, Pomona
Current Job Description:	Conducting research investigations and teaching environmental health, particularly indoor air quality, building science and sustainability.
Other Information:	Conducts comprehensive indoor air quality investigations in an interdisciplinary team for those cases that could not be solved by other indoor air quality consultants. Also designs buildings such as medical clinics, institutions and residences that feature low toxicity.
Disclosure Statement:	None

SPEECH TITLE: **"Courthouse Molds Caused by Air Migration"**

The speaker has provided the information below.

1.) Goals and objectives: To understand how air movement across the building envelope can cause interstitial condensation that cause mold growth.

2.) Outline of talk/abstract: In cold climates, air migrating across the building envelope will create condensation. This condensation is an amplifier for mold growth. Courthouse occupants became ill when the air quality consultant recommended more outdoor air but did not balance the air pressure with proper exhaust

3.) Conclusion of what is to be learned: It is important to prevent air migration across the building envelope to prevent condensation and mold growth.

4.) References:

Lee, T.G. and Stookes, T. ***Environmental assessment of the Alberta Court of Appeals building.*** Report for Alberta Justice and Alberta Infrastructure, Edmonton, Alberta, March 5.

Lee, T.G. and Stooke, T. ***Mould propagation resulting from air pressure differences across the building envelope.*** *Proceedings of the 9th International Conference on Indoor Air Quality and Climate (Indoor Air 2002)*, Monterey, California, June 30 B July 5.

Abstract Information & Notes

Bertie B. Griffiths, Ph.D.

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

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

Date of talk: Sunday, June 22, 2003, 11:30pm

Medical School Attended: Graduate of the University of Wisconsin and University of the West Indies, Faculty of Medicine

Current Faculty Appointments: Professor and Consultant in Microbiology and Infectious Diseases

Current Job Description: Director of EHC-D Laboratory

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

Disclosure Statement:

SPEECH TITLE: **"Mycotoxins as Vaccine Stimulants"**

The speaker has provided the information below.

1.) Goals and objectives:

2.) Outline of talk/abstract:

3.) Conclusion of what is to be learned:

4.) References: