

Fungus & Cancer

Source: [Know the Cause](#)

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Every year, October is reserved for Breast Cancer Awareness month in America and each year in October, I dedicate my article to breast cancer and fungus. This year, I'm going to maintain my educational approach, but in lieu of addressing breast cancer alone, I'm going to beg the question that all men and women with breast, or any other type of cancer, have asked.

As some of you must know, I spoke at the Cancer Control Society (CCS) meeting in Hollywood, California, in late August. This lecture was special to me for so many reasons.

First, after decades of traveling and speaking, I've decided to wind it down a bit and spend more time studying, and making the TV show better. Second, I saw so many old friends in Hollywood that I hadn't seen in some time and it was such a joy. Last, but not least, both of my sons and their wives showed up to support me at the lecture. As life progresses, simply being in a room with my wife and all four of them present, excites me and defines the word "love" for me.

What a lucky man I am. The lecture went well and afterwards many attendees surrounded me for more information. I believe I've spoken at this event for some 14 years off and on and always on "the fungus link to cancer." There is a special place in heaven for Lorraine Rosenthal, who has spearheaded CCS for 41 years. She is an amazing friend to many, including me.

The 2013 version of "the fungus link to cancer," is noteworthy

because I continue to put the pieces of this complex cancer puzzle together. In this newsletter, I will update you on the question mentioned above that every person diagnosed with cancer has asked their doctor. The question carefully deals with the words, “why me?” In proper context, they are seeking to find out what happened to their immune defenses when they needed them the most! And what about this “superman” gene, the p53, that supposedly protected them against cancer. Did both of these safeguard systems fail them? WHY?

We will address those concerns, but let me start by giving you a “hot off the press” headline that my friend, Luke Curtis, MD, just sent me. It deals with 27 lung “cancer” patients who were later diagnosed with lung “fungus” instead of lung cancer. Mind you, this paper was just published. But the confusion that it confirms has been going on in medicine for as long as disease has been in existence. **Doctors do not know what causes the majority of human diseases.**

Fungal Infections Mimicking Pulmonary Malignancy

Relevant Sentence:

“Fungal infection can present with clinical and radiological features that are indistinguishable from thoracic malignancy, such as lung nodules or masses.”

To You and Me

It is impossible to tell lung fungus from lung cancer.

My Take

Unfortunately, the doctors who diagnose lung cancer are unaware of the fact that cancer mimics fungal infections. Unless one of the researchers who wrote the above paper are present during your lung cancer diagnosis, 100% of “lung nodules or masses” are diagnosed as “cancer” and 100% of you will begin invasive cancer treatment. I would certainly

recommend that you tell your doctors to “fully rule-out fungus” as a causative factor before cancer therapies are initiated.

According to Milton White, MD, cancer is “neither the result of a virus nor the consequence of an inherited gene defect. Cancer is a hybrid. It is due to a plant bacterium (conidia) derived from an Ascomycete strain of fungus...”

With the exception of the word “bacterium” I’d agree 100%. Dr. White felt that bacteria and human cells somehow merged and a new hybrid formed. I had the pleasure of meeting Dr. White in his Michigan hospital laboratory in 2001, while working on my book, [The Germ That Causes Cancer](#). Prior to his wife succumbing to breast cancer, he told me that she grabbed his arm and said, “you’re a smart man, Milton, so please figure out what causes cancer.” In her honor, I believe, he came within inches of a major discovery-but no one was listening to him. He ended up publishing his extraordinary cancer findings in medical journals that dealt with theoretical, rather than factual data, but such is the plight of a brilliant cancer pioneer. Few physicians regard theoretical data as medically relevant. Dr. White passed shortly after our meeting, but I’ve long felt like he and his wife are cheering me on from above! He deserves much credit in helping me fit the pieces of the cancer puzzle together.

If cancer is due to a disease-causing fungus, why don’t our two strongest defenses (germ gobbling white blood cells-a process called phagocytosis-and our p53 gene) prevent the fungus from overtaking our tissues?

I’m glad you asked. I recall a physician in Florida questioning my fungus/cancer lecture many years ago. She said, if bacteria or fungus caused cancer, it would be phagocytized (by our white blood cells) long before it could enable a disease like cancer. Mind you, some pieces of the

cancer puzzle were very difficult to find and this one took almost 15 years to piece together. After all, her question was extremely relevant!

In 2007, I was standing in a bookstore here in Texas called Half-Price Books. I love these stores because they all have a science section where I can pull up a chair and study. I pulled a dusty old 1989 medical book off the shelf called Mechanisms of Microbial Disease, and began reading. Would you know it, Someone had the page coincidentally fall open to this exact sentence;

Cryptococcus neoformans is a fungus that “escapes phagocytosis because the spores are surrounded by a thick viscous capsule.”

WOW! Keep in mind that this same fungus is in the family of “ascomycete,” or sac fungi. The fungal spores reproduce and grow, making the sac larger and larger, without oxygen (as do cancer tumors). The fungal sacs are thick viscous capsules, so what prevented the fungus from being gobbled up? Although this probably wasn’t known in 1989, most likely the ascomycete sac that these fungi thrive in enabled the fungus to thrive!

I sat down immediately and kept reading.

“In tissue, yeast cells of histoplasma capsulatum, are found within macrophages only. However, phagocytosis does not always lead to killing and the intracellular habitat paradoxically results in protection of the fungus from other defenses of the host.”

WOW-SQUARED! Let me put this in layman’s terms, because this is relevant if fungi contribute to or cause cancer;

Histoplasma capsulatum, a fungus that causes a serious and sometimes life threatening human disease called “histoplasmosis” is sometimes (cancer patients?) found in a

type of human white blood cell called a “macrophage.” These are also called “macrophagocytes” because they slowly gobble up and digest foreign (non-self) debris that enters our blood stream. Fungus would be foreign debris. But in the case of histoplasma capsulatum, itself a sac fungus, when confronted by the macrophages, they ingest the fungus, but instead of killing it and digesting it, something very unusual takes place. The enemy now protects the fungus as “friend” and begins assisting it in hiding from our other immune defenses. Why does this occur? As you may recall, in a human cell/fungal cell relationship, fungal cells always become the dominant cells, even to the detriment of human cells! Shall we take one giant hypothetical step forward?

Doug’s Cancer Hypothesis

I believe that Dr. White was so close! According to my hypothesis, cancer begins when the DNA from Fungus and the DNA from our white blood cells merge to form a new hybrid “tumor, or sac.” This hybrid attains a life of it’s own now, bypassing our immune defenses because it is 50% human, and therefore just enough to be recognized as “self.”

Critics argue that simply because our white blood cells fail to gobble up fungus, this would be almost insignificant compared to what would occur if our cancer tumor suppressor gene (p53) became inactive during cancer cell invasion. The critics are correct!

Along with phagocytosis, our p53 gene plays one of the most important roles in protecting us against cancer.

It not only stops cancer invasion, but it also kills tumor cells, thereby preventing cancer from even starting.

But in over 50% of all cancers, scientists have discovered that the patient’s p53 gene was mutated and unable to stop cancer from initiating. According to the American Cancer

Society, the p53 gene is the most studied of all genes because damage to this gene allows cells with damaged DNA, like cancer cells, to proliferate.

You know me. After hearing those dreaded 3 words, “you have cancer,” my first question would be, “doctor, what caused my p53 gene to mutate? Why didn’t it protect me?”

Of course, most doctors would respond by saying, “we don’t know what causes the p53 gene to mutate.” He may not, but now you will.

According to the medical journal, *Liver, International* (April 2011),

“aflatoxin genotoxicity is associated with a defective DNA damage response bypassing p53 activation.”

This means that the mycotoxin, aflatoxin, sometimes found in our food supply, is capable of inactivating the p53 gene at a time when we need it the most. The Proceedings of the National Academy of Science stated in 1993, that the mycotoxin, aflatoxin b1, made by *Aspergillus* fungus, is known to cause p53 mutations. It is all published.

Mycotoxins are made by fungus, yet few of our healthcare providers acknowledge that fungus contributes to cancer. I’ve said it thousands of times, but let me repeat it again. When it comes to medical treatment, to err is human. Let them be wrong, but don’t let them be dead wrong. You must be in control and ask the right questions in a doctor’s office. If that offends him, find one it won’t – it’s THAT important.