Instituut voor Studie van Schimmel in Menselijke Woningen
Institut pour l'Etude de Moisissure Fongique dans Habitations Humaines

Forschung Institut für Schimmelpilze in Innenräumen

MYCOLOGICAL INSTITUTE

for the study of

FUNGAL MOLD IN HUMAN HABITATIONS

highly recommend the signifigant reference work by

A.V. Costantini, Lars I. Qvick & Heinrich Wieland authors of " Fungalbionics"
Finally the proof that most (nearly all) major diseases of unknown etiology have their origin in fungal mold!
See Mycotoxins in Human Health http://www.micotoxinas.com.br/boletim28.htm

THE TROJAN HORSE:
FUNGI AND THEIR TOXINS IN THE FOOD CHAIN.....
THIS IS THE CAUSE OF THE MAJOR HUMAN DISEASES

The cancerous and degenerative diseases are the major causes of disability and death. The cause of these devastating diseases has been unknown. What is known is that they are all in some unexplained way related to food, alcohol or tobacco. However, confusion reigns supreme, for ***we all know that normal food itself cannot be poisonous.***

Very few of us know that poisonous toxin-producing fungi (molds) are characteristically present in stored food, and fungal-fermented food, drink and tobacco. These fungi are using what we eat, drink and smoke as a Trojan Horse to silently enter our bodies where they conquer and destroy us.

Thousands of studies have documented that fungi and their toxins (mycotoxins) cause virtually every type of cancer and degenerative disease in animals and/or in humans.

One might well ask why these fungal and mycotoxin facts have been ignored by all medical researchers and practicing physicians. It is probably for the same reasons that allowed the saga of the Trojan Horse to have occurred.

THE AUTHORS IN THEIR MYCOTOXIN RESEARCH LABORATORY

PROFESSOR COSTANTINI (Center)

PROFESSOR HEINRICH WIELAND (Right)

DR. LARS I. QVICK (Left)

FUNGALBIONICS PROVIDES AT LONG LAST THE MISSING FACTS OF THE "CAUSE AND CURE" OF CANCER AND DEGENERATIVE DISEASES.

MYCOTOXINS IN HUMAN HEALTH
NEWSLETTER

WHO Collaborating Center For Mycotoxins In Food

Volume 1 - Number 1 January 1994

INTRODUCTION

The Editorial Board of The World Health Organization (WHO) Collaborating Center For Mycotoxins In Food is pleased to present this first issue of its newsletter, MYCOTOXINS IN HUMAN HEALTH.

Mycotoxins in human health is a new field in medicine. It brings to the scientific world, particularly the medical community, a new etiologic concept which implicates the toxicogenic fungi and their specific toxins (mycotoxins) as the cause of the major non-communicable diseases.

DEFINITION OF MYCOTOXIN: THE FOOD/DRINK CONNECTION

The term mycotoxin is derived from the Greek words "mykes", meaning fungus, and "toxicum", meaning toxin or poison.

Mycotoxin is the generic term used to describe a fungal-produced toxic metabolite which can cause disease leading to death. The modes of entry are ingestion, inhalation, or skin contact.

Fungal invasion of stored foods, particularly grains and nuts, can result in significant production of mycotoxins. Humans who eat these foods are ingesting both the toxicogenic fungi and their mycotoxins. These fungi are capable of surviving in the intestinal stream where they may continue to produce their toxins.

Similarly, animals fed fungal-colonized/ mycotoxic feed are not only at risk for developing mycotoxicoses, their meat and their fat, constitute another vehicle for human exposure to excessive mycotoxin intake. Animal fat is increasingly being documented to be a major risk factor for a number of human cancers and atherosclerosis.

The fungal fermentation processes, such as making bread, beer, wine, cheese, smoking/ chewing tobacco, aging/curing meats, etc., constitutes yet another part of the human food chain which places humans at potential risk. Bread has been epidemiologically incriminated as a cause of breast cancer in Japan and atherosclerosis in the United States.

Alcoholic beverages correlate not only with cirrhosis of the liver, but a wide range of other diseases which including brain damage, cancers, fetal injury, etc. Alcohol is a fungal-produced toxic metabolite and the conditions that is produces are as much mycotoxicotic in nature as ergotism or aflatoxicosis.

Cured mutton consumed by women at the time of conception results in the birth of diabetic infants, a fact not yet taken into consideration in efforts to find the cause of the markedly increasing incidence of this disease in some parts of the world.

KLINIKUM DER ALBERT-LUDWIGS-UNIVERSITÄT FREIBURG
MEDIZINISCHE UNIVERSITÄTSKLINIK UND POLIKLINIK

FUNGAL/MYCOTOXIN-INDUCED DISEASES
A number of the various diseases related to fungi/mycotoxins are summarized TABLE 1 and TABLE 2.

These clinical entities include the major "degenerative diseases" such as atherosclerosis, a number of specific malignancies, the major types of arthritis including gout, all of the "autoimmune diseases", hypertension, cirrhosis, nephritis and diabetes.

FUNGI/MYCOTOXINS-THE MISSING ETIOLOGICAL LINK
The remarkable feature of all of these quite different diseases is that the actual cause of each condition has escaped full elucidation despite extensive research activities.

Even when a good causal relationship is well documented, such as that of tobacco and lung cancer, debate still continues as to the actual mechanism. In the case of tobacco, it has been found that fresh tobacco does not possess any significant carcinogenicity, while after being cured (fungal-fermented), it does.

The fact that cigarette tobacco contains several cancer-producing mycotoxins has not been fully appreciated.

The postulate is that direct contact between the cigarette tar and the bronchial lining initiates the lung cancer. However, this does not explain the relationship between smoking and other tobacco-related cancers which develop in organs located some distance away from the respiratory tract. The presence of absorbable carcinogenic mycotoxins in tobacco does provide a much more realistic explanation.

The above described relationship between cured/stored/processed/mycotoxic tobacco leaves and the etiology of cancer, provides a template of the known relationship of other stored foods to various human diseases.

SURGEON GENERAL'S FOOD LINK TO DISEASE-NUTRIENTS OR MYCOTOXINS
It is the position of the WHO Collaborating Center For Mycotoxins In Food that there is validity of the United States Surgeon General's published data relating foods to all of the major non-communicable causes of death. However, it must be noted that this unique relationship has not been fully explained from foods' nutrient perspective.

This WHO Collaborating Center takes the position that, based upon the already published mycotoxin literature, the Surgeon General's report becomes quite logical when viewed from the new perspective of food contamination by the toxicogenic fungi and their specific mycotoxins.

AN IMPOSSIBLE HUMAN EXPERIMENT-FEEDING HUMANS MYCOTOXINS
There is a major stumbling block to proving that the ingestion of mycotoxins causes any disease in humans; we can never conduct an experiment of feeding humans mycotoxins.

To place the toxicity of mycotoxins in proper perspective, it is necessary to point out that the mycotoxins are potent poisons. Poisonous mushrooms are actually mycotoxin-containing fruiting bodies of underground fungi.

Sporidesmin, a mycotoxin produced by the fungus Pithomyces chartarum, is one of the most poisonous agents discovered, second only to the venom of the infamous orange frogs of South America. In 1981, sporidesmin caused the death of a very large number of sheep, as well as other farm animals, in New Zealand. The economic loss of 100 million dollars significantly reduced the gross national product of the country. Furthermore, the lipid-containing vascular lesions and low density lipoprotein (LDL) hyperlipidemia found in the afflicted sheep resembled the human atherosclerosis/LDL hyperlipidemia complex.

Equally significant, the sheep disease responded to cholestyramine therapy as does human atherosclerosis/LDL hyperlipidemia. Cholestyramine is also an effective treatment to reduce the degree of toxicity found in experimentally mycotoxin-dosed animals. All of this is more than coincidence; it spells out a triad of a toxin etiology, etiology-induced lesions, and therapy based upon the etiology.

Obviously, the experimental mycotoxin dosing of humans is an experiment which will never be conducted. However, the experiment has inadvertently been performed for us by the use of cyclosporin, a mycotoxin, administered to patients having organ transplantation.

CYCLOSPORIN; THE INADVERTENT HUMAN MYCOTOXIN EXPERIMENT

Cyclosporin causes a remarkable degree of immunosuppression which is sufficient for the host to accept someone else's organ. Follow up of such patients has revealed that every long-term surviving patient develops a number of life-threatening complications entirely unrelated to the status of the patient before undergoing transplantation.

CYCLOSPORIN MYCOTOXICOSIS-INDUCED ATHEROSCLEROSIS, HYPERTENSION, GOUT AND VARIOUS CANCERS

The administration of high enough doses of the mycotoxin cyclosporin to injure the immune status of transplantation patients causes severe hyperlipidemia and markedly accelerated atherosclerosis in every long term surviving patient. Most of these patients also develop hyperuricemia with many of them demonstrating gout. In addition, these patients develop hypertensive cardiovascular disease and a wide range of malignancies.

Here is the human model of a cyclosporin mycotoxicosis causing a significant number of the diseases which the pertinent literature is identifying as having a fungal/mycotoxin etiology.

This cyclosporin mycotoxicosis, with its mycotoxin-induced diseases, is <u>not</u> a postulate or a matter of drug side effects. It is absolute proof of a mycotoxin causing disease states in humans which can and do result in the death of these patients.

It is also interesting to note that decreasing the dose and length of treatment with cyclosporin decreases the incidence of these dire complications; an observation consistent with the mycotoxin exposure studies in animal models.

Table 1. MYCOTOXICOSES IN WHICH EXPERIMENTAL AND/OR EPIDEMIOLOGIC DATA RELATE TO HUMAN RISK

DISEASE	SPECIES	FOOD/FEED	MYCOTOXIN
Atherosclerosis/ Hyperlipidemia	Sheep	Pasture	Sporidesmin
	Man	Drug	Cyclosporin
	Man	Bread	S. cerversiae
	Primates	Corn	Fumonisin
Cardiac Ischemia with Arrhythmias	Rabbit		Citreoviridin/ Penicillium
Vasculitis	Man		Penicillin
Hypertension	Man		Alcohol
	Rat		T-2 Toxin
Gout/Hyper-uricemia	Fowl	Moldy Corn	Ustilago maydis
	Fowl	Barley	Ochratoxin
	Chicks		Kojic acid
	Chickens		Oxalic acid
	Pigeons		Alloxan
	Primates		Aflatoxin
	Man	Drug	Cyclosporin
	Man	Drug	Penicillin
	Man	Beer/Wine/Bread	Multiple
	Man	Meat Products	Multiple
	Man	Rye	Ergotamine
Multiple Sclerosis	Man	Rye?	Ergot?
Pulmonary Hypertension	Swine	Grains	T-2 Toxin
Scleroderma	Man	Poison Mushroom	Amanita
Diabetes	Man	Cured Mutton	Multiple
	Rats		Alloxan
Crohn's Disease	Man	Fermentation	S.cerversiae
		Stored Food	Aflatoxin
Lung Cancer	Man	Tobacco	Fusarium Toxins
Lymphoma	Man	Drug	Cyclosporin
Kaposi Sarcoma	Man	Drug	Cyclosporin
Malig. Melanoma	Man	Drug	Cyclosporin
Carcinomas	Man	Drug	Cyclosporin
Esophageal carcinoma	Man	Grains	Fusarium Toxins
Breast Cancer	Man	Bread	S. cerversiae
Endometrial CA	Man	Grains	Fusarium
Colon CA	Man	Grains	Fusarium
Hepatocellular Carcinoma	Man	Grains, Peanuts	Aspergillus Aflatoxin
Hepatoma	Man	Peanuts	Aflatoxin
Cardiomyopathy	Man	Fermentation	Alcohol
Osteoporosis	Man	Fermentation	Alcohol
Ergotism	Man and Animals	Rye, Cereal	Ergot
Vascular Psychosis	Man		
Balkan nephro-pathy	Man	Grains	Ochratoxin
IGA Nephropathy	Mice		Vomitoxin
Reye's Syndrome	Man	Grains	Aflatoxin
Psychosis	Man	Fermentation	Alcohol

Table 2. FUNGAL/MYCOTOXIN-POSTULATED DISEASES BASED UPON ANTIFUNGAL RESPONSIVENESS

COLCHICINE-RESPONSIVE:
- Acute Gouty Arthritis
- Alcoholic Cirrhosis
- Familial Mediterranean Fever
- Mollaret's Meningitis
- Bechet's Syndrome
- Psoriasis
- Thrombocytopenic Purpura
- Chronic Lymphocytic Leukemia
- Amyloidosis North African
- Leukocytoclastic Vasculitis
- Sarcoid Arthritis
- Rheumatoid Arthritis (some)
- Calcium Pyrophosphatopathy
- Hyperlipidemia

COLCHICINE PREVENTS IN EXPERIMENTAL ANIMALS:
- Atherosclerosis
- Casein-Induced Amyloidosis

NYSTATIN-RESPONSIVE:
- Hyperlipidemia
- Inflammatory Bowel Disease
- Hyperactivity Syndrome
- Multiple Sclerosis

GRISEOFULVIN-RESPONSIVE:
- Atherosclerosis (Angina)
- Systemic Sclerosis
- Raynaud's Syndrome/Disease
- Shoulder-Hand Syndrome

ALLOPURINOL-RESPONSIVE:
- Sarcoidosis
- Oxalate Nephrolithopathy
- Idiopathic Respiratory
- Fetal Distress Syndrome
- Duchenne's Muscular Dystrophy

KETOCONAZOLE-RESPONSIVE:
- Disseminated Intravascular Coagulation
- Precocious Puberty in Boys
- Hyper-Low Density Lipoproteinemia
- Cushing's Disease
- Secondary Hyper-aldosteronism
- Prostatic Carcinoma
- Female Infertility

Note: The antifungal nature of colchicine and allopurinol has been fully documented. (References Available)

TREATMENT MUST ALWAYS MATCH THE ETIOLOGY

While the data supporting the etiological arm of this new fungal/mycotoxin discovery is being presented in this and future issues of the Newsletter, emphasis will be also be placed upon the preventive and therapeutic arm of the fungal relationship.

It will be shown that all of the dietary and drug modalities which have been documented to be effective in these fungal/mycotoxin-induced clinical entities share no mode of action except that of being antifungal and/or antimycotoxin. Table 2 clearly demonstrates this simple fact.

What is most remarkable about the data summarized in Table 2 is that no one has ever been able to explain these therapeutic responses. Because the cause of these diseases was entirely unknown, it was left to pure speculation and unproven postulations.

The fact that these agents were antifungal was ignored. Sometimes, the simplicity of truth in science is so simple that it is overlooked due to the complexity of man's thinking process.

REGRESSION OF ATHEROSCLEROSIS WITH ANTIFUNGAL DRUGS (FLUCONAZOLE) (LOVASTATIN)

A neurologist colleague, learning of the fungal/mycotoxin etiology of atherosclerosis, has documented that his own atherosclerosis of the carotid arteries has significantly decreased with the self-administration of fluconazole, an antifungal drug. Of course, he is quite pleased with the results considering that neurologists are the specialists who are usually summoned to see patients with stroke secondary to carotid atherosclerosis.

The case is not strange at all considering the fact that the major drug being used worldwide to treat atherosclerosis is lovastatin, an originally developed antifungal antibiotic. Of course, its antifungal nature has been ignored by all cardiologists who prescribe it.

THE TYPES OF FOOD SUBJECT TO FUNGAL/MYCOTOXIN CONTAMINATION

Table 3 outlines some of the foods and the nature of their fungal colonization and the types of mycotoxins characteristically found on these foods.

JUST HOW FUNGAL-COLONIZED IS OUR STORED FOOD?

The first question which must be answered in order to support a fungal/mycotoxin approach is just how much fungal-colonization of our food chain has been actually documented. Could our food be the source of that much toxic fungi and their multitude of mycotoxins?

If food is loaded with fungi, then the mycotoxin concept is fully operative and the disease-producing potential is more than obvious.

This important question of how much fungal colonization of food exists is answered by a most recent reported mycological study of some quite representative foods; corn kernels, peanuts, cashew nuts and copra (dried coconut). (TABLE 4)

Table 3. NATURAL OCCURRENCE OF MYCOTOXINS IN FOODS

Mycotoxin	Producing fungi	Occurrence
Aflatoxin	Aspergillus flavus, A. paraciticus	Corn, Peanuts, Cotton Feed, Rye, Barley, etc.
Trichothecenes	F. roseum F. tricinctum F. nivale	Corn, Barley
Fumonisin	Fusarium	Corn
Oosporein	Chaetomium Ustilago maydis	Corn
Citrinin	Penicillium citrinum P. veridicatum	Wheat, Barley, Peanuts
Ochratoxin A	A. ochraceus P. veridicatum P. cyclopium	Corn, Barley, Wheat, Bats, Rice
Sterigmatocystin	A. versicolor A. flavus, A. ruber P. lutem	Wheat, Rice, Peanuts
Zearalenone	Fusarium roseum F. moniliforme F. nivale, F. oxysporum	Corn, Sorghum, Wheat
Patulin	A. clavatus P. patulum	Silage, Apples
Penicillic acid	A. clavatus P. puberulum	Corn, Beans
Alternariol, Alternariol monomethyl ether	Alternaria tenuis, A. dauci	Weathered Grain Sorghum Pecan Pickouts
Tenuazonic acid	Alternaria tenuis, A. tamarii, Sphaeropsidales sp., Pyricularia oryzae, Phoma sorghina	Diseased Rice Plants
Ergot alkaloids (ergotamine, etc.)	Claviceps spp., Aspergillus spp., Penicillium spp.	Ergots, Ergot-Infected Pasture Grass
Sporidesmin	Pithomyces chartarum	0.1% in spores on dead pasture grass
PR toxin	Penicillium roqueforti	Silage
Kojic acid	A. flavus, A. oryzae	Moldy corn

THE TROJAN HORSE: FUNGI AND MYCOTOXINS IN THE FOOD CHAIN

Most of us know that food itself cannot be considered poisonous; a few of us know that the toxicogenic fungi and mycotoxins which are characteristically present in stored and fermented food are using our food as a Trojan Horse.

TABLE 4. Food from farmers, middlemen, and retail outlets in Bangkok. Note: Surface was sterilized prior to fungal study.

CORN KERNELS

Fungal infection in 154 samples of surface disinfected maize kernels [a]

Fungus	No. (%) of infected samples	Average % infected particles in infected samples	Range of % infection in infected samples
Acremonium strictum	36 (23)	9	2– 30
Aspergillus flavus	131 (85)	20	2–100
A. niger	99 (64)	7	2– 50
A. tamarii	22 (14)	8	2– 28
A. wentii	31 (20)	9	2– 24
Arthrinium phaeospermum	4 (3)	4	2– 10
Bipolaris maydis	10 (6)	4	2– 12
Chaetomium globosum	18 (12)	6	2– 30
Chaetomium funicola	17 (11)	5	2– 12
Chaetomium spp.	8 (5)	8	2– 30
Cladosporium cladosporioides	6 (4)	2	
Curvularia lunata	15 (10)	5	2– 14
C. lunata var. *aeria*	6 (4)	3	2– 4
Eurotium amstelodami	12 (8)	9	2– 40
E. chevalieri	35 (23)	13	2– 50
E. rubrum	26 (17)	14	2– 38
Fusarium moniliforme	149 (97)	27	2– 68
F. proliferatum	13 (8)	12	2– 24
F. semitectum	70 (45)	8	2– 22
Lasiodiplodia theobromae	89 (58)	12	2– 46
Nigrospora oryzae	42 (27)	11	2– 72
Penicillium citrinum	103 (67)	10	2– 60
P. funiculosum	65 (42)	10	2– 56
P. pinophilum	8 (5)	11	2– 32
P. raistrickii	2 (1)	8	4– 12
Phoma spp.	14 (9)	4	2– 12
Rhizoctonia solani	41 (27)	11	2– 64
Rhizopus oryzae	32 (21)	5	2– 28
R. arrhizus	5 (3)	6	2– 10
Syncephalastrum racemosum	8 (5)	7	2– 20
Trichoderma harzianum	19 (12)	9	2– 28
Wallemia sebi	4 (3)	16	6– 20
Samples infected	154 (100)	88	32–100

[a] Other species isolated at low frequency were *Absidia corymbifera*, *Aspergillus clavatus*, *A. nanus*, *A. ochraceus*, *A. parasiticus*, *Botryotrichum piluliferum*, *Curvularia clavata*, *C. eragrostidis*, *C. oryzae*, *C. pallescens*, *Eurotium repens*, *Exophiala jeanselmei*, *E. castellanii*, *Fusarium equiseti*, *F. longipes*, *Geotrichum candidum*, *Mucor circinelloides*, *M. hiemalis*, *M. piriformis*, *Penicillium crustosum*, *P. glabrum*, *P. minioluteum*, *P. oxalicum*, *P. raistrickii*, *P. purpurogenum*, *P. variabile*, *Phoma herbarum* and *Stemphylium botryosum*.

COPRA

Fungal infection in 21 samples of surface disinfected copra [a]

Fungus	No. (%) in infected samples	Average % infected particles in infected samples	Range of % infection in infected samples
Aspergillus candidus	1 (5)	20	
A. clavatus	3 (14)	23	2– 46
A. flavus	18 (86)	23	2– 73
A. niger	9 (43)	42	3– 86
A. sydowii	1 (5)	22	
A. tamarii	5 (24)	8	6– 14
A. versicolor	1 (5)	22	
Chaetomium globosum	4 (19)	4	2– 6
C. funicola	1 (5)	28	
Chaetomium spp.	3 (14)	6	2– 14
Corynascus sepedonium	1 (5)	3	
Endomycopsis fibuliger	4 (19)	11	8– 14
Eurotium amstelodami	3 (14)	16	12– 20
E. chevalieri	9 (43)	31	2– 80
E. repens	4 (19)	15	5– 30
E. rubrum	8 (38)	29	5– 83
Mucor spp.	3 (14)	35	10– 83
Nigrospora oryzae	6 (29)	6	2– 14
Penicillium citrinum	8 (38)	18	2– 48
Pestalotiopsis guepinii	1 (5)	11	
Phoma sp.	1 (5)	6	
Rhizopus oryzae	11 (52)	48	14– 98
Sordaria fimicola	8 (38)	29	3– 50
Samples infected	21 (100)	76	6–100

[a] Other fungi isolated at low frequency were *Arthrinium phaeospermum*, *Aspergillus japonicus*, *A. nanus*, *Chaetomium brasiliense*, *Cladosporium cladosporioides*, *Exophiala castellanii*, *Humicola grisea*, *Macrophomina phaseolina*, *Penicillium aurantiogriseum*, *Penicillium citrinum* and *Syncephalastrum racemosum*.

Source: Pitt JI, Hocking AD, Bhudhasamai K, Miscamble BF, Wheeler KA, Tanboon-Ek P: The normal mycoflora of commodities from Thailand. 1. Nuts and oilseeds. International Journal of Food Microbiology

PEANUTS

Fungal infection in 109 samples of surface-disinfected peanut kernels [a]

Fungus	No. (%) of infected samples	Average % infected particles in infected samples	Range of % infection in infected samples
Aspergillus candidus	4 (4)	14	2–50
A. flavus	103 (95)	44	2–100
A. niger	94 (86)	38	3–100
A. tamarii	34 (31)	11	2–40
A. wentii	22 (20)	22	2–80
Chaetomium globosum	9 (8)	2	
C. funicola	5 (5)	9	2–20
Chaetomium spp.	10 (9)	5	2–28
Cladosporium cladosporioides	16 (15)	3	2–40
Eurotium amstelodami	10 (9)	16	2–24
E. chevalieri	50 (46)	33	4–100
E. repens	7 (6)	11	4–20
E. rubrum	56 (51)	28	2–85
Fusarium equiseti	11 (10)	4	2–14
F. semitectum	21 (19)	4	2–10
F. solani	3 (3)	6	2–10
Lasiodiplodia theobromae	36 (33)	12	2–40
Macrophomina phaseolina	53 (49)	16	2–55
Nigrospora oryzae	24 (22)	4	2–16
Penicillium aethiopicum	4 (4)	15	4–24
P. aurantiogriseum	6 (5)	36	2–100
P. brevicompactum	2 (2)	10	4–16
P. citrinum	50 (46)	14	2–60
P. funiculosum	15 (14)	18	2–92
P. glabrum	3 (3)	6	5–10
P. janthinellum	3 (3)	13	4–30
P. olsonii	9 (8)	5	2–12
P. pinophilum	4 (4)	17	8–28
Rhizopus oryzae	65 (60)	25	2–95
Syncephalastrum racemosum	9 (8)	10	2–30
Wallemia sebi	13 (12)	42	18–98
Samples infected	109 (100)	84	6–100

[a] Other fungi isolated at low frequency were *Acremonium phaeospermum*, *Aspergillus awamori*, *A. nanus*, *A. japonicus*, *A. niveus*, *A. terreus*, *A. ochraceus*, *A. parasiticus*, *A. sydowii*, *A. terreus*, *A. versicolor*, *Cladosporium cladosporioides*, *Curvularia lunata*, *Fusarium moniliforme*, *F. oxysporum*, *Geotrichum candidum*, *Gliocladium* sp., *Malbranchea sulfurea*, *Penicillium chrysogenum*, *P. expansum*, *P. griseofulvum*, *P. pinophilum*, *P. waksmanii*, *Rhizopus stolonifer* and *Trichoderma harzianum*.

CASHEW NUTS

Fungal infection in 45 samples of surface disinfected cashew kernels [a]

Fungus	No. (%) of infected samples	Average % infected particles in infected samples	Range of % infection in infected ssamples
Alternaria alternata	3 (7)	2	
Aspergillus flavus	27 (60)	8	2–35
A. niger	24 (53)	10	2–66
A. sydowii	5 (11)	18	4–70
A. tamarii	3 (7)	3	2– 4
A. wentii	4 (9)	15	5–28
Chaetomium globosum	21 (47)	7	2–30
C. brasiliense	5 (11)	5	2–10
C. funicola	6 (13)	5	2–10
Chaetomium spp.	4 (9)	9	2–20
Chrysonilia sitophila	1 (2)	32	
Cladosporium cladosporioides	17 (38)	6	2–22
Curvularia lunata	2 (4)	2	
Epicoccum nigrum	3 (7)	10	5–15
Eurotium amstelodami	7 (16)	20	2–35
E. chevalieri	18 (40)	8	2–24
E. rubrum	14 (31)	20	2–90
Nigrospora oryzae	26 (58)	8	2–22
Penicillium aurantiogriseum	1 (2)	30	
P. citrinum	13 (29)	7	2–20
P. implicatum	2 (4)	19	10–28
P. olsonii	3 (7)	30	8–40
P. solitum	2 (4)	6	2–10
Phoma spp.	5 (11)	5	
Rhizopus oryzae	5 (11)	11	8–15
Syncephalastrum racemosum	3 (7)	20	2–30
Wallemia sebi	4 (9)	12	10–16
Samples infected	45 (100)	40	6–90

[a] Other fungi isolated at a low frequency were *Acremonium strictum*, *Aureobasidium pullulans*, *Aspergillus candidus*, *A. japonicus*, *A. ochraceus*, *Botryotrichum piluliferum*, *Cladosporium macrocarpum*, *Curvularia affinis*, *C. lunata* var. *aeria*, *Cylindrocarpon pseudofaginum*, *Paecilomyces variotii*, *Penicillium aurantiogriseum*, *P. brevicompactum*, *P. chrysogenum*, *P. citrinum*, *P. implicatum*, *P. janthinellum*, *P. olsonii*, *P. solitum*, *P. waksmanii*, *Phoma* sp., *Rhizopus stolonifer* and *Trichoderma harzianum*.

THE FORMAT OF FUTURE ISSUES OF THE NEWSLETTER

The future issues of the Mycotoxins in Human Health Newsletter, will present data supporting the concept of fungal/mycotoxin-induced diseases. In addition, the results of the WHO Mycotoxin Collaborators testing of the mycotoxin content in the blood of patients with various disease states will be presented.

THERE ARE MYCOTOXINS FOUND IN HUMAN BLOOD AND BREAST MILK

In respect to the presence of mycotoxins in humans, it has already been documented by several of our collaborators that over half of German adults have ochratoxin in their blood, that leukemic children have aflatoxin in their blood, that patients with urinary tract cancers have ochratoxin in their blood, that patients with Crohn's Disease have aflatoxin in their blood, and finally, a small percentage of nursing mothers have mycotoxins in their breast milk.

Obviously, the problem of mycotoxins in human health is quite real and requires full elucidation, particularly since we all know that food is in some way connected to the major disease of humans.

THE COLLABORATING TEAM

The WHO Collaborating Center For Mycotoxins In Food was created as a multi-disciplined, multinational task force to more fully elucidate the problem of mycotoxins in human health. The Center is located at the Medical School of the Albert Ludwigs University in Freiburg, Germany.

The Executive Committee is composed of the Head of the Center, A.V. Costantini, M.D., its Medical Director Heinrich Wieland, and its Co-Medical Director, Lars I. Qvick, M.D.

The Advisory Board is in the process of being expanded. Its present members include Dr. Johann Bauer, Drs. John Richard, William Norred of the U.S. Department of Agriculture (USDA), Drs. Same Page and Lori Love of the U.S. Food and Drug Administration. We are in the process of inviting representatives from a number of other governmental agencies and research institutions to serve on the Advisory Board.

Individual mycotoxin researchers throughout the world are being invited to participate as collaborators as well as individual physicians who deal with the various mycotoxin-related disease entities.

The Center will be working closely with the staff of the WHO Food Safety Unit. The results of the thus planned multi-disciplined research activities will be reported to the World Health Organization for its appropriate review and subsequent dissemination worldwide through the many Health Ministers who constitute the active membership of the WHO.

IN OUR NEXT ISSUE:
THE FUNGAL-MYCOTOXIN ETIOLOGY OF CROHN'S DISEASE

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