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DEFINITION OF FUNGALBIONICS

The term FUNGALBIONICS was created in an attempt to describe one of the most dynamic microbial chemical factories ever encountered in the history of scientific exploration: the fungus

Fungi are masters at producing a wide array of biologically active substances which serve the producing fungus extremely well. These biological metabolites are anti-predatory, i.e. territory-protective, and exist to ensure that the fungus will survive as long as possible in this quite hostile world.

These metabolites are anti-viral, anti-bacterial, anti-protozoan, anti-insect, anti-animal and, of course, anti-human. These metabolites are referred to as mycotoxins. The term is derived from the Greek *mykes*, meaning fungus, and *toxicum*, meaning toxin or poison.

Mycotoxins Are Poisons.

One could test the validity of this most biologically potent fungal reality by eating a cupful of poison mushrooms, a species of fungus. However, it would be less fatal to simply read about the deadly effect which they have on humans and all other animals. For that mushroom, the name of the game is food, for in nature the animal which nibbles on them dies and is slowly consumed by the (root-like) mycelia under the ground which grow up into the hapless and dead creature. Many plants meet this same fate.

Thus, the term FUNGALBIONICS attempts to convey this remarkable degree of biological activity which these simple single-celled fungi demonstrate. *All* fungi are so empowered, some less against humans, some more so. While fungi are potentially our enemies, some of their mycotoxins, such as penicillin, have proven to be beneficial to humans who suffer from bacterial infections or other diseases.

The "bionic" nature of fungi is seen by the magnificent power to save human lives from bacterial infection which penicillin has demonstrated. That is indeed a bionic miracle. Other fungal-derived drugs are just as miraculous, as will be described later.

This series of FUNGALBIONICS books provide documentary evidence that fungi and their biological metabolites, the mycotoxins, are silent and relentless attackers of human health in that they cause the major "degenerative" and "cancerous" diseases which plague mankind.

FUNGALBIONICS appears to be a most appropriate term to describe the fungal/mycotoxin findings which will be presented in the following pages. It is a term which the physician author and his research associates have found to be acceptable. We hope that the reader will agree with us.

WHAT ARE FUNGI?

Fungi are single celled living forms of life which inhabit the land, air and waters of the earth. They are everywhere. They are more highly developed than the bacteria and viruses, and there are many more species among the fungi than are found among the other microbes. It is estimated that there are over 500,000 different species.

Fungi have existed on earth hundreds of millions of years and, quite remarkably, have experienced little genetic change during that period of time. They are survivalists. Viable fungi can grow from spores which have been dormant for thousands of years, such as has been observed in spores which were found in Egyptian tombs.

Single fungal cells can only be seen under the microscope, but a colony of these cells makes a visible presence in the form of mushrooms, toad stools and molds on food or elsewhere. While plants, animals and humans are alive and well, the fungi around them are unable to overcome the natural defense mechanisms which higher forms of life possess. Once death of the living organism has occurred, however, the fungi become the principle undertakers and managers. They reduce all that has ever lived into the molecules from which they were assembled. Biologists call this the carbon cycle, while Christians describe it as „dust to dust“.

Unfortunately, though, there is one exception to this simple balanced equation of life and death and that is that the fungi can also attack the living *while* they are alive. At its most simplistic perspective, one has many fungi entering the intestinal tract, the nose and lungs, and organs exposed to the outside world. Though we generally do not develop an infection from such intrusions, some persons might contract a fungal infection such as "athlete's foot" or "ring worm" on the skin.

At the opposite extreme is the patient with AIDS who faces major life-threatening fungal infections because the immune system has lost its ability to protect the body from organisms which invade the body, such as fungi. In between these extremes are fungal infections associated with diseases such as diabetes, cancer as well as conditions which include cross infections amongst humans. Fortunately, though, the average person does not succumb to a serious fungal infection and the average life expectancy extends into the 70's.

All humans are colonized by *Candida albicans* and normal healthy persons do not die from this organism. This organism plays a very little role in causing human diseases.

(The concept that *Candida* causes many diseases is NOT a part of Fungalbionics nor is it supported by the extensive medical literature relative to *Candida*.)

WHAT ARE MYCOTOXINS?

All physicians are familiar with fungal infections and the drugs used to treat them. With the exception of poisonous mushrooms, which are deadly to those foolish enough to eat them, few physicians are aware of the fact that fungi make toxins.

MYCOTOXINS-FRIENDS?... OR ENEMIES?... THEY ARE BOTH...**AS ENEMIES**

As many as 1,000 compounds, classifiable as mycotoxins, were studied by the pharmacology industry as potential antibiotics in the 1930s and 1940s only to be discarded as being too toxic for higher life forms to be of value in treating bacterial diseases in humans. Little, if any of the discarded data was published. Yet what these toxicity studies actually documented was the existence of a large number of fungal-derived toxins which caused serious target organ injury in various animal models. Obviously, in retrospect, what was being seen was the pathology produced by the mycotoxins. In order to understand this toxicity, one only has to look at what some of these mycotoxins, used as medications, causes in humans:

The mycotoxin cyclosporin used for Transplantation causes cancer and atherosclerosis, complete with hyperlipidemia, in ALL humans who have received it. Many others develop gout and other diseases.

AS FRIENDS

However, to place the matter in proper perspective, the study of such fungal metabolites gave us penicillin at the beginning, quite later on cyclosporin, the most potent immunosuppressant Transplantation drug, lovastatin, and the other statins which have revolutionized the treatment of hyperlipidemia and atherosclerosis. The latter group is quite interesting in that they were initially developed as antifungal agents which just happened to have an effect in lowering blood levels of low density lipoproteins (commonly referred to as "bad cholesterol").

The members of this group of drugs are joined by another antifungal antibiotic, griseofulvin, which is also a remarkably efficient anti-atherosclerosis drug. All of this goes a long way to confirm the fungal etiology of atherosclerosis. This appears to be a quite valid conclusion since all of the other effective anticholesterol and/or anti-atherosclerotic therapeutic modalities share nothing in common except that they possess antifungal and/or antimycotoxin activity.

DISEASES RESPONDING TO ANTIFUNGAL DRUGS

The Fungalbionic Series of Books present data documenting the fungal/mycotoxin cause of a number of diseases. Equally important, the series also documents that each and every dietary measure or drug found to be effective in treating these diseases share nothing in common except that they are all antifungal and/ or antimycotoxic.

The importance of this therapeutic responsiveness should not be underestimated. If a cause of a disease is a microbe, it must respond to an appropriately selected antimicrobial agent.

In addition, diseases of unknown etiology which respond to antifungal-effective drugs suggest the probability that they have a fungal origin, particularly when there is no other proven explanation as to how the drug is working. Table 1 provides a number of human diseases which so respond and suggest a fungal/mycotoxin origin.

Table 1
FUNGAL/MYCOTOXIN-RELATED DISEASES

COLCHICINE-RESPONSIVE:	GRISEOFULVIN-RESPONSIVE:
Acute Gouty Arthritis	Atherosclerosis (Angina)
Alcoholic Cirrhosis	Systemic Sclerosis
Familial Mediterranean Fever	Raynaud's Syndrome/Disease
Mollaret's Meningitis	Shoulder-Hand Syndrome
Bechet's Syndrome	
Psoriasis	
Thrombocytopenic Purpura	ALLOPURINOL-RESPONSIVE:
Chronic Lymphocytic Leukemia	Sarcoidosis
Amyloidosis North African	Oxalate Nephrolithopathy
Leukocytoclastic Vasculitis	Idiopathic Respiratory Distress

Sarcoid Arthritis Rheumatoid Arthritis (some) Calcium Pyrophosphatopathy Hyperlipidemia Inflammatory Bowel Disease	Syndrome/Newborns Duchenne's Muscular Dystrophy
COLCHICINE-RESPONSIVE IN EXPERIMENTAL ANIMALS:	KETOCONAZOLE-RESPONSIVE:
Atherosclerosis Casein-Induced Amyloidosis Cushing's Disease	Inflammatory Bowel Diseases Disseminated Vascular Coagulation Idiopathic Female Infertility Precocious Puberty In Boys Hyper-Low-Density Lipoproteinemia Hyperaldosteronism Prostatic Carcinoma
NYSTATIN-RESPONSIVE:	TAMOXIFEN-RESPONSIVE:
Psoriasis Inflammatory Bowel Disease Hyperactivity Syndrome Multiple Sclerosis	Breast Cancer
TAXOL-RESPONSIVE:	<i>Note: The antifungal nature of colchicine, allopurinol, tamoxifen, and taxol is fully documented.</i>
Breast Cancer Ovarian Cancer	

THE TROJAN HORSE: FUNGI AND MYCOTOXINS IN THE FOOD CHAIN

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

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 really be the source of that much toxic fungi and mycotoxins?

If, in fact, food is contaminated 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 recent reported mycological study of some representative foods: corn kernels, peanuts, cashew nuts and copra (dried coconut). Table 2 demonstrates the remarkable degree to which the *interior* of corn kernels and peanuts can be colonized by fungi. Humans who eat these foods are ingesting both the toxicogenic fungi and their mycotoxins. The fungi themselves are capable of surviving in the intestinal stream where they can continue to produce their toxins. (Table 2 not included in this excerpt)

Similarly, animals fed fungal-colonized/mycotoxin feed are not only at risk for developing mycotoxicoses, their meat, their milk and their fat constitute another pathway through which human exposure to mycotoxins can occur. Animal fat, coincidentally, is increasingly being documented to be a major risk factor for a number of human cancers and atherosclerosis.

Mycotoxins have been documented to cause a number of specific types of diseases and very specific organ lesions both in animals and in humans. Table 3 provides a summary of some of this documentation. (Table 3 not included in this excerpt) 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 recently 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 includes brain damage, cancers, fetal injury, etc. Alcohol is a fungal-produced toxic metabolite and the conditions that it produces are as much mycotoxicotic in nature as egotism or aflatoxicosis.

Cured mutton consumed by women at tile time of conception results in the birth of diabetic infants. This is 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.

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, 18 to 90 % 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.

DIET CHOICES INCREASE OR DECREASE THE MYCOTOXIN CONNECTION

Public awareness is particularly important in that the major means of preventing the development of these diseases rests most significantly upon the informed/Intelligent selection of what the public eats and drinks.

A person's dietary choices play the critical role in the causation or in the prevention of all of the mycotoxin-caused diseases, not only for himself, but also for his offspring.

The selection of foods for children is going to determine the life expectancy and quality of health for these adults-to be.

The dietary choices required for controlling the degree of mycotoxicity are all based upon documented facts found in the scientific literature. The diet must reduce the intake of mycotoxin-containing foods, not feed the fungi living within us, and decrease the toxic of the mycotoxins which do enter our body.

DIETARY AVOIDANCE OF MYCOTOXIN-CONTAINING FOODS

Reduce the intake of fungal toxins which are present in stored grains, nuts, seeds, meats, grain-fed animal products (meat, animal fats, butter, whole milk) and fermented foods such as beer, bread, cheese and wine.

DIET CONTROL OF MYCOTOXIN-PRODUCING FUNGI IN THE HUMAN BODY

Fungal toxins are constantly being absorbed from toxin-producing fungi living in the host, particularly in the gut.

Increased fungal growth/toxin production is caused by diets high in sugar, fruit, oils, fats, and fermented foods such as beer, wine, bread and cheese.

A decreased fungal growth/toxin production is due to the antifungal action of fish/ fish oils, garlic, onion, herbs, spices, soya, yogurt and green vegetables.

DIETARY CONTROL OF DEGREE OF TOXICITY CAUSED BY MYCOTOXINS

Toxicity caused by mycotoxins is significantly reduced by increasing the amount of fiber in the diet. This is done by increasing the amount of vegetables in the diet. While fruit is also a source of fiber, the high sugar (fructose) content of fruit stimulates fungal growth (fructose increases blood cholesterol and uric acid levels which are associated with increased risk of hypertension and atherosclerosis).

MEDICATIONS

Unlike the other dietary approaches to the prevention and treatment of human diseases, the mycotoxin concept does not exploit the adverse effects of drugs in an attempt to support a diet-only attitude. It should be noted that almost all medications are plant derived or chemical derivatives thereof. Aspirin derives from the bark of the willow tree. Colchicine derives from a plant. Both aspirin and colchicine possess significant antifungal activity as do most plant-derived drugs. (They protect living plants from the fungi.)

Similarly, all of the other anti-inflammatory drugs possess significant antifungal activity. These drugs are cyclo-oxygenase inhibitors and fungal survival is dependent upon the competency of their cyclo-oxygenase-related metabolic pathways.

Interestingly, corticosteroids not only significantly reduces the toxicity of mycotoxins but are also antifungal against a number of fungi.

Actually, all of the medications proven to be effective in the treatment of the mycotoxin-Induced diseases possess antifungal and/or antimycotoxin activity. It is a point overlooked by pharmacologists.

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Chapter 28

BREAST CANCER: HOPE AT LAST

- **THE CAUSE OF BREAST CANCER
PREVIOUSLY UNKNOWN**
- **THE CAUSE OF BREAST CANCER
DISCOVERED TO BE FUNGI/MYCOTOXINS**
- **BREAST CANCER IS NOT GENETIC; IT'S A
FOOD CONTAMINATION DISEASE**
- **WOMEN CAN NOW PREVENT BREAST CANCER**

CLINICAL PERSPECTIVE

The Cause Of Breast Cancer Has Been Unknown

It is clearly stated in medical textbooks that the cause of breast cancer is unknown.

There are risk factors which are found in the history of some, but not all, patients who develop breast cancer. The problem of understanding the true role of these risk factors is complicated by the fact that they are not consistently found in all breast cancer studies. Furthermore, there is no explanation as to precisely how these risk factors are involved in the entire cancer process. Clearly, something is missing.

THE REPORTED CLINICAL FACTS

Hopeless Nature Of Most Breast Cancer Risk Factors

Colditz (1993) reviewed the epidemiology of breast cancer "in the context of hormonal, hereditary, histologic, and dietary risk factors." The article provides a review of the pertinent breast cancer literature.

The conclusion of the review article is quoted here, for it provides the reader with a clear view of the helpless position in which the medical profession finds itself when dealing with breast cancer:

"Few of these associations offer the potential for intervention to reduce the breast cancer risk."

CLINICAL PERSPECTIVE

Breast Cancer Is Entirely Preventable Based Upon The Published Data In This Book

The published research data in this book is intended to replace this state of helplessness with a new level of understanding. This book provides the answers to the unanswered questions raised with respect to the issue of breast cancer. It provides the etiology, as well as the preventive measures based upon it which must be taken to prevent breast cancer.

The fact is that cancer can only be conquered when the true cause has been determined and then eradicated.

Fungi/Mycotoxins Are The Cause Of Breast Cancer

The concept of the fungal/mycotoxin cause of breast cancer provides us with the missing piece of the puzzle. Each risk factor is fully explained by a fungal/mycotoxin etiology.

There is no other documented cause of breast cancer. The medical literature is crystal clear: breast cancer is a disease which is not caused by genes, viruses, insecticides, stress or lack of exercise.

Antifungal/Antimycotoxin Nature Of Effective Anti-Breast Cancer Measures

It should be noted that each and every effective (non-surgical) preventive measure and therapeutic agent employed against breast cancer share an antifungal and/or an antimycotoxin mode of action. This includes all chemotherapy drugs and radiation therapy. The anti-breast cancer drug tamoxifen, for example, is antifungal as well.

Role Of Fungi/Mycotoxins In Human Food And Health Ignored By Medical Researchers

Fungi and their toxins (mycotoxins) as a cause of degenerative and cancerous diseases in both animals and humans is a subject which has essentially been ignored by physicians and medical researchers.

The reason for the existence of this blind spot is that the tens of thousands of published articles on fungi and mycotoxins appear in the food science and microbiology literature and only seldom (if ever) in medical journals. Physicians simply have not been exposed to the information on mycotoxins nor to the globally-recognized and serious nature of the carcinogenicity and pathogenicity of these toxins.

Many studies present inconsistencies with respect to risk factors for breast cancer. One example is meat: some studies have shown it as being a risk factor, while others have found no association. These inconsistencies of the various risk factors related to breast cancer are easily explained by the fact that mycotoxins are not always present to the same degree, from time to time, place to place, in any particular food.

Storage of foods and the fermentation processes used to make foods such as bread, cheese, beer, wine and other alcoholic beverages, may be contaminated by variable amounts of fungi and their toxins.

Fresh foods, particularly fresh fish, fresh fruits and vegetables, are relatively free of fungi and mycotoxins because of the protective antifungal metabolites they still contain. Wherever fruits or vegetables are implicated as being a risk factor, further investigation indicates that the quality of the food was compromised by fungal contamination.

Women Can Prevent Breast Cancer

Knowledge of the fungal/mycotoxin etiology of breast cancer allows women to now make the appropriate dietary choices which will prevent the loss of their breast(s) and the premature loss of their lives. If the cancer has already taken hold, these same choices will help to improve the management and outlook of the disease, while simultaneously decreasing the chance that new breast cancers may take hold.

It should be clear both to the patient with breast cancer and every health professional involved in that patient's care, that the causative fungal/mycotoxin type foods must be entirely removed from the diet to prevent a recurrence.

The concept that breast cancer is inherited is based upon a number of reports documenting that patients with this cancer often have a family history of the disease. There is also some genetic research which has become popular in the public media in the past decade.

However, it should be stressed, that most patients do not have a family history of breast cancer nor do they show evidence of a genetic cause for their cancer.

The following sections provide a fungal/mycotoxin explanation for what appears to just be "family history".

THE REPORTED CLINICAL FACTS

Increased Incidence Of Breast Cancer In First-Degree Relatives In Japan

Hirose et al. (1995) conducted a large-scale case-control study to evaluate the differences and similarities in the risk factors of female breast cancer. The study involved 1,186 women with breast cancer.

The incidence of at least one breast cancer case among the subjects' first-degree relatives was relatively high.

Increased Incidence Of Breast Cancer In First-Degree Relatives In American Women

Madigan et al. (1995) evaluated the data from the first National Health and Nutrition Examination Survey which was a probability sample of the U.S. population. 7,508 female participants were surveyed in the early 1970's, and followed up between 1982 and 1984 and again in 1987. 193 breast cancer cases were accrued for study.

It was found that women with a family history of breast cancer had an increased incidence of breast cancer.

Increased Incidence Of Breast Cancer In First-Degree Relatives In Hispanic Families

Mayberry and Branch (1994) reported on a study of risk factors for breast cancer among Hispanic women in a case-control study of 148 subjects.

Women who had a mother or sister with breast cancer were nearly twice as likely to have had breast cancer compared to women with no such family history.

CLINICAL PERSPECTIVE

The increased incidence of breast cancer in first degree relatives of breast cancer patients led to a search for cancer-related genes. This search was rewarded by the finding of gene abnormalities in cancer cells. It is important to stress, however, that these gene findings did not involve normal genes, but rather damaged/mutated ones.

Normal genes cannot cause cancer any more than an elephant's genes can make it fly.

So what the genetic researchers found, then, is that a normal gene was mutated in some breast cancer patients. The finding of a gene mutation must always raise the question of how that gene became mutated in the first place. It also raises the question of whether or not this mutated gene in the familial gene pool is being transferred from the mothers to their daughters. This is unlikely, though, because only a small percentage of related breast cancer patients share identical mutated gene patterns.

It also doesn't fit the clinical reality that environmental factors, particularly stored and fermented foods in the diet, cause breast cancer.

Finally, the virtual absence of breast cancer in several Oriental countries prior to World War II being replaced by a breast cancer epidemic traced to the Western diet is not an inherited genetic phenomenon.

This leaves us with the alternative possibility which is that there is an environmental gene-mutating toxin present in the nourishment which the mother transfers through the placenta to the fetus, in the mother's breast milk during lactation, and, lastly, in the food which the infant will consume during its childhood and later on as an adult.

The P53 Gene Mutation Found In Breast Cancer

Abnormalities in the p53 gene are reported in more than 50% of malignant tumors (Livni et al. [1995]). The cancers involved include those of the lung, breast and colon.

When the increased incidence of breast cancer in family members is viewed in light of an abnormal gene found in some patients, one can readily see how the p53 mutated gene mistakenly leads to the genetic theory of breast cancer.

THE CORRELATIVE FUNGAL / MYCOTOXIN FACTS

The P53 Gene Mutation Due To Aflatoxin

In his 1995 Deichmann Lecture presentation, Harris provides an update of the genetic aspects of cancer. Cancer is a multistage process involving the activation of proto-oncogenes, for example ras, and the inactivation of tumor suppressor genes, such as p53 and p16INK4.

Harris points out that p53 is a prototype tumor suppressor gene that is the most common genetic lesion in human cancers including breast cancer.

In 1991, Harris stated that the p53 mutation is found in hepatocellular carcinomas from both Qidong, People's Republic of China, and southern Africa. Harris continues:

"This observation links exposure to aflatoxin, a known cancer risk factor in these geographic regions, with a specific mutation in a cancer-related gene."

Simply stated, the mutation found in the p53 gene is due to a mycotoxin. (See also Lilleberg [1992].)

Aflatoxin Found In Human Breast Cancer Tissue

Harrison et al. (1993) examined human breast cancer tissue for evidence of the presence of aflatoxin, a recognized potent carcinogenic mycotoxin.

The researchers examined human DNA from a variety of tissues and organs to identify and quantify aflatoxin DNA-adducts. Such adducts are considered to be proof of the mycotoxin's presence in a particular tissue. (These researchers had already proved the value of this method in the detection of aflatoxin-DNA adducts in tissue from a case of acute aflatoxin poisoning in Southeast Asia.)

DNA from normal and tumorous tissue obtained from patients with cancer of the breast was examined. Tumor tissues had higher aflatoxin-adduct levels than did normal tissue from the same individual.

The result of this study is that it verifies the presence of carcinogenic aflatoxin within the cancer tissue and thus implicates aflatoxin as a cause of breast cancer.

Aflatoxin-Induced Liver Cancers In Rats Show P53 Expression

Lilleberg et al. (1992) examined rat hepatocellular carcinomas which had been induced by aflatoxin treatment. The tissues were examined for their changes in the p53 tumor suppressor gene. All of the 11 aflatoxin-dosed animals exhibited mutation of the p53 gene.

When compared with non-tumor liver tissue from the same animal, the tumors with p53 gene alterations showed dramatically reduced levels of p53 mRNA and protein.

It was concluded that, according to the data, alterations of the p53 tumor suppressor gene are involved in the induction of rat liver cancer by aflatoxin.

CLINICAL PERSPECTIVE

The cancer research relative to abnormal genes is quite exciting when it correlates with mycotoxins such as aflatoxin. The erroneous impression of the general public that there is a hereditary genetic cause of cancer must by all means be corrected, for it leads to the fatalistic perception that, if cancer is "inherited", nothing can be done to prevent it.

Cancers Caused By Pre-Natal And Post-Natal Aflatoxin-Exposed Rats

Goettler et al. (1980) reported what appears to be one of the most significant aflatoxin exposure studies ever reported. Pregnant rats were intraperitoneally dosed with aflatoxin for a 3-day period of time and the offspring were dosed for 3 days following delivery. The doubly-exposed offspring developed the following array of benign and malignant tumors:

Benign Tumors
Hepatocellular Adenomas
Cholangiocellular Adenomas
Papillomas of the Stomach
Papillomas of the Intestine
Adenomas of the Stomach
Adenomas of the Intestine
And Of These Other Organs:
Pituitary

Adrenal
Ovaries
Testes
Thyroid
Parathyroid
Pancreas
Central Nervous System
Peripheral Nerves
Skin
Urogenital
Skeletal
Nasal Cavity
Malignant Tumors
Hepatocellular Carcinoma
Stomach Adenocarcinoma
Intestine Adenocarcinoma
Leukemia
And Of These Other Organs:
Adrenal
Thyroid
Pituitary
Central Nervous System
Peripheral Nerves
Skin
Uterus
Vagina
Skeletal
Nasal Cavity

This study mimics the real life situation of a pregnant woman and her baby being exposed to dietary aflatoxin at various stages:

1. During the pregnancy through the placenta via the umbilical cord;
2. Through breast-feeding of aflatoxin-contaminated milk by the mother; and then,
3. By feeding the infant aflatoxin-contaminated commercial infant formula;
4. By feeding the growing child aflatoxin-contaminated food.

The following published human and animal studies document that these 4 aflatoxin exposures do, in reality, occur.

Mycotoxins Found In Umbilical Cord Blood Of Pregnant Women In Sierra Leone

Johnson et al. (1995) analyzed 64 umbilical cord blood samples from pregnant women in Sierra Leone. The analyses revealed the presence of ochratoxin and aflatoxin in 25% and 58% of the samples, respectively. Of eight maternal blood samples collected during delivery, one contained ochratoxin and six contained aflatoxins.

Aflatoxin And Other Mycotoxins Found In Human Breast Milk (Sierra Leone)

Jonsyn et al. (1995) tested breast milk in Sierra Leone for the presence of mycotoxins. Only 10 of 113 breast milk samples were mycotoxin-free. Eighty-eight percent of samples contained various aflatoxins and 35% contained ochratoxin. Very few samples (15%) had only a single mycotoxin. Thirty-six samples (32%) had two mycotoxins; 50 samples (40%) had three or more.

It was concluded that infants in Sierra Leone are exposed to aflatoxins and other mycotoxins at levels which, in some cases, far exceed those levels permissible in animal feed in developed countries.

This is not a problem unique to African nations, as has been all too often claimed by the developed nations. The following report documents that the problem also exists in Europe. This suggests that if the human breast milk in other countries were to be checked, similar findings would be found.

Ochratoxin Found In Human Breast Milk From Nursing Italian Mothers

Micco et al. (1991) studied the possibility that ochratoxin could be transmitted in human milk from mother to child during breast-feeding.

Fifty samples of human milk were collected randomly over one year from Italian nursing mothers and analyzed for ochratoxin. Nine samples (18%) were found to contain ochratoxin.

Aflatoxin In Breast Milk Causes Liver Cancer In Young Rats

Several published studies on breast-fed rats have shown cases of liver cancer in offspring as a result of the ingestion of aflatoxin from the breast milk (Grice et al. [1973]; Wakhisi [1989]).

Human Infant Exposure To Aflatoxin Causes Liver Cancer In Young Adults

Van Rensburg et al. (1985) found a high incidence of primary liver cancer in young males and females (20-30 years old) in Inhambane Province, Mozambique. It had been postulated that one factor contributing to this early onset of the disease could be exposure to environmental carcinogens at or soon after birth (Wild et al. [1987]).

The presence of various mycotoxins, especially a highly toxic one like aflatoxin, in human breast milk provides proof of very early exposure of humans to mycotoxin carcinogens.

Aflatoxin In Infant Powdered Milk

Jesenska and Polakova (1978) studied the problems of the presence of potential mycotoxin producing fungi in powdered milk preparations marketed for infants. The study was prompted by the finding of aflatoxin in the livers of deceased children and in some samples of milk powder.

Commercial samples of domestic and foreign milk powder intended for babies were examined and, as expected, 29 different species of molds were isolated. The authors concluded that these results must lead to a revision of views on the microbiological standards and the production and packaging technologies for baby foods.

Aflatoxicosis Traced To Aflatoxin-
Contaminated Infant Milk Food

Dvorackova et al. (1977) in their studies of encephalopathy with fatty degeneration of viscera (Reye's syndrome) found that the disease appeared to be associated with aflatoxin-contaminated milk food, a finding supported by a number of other researchers.

CLINICAL PERSPECTIVE

Breast Cancer Is Indeed Preventable

It is unfortunate that the strong correlation between family history and breast cancer has been interpreted in terms of a genetic etiologic concept.

The concept requires that a mother's genes be passed on to the daughter who is then, by the nature of her genes, pre-determined to develop breast cancer. If this were true, then it would be one of the most hopeless situations women face in their attempts to prevent breast cancer.

Fortunately, though, the genetic concept is fatally flawed. The clinical facts speak clearly. Much of the Oriental populations, constituting half of the world's population, were virtually free of breast cancer prior to end of World War II in 1945. This marked the beginning of the entry of Western foods into the Orient.

An increasing number of studies now points to Western stored and fermented foods as being the cause of a rapidly increasing epidemic of breast cancer in Asia. Furthermore, there is increasing evidence which points to a lower incidence of breast cancer in women who consume a traditional, more-fresh-food diet versus one of stored/fermented foods.

If this book shall serve no other purpose than to disprove the flawed concepts about a genetic etiology of breast cancer, which make women believe that it is inevitable that they will die of the same disease as their mother, then it will have well served its purpose.

A woman must learn to use her own instinctive and "protective" mind power to decide whether or not she sees in this book a sufficient data-based explanation of the dietary toxin cause of breast cancer. If so, she can then adopt the appropriate dietary and behavioral measures which will prevent cancer from destroying her breasts and her life. Furthermore, she will then be in a position to protect the lives of her female offspring by instilling into them the dietary habits which are protective against breast cancer.

For further information relative to the several dozen other cancer-related gene mutations being researched, the reader is referred to Dr. Robert A. Weinberg's recent article in the September, 1996 issue of Scientific American.

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Chapter 29

FUNGI/MYCOTOXINS ARE THE CAUSE OF BREAST CANCER

FUNGI/MYCOTOXINS CAUSE BREAST CANCER IN HUMANS

CALCIFICATIONS IN BREAST CANCER LESIONS ARE FUNGAL

FUNGI/MYCOTOXINS CAUSE BREAST CANCER IN ANIMALS

THE REPORTED CLINICAL FACTS AND THE CORRELATIVE FUNGAL/MYCOTOXIN FACTS

Aflatoxin Found In Human Breast Cancer Tissue

Harrison *et al.* (1993) examined human breast cancer tissue for evidence of the presence of aflatoxin, a recognized potent carcinogenic mycotoxin. The researchers examined human DNA from a variety of tissues and organs to identify and quantify aflatoxin DNA-adducts. Such adducts are considered to be proof of the mycotoxin's presence in a particular tissue. (These researchers had already proved the value of this method in the detection of aflatoxin-DNA adducts in tissue from a case of acute aflatoxin poisoning in Southeast Asia.)

DNA from normal and tumorous tissue obtained from patients with cancer of the breast was examined. Tumor tissues had higher aflatoxin-adduct levels than did normal tissue from the same individual.

The result of this study is that it verifies the presence of carcinogenic aflatoxin within the cancer tissue and thus implicates aflatoxin as a cause of breast cancer.

Cyclosporin (A Mycotoxin) Causes Breast Cancer In Humans (3 Studies)

Cyclosporin is a fungal derived drug. It is classified as a mycotoxin in the mycology literature (Betina [1989]).

1. Vogt *et al.* (1990) reported the occurrence of *de novo* malignant tumors occurring in 598 renal transplant recipients who were immunosuppressed with cyclosporin.

Eighteen of 598 patients receiving their first renal graft along with cyclosporin treatment between 1981 and 1986 developed a malignancy at a mean interval of 33 months. The cyclosporin-induced cancers included breast cancer.

2. Escibano-Patino *et al.* (1995) reported the occurrence of breast cancer as a complication of cyclosporin use in their series of kidney transplant recipients.

3. Penn and First (1986) reported 88 tumors in eighty-seven organ transplant recipients after the use of cyclosporin. Malignancies appeared an average of 14 months after the cyclosporin treatment. There was a surprising frequency of endocrine-related malignancies (ovarian, testicular and breast) among these malignancies.

Aflatoxin Induces Malignant Changes In Human Breast Cells

Eldridge *et al.* (1992) noted that some environmental chemicals are stored in human breast fat which are documented to be rodent mammary carcinogens. These researchers stressed the importance of determining the cancer potential of environmental agents in this key target tissue.

An assay was developed for detecting cancer potential using cultures of normal human breast epithelial cells derived from 5 different women. A positive response was observed with aflatoxin.

The conclusion of this study was that aflatoxin causes normal human breast cells to become cancerous.

Moldy Cheese Causes Breast Cancer In French Women

Le *et al.* (1986), in a French case-control study of 1,010 breast cancer cases and 1,950 controls with nonmalignant diseases, found that breast cancer was found to be associated with increased frequency of mold-fermented cheese consumption (see Chapter 41, entitled *Cheese Causes Breast Cancer*, for other reported studies).

Oxalic Acid (A Mycotoxin) Found In Breast Cancer Lesions

Going *et al.* (1990) found that weddellite (calcium oxalate) crystals are present in calcifications found in the breast tissue of patients with breast cancer. Calcium oxalate crystals are formed when calcium binds with oxalic acid. In human and animal systems, this is a protective process which considerably reduces the severe toxicity of oxalic acid. Oxalic acid is a powerful corrosive agent and oxalate salts are widely used for their cleaning and bleaching properties!

Oxalic acid happens to be a mycotoxin which can be produced by a number of different fungal species. Some fungi produce such large amounts of oxalic acid that they are used for commercial production of the chemical.

Aspergillus niger fungal infection in human lungs produces large amounts of oxalic acid which is extremely toxic to the blood vessels and which may cause fatal pulmonary hemorrhages. Consequently, oxalic acid (calcium oxalate crystals) in the sputum or lung specimens of patients is also an indication of an *Aspergillus* infection of the lung. These calcium oxalate crystals are the same as the calcium oxalate found in breast cancers.

The presence of oxalates in the breast is indicative of the presence of fungi interwoven within the stages of breast cancer development. Since humans do not make oxalic acid themselves, this is an appropriate conclusion.

Breast Oxalate Calcifications In Mammographic Examinations

Thomas *et al.* (1993) examined calcifications found in breast mammograms and evaluated their relationship to the risk of subsequent breast cancer. The presence, morphology, and

distribution of calcifications visualized on baseline mammograms of 686 women who developed breast cancer over a 7- to 10-year follow-up period were compared with those of 1,357 controls who remained cancer free. It was found that there was a significant correlation between such calcifications and subsequent development of breast cancer.

Breast Cancer Calcifications Decrease With Tamoxifen (Antifungal) Treatment

Taylor and Georgian-Smith (1994) reported the regression of breast cancer in four patients who had been treated with tamoxifen. The patients were closely monitored with physical examination and mammography for a minimum of 2 years.

In all cases, the features of malignancy which were seen on mammograms regressed. These results were documented by a decrease in the number of calcifications and in the size of spiculated masses. These results suggest that these breast calcifications are dynamic in nature, being able to regress as effective treatment reduces the cancer.

CLINICAL PERSPECTIVE

The presence of oxalate calcifications in the breasts of virtually every patient with breast cancer, and their subsequent regression as a result of treatment with the antifungal agent tamoxifen, points to the strong possibility that there is a fungal role in this cancer. There have even been reports of fungal cells growing out of cancer cells. The existence of a viable fungal sub-forms—with its DNA co-mixed with a human's own DNA—could well explain the bizarre appearance of the DNA in cancer cells. Support for such a "science fiction" type scenario is found in the observation that a lectin staining procedure, used to find "invisible" fungi in tissue specimens, happens to identify breast cancer cells. Normal cells do not stain with these same lectin staining procedures.

The lectin stain is also taken up by strange multinucleated giant cells which suggests that these cells may, in fact, be fungal cells. This could explain the presence of oxalates in breast cancer tissue, a metabolite produced by fungi and not by humans. It might also help explain how breast cancer is caused by a number of fungal-fermented foods, particularly those made with Baker's or Brewer's Yeast (both being *Saccharomyces cerevisiae*), known producers of uric acid which degrades to oxalic acid (Costantini [1989]).

Baker's yeast is used to make bread, a documented cause of breast cancer in Japanese women. Brewer's yeast is used to make many alcoholic beverages, all of which are known to cause breast cancer in every country where the connection has been investigated, a fact which is well documented (See Chapters 27, 30-32, relative to alcohol, beer and wine causing breast cancer.)

Aflatoxin Causes Breast Cancer in Rats

Leszczyszyn (1986) reported the results of experiments in which aflatoxin induced mammary cancer in rats.

Breast Tumors In Rats Caused By The Fungus *Penicillium camemberti*

Gibel *et al.* (1971) conducted experimental studies of the cancer-causing fungus *Penicillium camemberti* var. *candidum* in which mammary neoplasms were induced in rats. (*Penicillium camemberti* is the fungus which is used to make Camembert cheese, frequently consumed in the Western diet.)

T-2 Toxin (*Fusarium*) Causes Breast Tumors In Rats

Schoental *et al.* (1979) reported that breast cancers were induced in rats which were dosed with T-2 Toxin.

T-2 Toxin is a *Fusarium* toxin frequently found in the human food chain. The fact that T-2 Toxin induced breast cancer in an animal model is most significant, for this cancer occurs so often in humans.

Furthermore, antibodies against *Fusarium* fungi are frequently found in human blood. These fungi and their toxins are the most frequently encountered contaminants found in animal feed and human foods. See also Saito (1971) and Corrado (1971), both of whom induced breast cancer in mice using moldy rice and its extracts.

Ochratoxin Causes Breast Tumors In Mice

Fibroadenomas in the mammary gland were found in over half of a group of female mice which were dosed with ochratoxin (Boorman [1988]). In humans, fibroadenoma is a documented risk factor for breast cancer (Dupont *et al.* [1994]).

Ochratoxin Causes Breast Fibroadenomas In Animals

Huff (1991) investigated the carcinogenicity of ochratoxin, a naturally occurring mycotoxin of the fungal genera *Aspergillus* and *Penicillium*, which was studied in three strains of mice and in one strain of rats.

It was found that fibroadenomas of the mammary glands were induced by ochratoxin administration. In humans, fibroadenoma is a documented long-term risk factor for breast cancer (Dupont *et al.* [1994]).

Penicillic Acid/Patulin Cause Breast Adenomas And Breast Sarcomas In Mice And Rats

Penicillic acid was found to induce mammary adenomas, as well as local sarcomas and fibrosarcomas in mice and rats. Patulin was also reported to cause mammary adenomas in mice and rats (Dickens and Jones [1965], Dickens [1967]). See also Ciegler *et al.* (1971).

Verrucaric Acid-Induced Breast Tumors In Mice

Jodczyk (1984) was able to induce breast tumors in mice by exposing them to a derivative of the mycotoxin verrucaric acid.

Moldy Rice Extract Causes Breast Cancer In Animals

Mammary cancers (breast cancers) were induced by feeding an alcohol extract of moldy rice to animals (Corrado [1971]). See also Saito (1971).

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Olive Oil Protects Against Breast Cancer In Italian Women

La Vecchia et al (1995) reported the data from a multicenter case-control study on breast cancer conducted in Italy which analyzed the relationship of olive oil and other dietary fats to breast cancer.

2,564 women hospitalized with histologically confirmed breast cancer and 2,588 matched controls were interviewed between 1991 and 1994 using a food-frequency questionnaire.

Olive oil was found to reduce the risk of breast cancer.

The study, based on a large dataset from various Italian regions, demonstrates a protective effect of the consumption olive oil against breast cancer. This protective effect was not observed with butter or margarine consumption.

United Nations Data

Documents That Olive Oil Protects

Against Breast Cancer In 26 Countries

Rose et al (1986) investigated the 1978-1979 mortality rates for cancers of the breast, prostate, ovary, and colon in 26 to 30 countries were related to the average 1979-1981 food availability data published by the United Nations.

The international comparisons support evidence from animal experiments that diets in which olive oil is a major source of fat are associated with reduced breast cancer risk.

Olive Oil Against

Breast Cancer In Greek Women

Trichopoulou et al (1995) have reviewed the experimental animal studies suggesting that olive oil consumption--in contrast to the consumption of other types of fat--does not enhance the occurrence of chemically induced mammary tumors.

The researchers studied the effect of consumption of olive oil, margarine, and a range of food groups on the risk of breast cancer. To this end a food-frequency questionnaire was administered to 820 women with breast cancer and 1548 control women.

It was observed that increased olive oil consumption was associated with significantly reduced risk of breast cancer.

No mycotoxins were detected in olive-oil destined for human consumption (6 samples) or olive-husks (3 samples) collected from oil-mills after the first pressing of olives.

THE CORRELATIVE FUNGAL/MYCOTOXIN FACTS

Olives are highly antifungal and seldom contain mycotoxins

Visconti et al (1986) conducted a limited survey of the natural occurrence of the major *Alternaria* mycotoxins, i.e. alternariol (AOH), alternariol methyl ether (AME), altenuene (ALT), altertoxin-I (ATX-I), and tenuazonic acid (TA) has been carried out on olives and related processing products (oil and husks).

The toxigenicity of *Alternaria* strains isolated from olives and the possible mycotoxin transfer into the oil have also been investigated.

Four out of 13 olive samples were contaminated by 2 to 4 *Alternaria* mycotoxins.

The highest contamination was found in a badly damaged sample containing 2.9, 2.3, 1.4 and 0.3 mg/kg of AME, AOH, ALT and TA, respectively.

No mycotoxins were detected in olive-oil destined for human consumption (6 samples) or olive-husks (3 samples) collected from oil-mills after the first pressing of olives.

Antimicrobial activity and inhibition of aflatoxin B1 formation

by olive plant tissue constituents.

Paster et al (1988) reported the antimicrobial activity and inhibition of aflatoxin B1 formation by olive plant tissue constituents.

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**WINE CAUSES
BREAST CANCER**

- WINE CAUSES BREAST CANCER
- WINE IS YEAST-FERMENTED FROM FUNGI/MYCOTOXIN-CONTAMINATED FRUITS
- WINE CONTAINS FUNGI AND MYCOTOXINS

THE REPORTED CLINICAL FACTS**Half A Bottle Of Wine A Day
Caused Breast Cancer In 6 Studies**

Howe *et al.* (1991) reviewed the data from 1,575 cases and 1,974 controls enrolled in 6 previously conducted case-control studies of diet and breast cancer with respect to alcohol intake. The 6 studies provided essentially the same finding; half a bottle of wine or an equivalent amount of alcohol per day was associated with a statistically significant elevated risk of breast cancer.

**Wine Causes Breast
Cancer In Dutch Women**

Van den Brandt *et al.* (1995) investigated the association of alcohol consumption and breast cancer in The Netherlands Cohort Study on Diet and Cancer which involved 62,573 women aged 55-69 years.

After 3 years of follow-up (1986-1989), the alcohol consumption data on 422 breast cancer cases were analyzed. An increased risk of breast cancer was found with higher levels of wine consumption.

**Wine Causes Breast
Cancer In Australian Women**

Smith (1989) measured per adult consumption of beer, wine, spirits and absolute alcohol for a 14-year period (1971-1984) and its relationship to female breast cancer mortality rates in Western Australia. During the 7 years of highest wine consumption, the highest rates for breast cancer occurred for females aged 30-59 years.

**Wine & Distilled Spirits Cause
Breast Cancer In Spanish Women**

Martin-Moreno *et al.* (1993) examined the relation between alcoholic beverage consumption and risk of breast cancer. Using data from a case-control study that included almost all cases occurring in five Spanish regions from 1990 to 1991, a total of 762 women with a diagnosis of breast cancer were compared with 988 control women.

It was found that even a very low alcohol intake (less than 8 grams/day) caused a 50 percent increase in risk of breast cancer. Consumption of 20 grams of alcohol or more per day was associated with a 70 percent elevation in the risk of breast cancer compared to no consumption at all.

An increased incidence of breast cancer was found for wine and distilled drinks, as well as total alcohol intake.

**Wine Causes Breast Cancer In Italian
Women Living In Grape-Farming Area**

Toniolo *et al.* (1989) conducted a study of alcohol consumption and breast cancer among 250 cases and 499 controls in a grape-farming area of northern Italy where wine consumption is widespread. In the study population, 30% of the women were abstainers and 15% reported alcohol intakes of 30 g/day or more.

A 2-fold increase in risk of breast cancer was observed with consumption of more than 40 g/day, the equivalent of about half a bottle of wine.

**Wine Causes Breast
Cancer In Italian Women**

La Vecchia *et al.* (1985) investigated the relationship between breast cancer and the consumption of alcoholic beverages in a case-control study of 437 women with breast cancer and 437 age-matched controls.

An increased incidence of breast cancer was found among those women reporting 1-3 or more alcoholic drinks per day. A positive trend in risk was evident for increasing daily consumption of wine alone, as well as for beer and spirits.

Allowance for all identified potential confounding factors, including the major risk factors for breast cancer and a few selected dietary items, did not appreciably change any of the alcohol-related estimates.

In addition, there was an increased risk of breast cancer associated in women who started drinking at a younger age.

THE CORRELATIVE FUNGAL / MYCOTOXIN FACTS**Ochratoxin Found In Red Wine And Dessert Wine:
Correlation With Ochratoxin In Human Blood**

Zimmerli and Dick (1996) analyzed various table wines from the Swiss retail markets for ochratoxin. Ochratoxin was found in a number of red wine and dessert red wines. Ochratoxin was also found in various grape juices. This fact suggests that the toxin in the wines was likely present prior to the making of the wines.

The researchers also observed that men living south of the Alps had significantly higher levels of ochratoxin in the blood than those living north of the Alps and that this appeared to be due to the consumption of red wines of southern origin, particularly southern Italian and southern Spanish wines.

**18 Species Of Living Fungi
Found In Spanish Wines**

Hidalgo and Flores (1991) analyzed the yeast flora in several wines from the Madrid area of Spain. From these 270 samples, they were able to isolate yeast strains belonging to eighteen different species. This finding is important because it demonstrates how severely fungal-contaminated food products can be. It is precisely this contamination which sets the stage for mycotoxin production to occur in wine and other fruits.

Fungi Found In Spanish Ribeiro Wine

Cansado *et al.* (1989) carried out an analysis of spontaneously fermented Ribeiro wine and was able to isolate 15 species of fungus and, in some cases, several different strains within a given

species. The researchers noted that several of the isolated fungi produce mycotoxins known to cause cancer in man.

Viable Fungi And Their Mycotoxins Found In High Quality Aged Wine

Schwenk *et al.* (1989) reported on the presence of the fungus *Trichothecium roseum* in several wines of high quality. They note that this fungus is capable of producing at least 3 mycotoxins, trichothecin, trichothecolone and rosenonolactone.

Furthermore, trichothecin (which is cytotoxic) was detected in some samples of longer-aged wines, suggesting that consumption of aged wines of higher quality may pose a greater risk of causing health problems than other younger wines. Also cytotoxic in wine is the alcohol itself, a fungal metabolite.

Filters Used In Wine-Making Are Ineffective Against Fungal Growth In Wine

In an assessment of the filtration processes used in the manufacture of wines, Thomas (1988) found that the growth of wine-spoiling yeasts and lactic acid bacteria were typically not inhibited by the filter. This result calls for the need for stricter microbial standards in the wine industry.

Aflatoxin-Producing Fungi Found In Wines in Thailand

Sripathomswat and Thasnakorn (1981) were able to isolate aflatoxin-producing fungi directly out of samples of Thai red, white, and rice sugar wine.

***Aspergillus* Found On California Wine Grapes**

Nelson and Ough (1966) showed in a test carried out in California that *Aspergillus flavus* can cultivate on wine grapes. This fungus is a producer of aflatoxin, a known human carcinogen.

Aflatoxin Found In Rice Wine In China

Ren (1984) reported the finding of aflatoxin in home-made yellow rice wine in China. Yellow rice is known to be heavily contaminated with fungi/mycotoxins and has been shown to be responsible for large numbers of deaths from cardiovascular disease.

Aflatoxin Found In Grape Wines In Poland

Lemieszek-Chodorowska *et al.* (1971) reported the finding of aflatoxin in grape wines in Poland.

Wine Cellar As A Cause of Acute Myocardial Infarction

Richter (1989) reported of a 50-year-old woman who was able to escape from the cellar in fermenting wine was being stored. Her husband, however, died in the cellar. Immediately after the incident she developed an extensive myocardial infarction of the anterior and septal wall in spite of an intravenous administration of streptokinase within 2 hours. Four weeks later coronary angiography showed normal vessels.

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