CHESSER-NAUGLE International Lectureship Grant:

The Fungal/Mycotoxin Connections: Autoimmune Diseases, Malignancies, Atherosclerosis, Hyperlipidemias, and Gout

October 11, 1993

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Twenty-Eighth Annual Meeting New Horizons in Chemical Sensitivities: State of the Art Diagnosis and Treatment

The Fungal/Mycotoxin Etiology of Malignancies and Auto-Immune Diseases

The vast majority of malignancies and all of the auto-immune diseases are of unknown cause. Fungi/mycotoxins have been for the most part ignored as documented cause of many malignancies and of auto-immune diseases. The etiopathogenetic mechanisms are not the usual patterns of the invasive-type mycoses nor of mycotoxicoses, but incorporate the occult features of both of these mechanisms. Some of the mycotoxin-induced malignancies are: hepato-cellular carcinoma, esophageal cancer, lung cancer, colon cancer, kidney cancer, breast cancer, colon cancer, endometrial cancer, leukemia, lymphoma, astrocytoma and Kaposi’s Sarcoma.

Auto-immune diseases are characterized by the finding of so-called auto-antibodies. It is a most popular concept but biologically fatally defective in that no species of life can make an antibody against itself; particularly causing fatal disease such as scleroderma. Scleroderma is considered to prove the validity of the auto-immune concept with the presence of auto-antibodies. However these are now documented to be antibodies against ubiquitin which is present in many species including fungi. Scleroderma responds well to the antifungal agent griseofulvin. Against whose ubiquitin is the host raising antibodies to, its own, or fungal-derived, in a disease state which responds to an antifungal drug? The auto-immune diseases responding to an antifungal antibiotic which has the same mode of action as griseofulvin, ketoconazole, neomycin, etc. (lovastatin, griseofulvin, ketoconazole, neomycin & hormones).

Gout and hyperuricemia are clinical entities of previously unknown etiology. Fungi & their toxins have been ignored as documented etiology of both entities. Hyperlipidemia is induced by a number of mycotoxins. Seasonal variations in hyperlipidemia correlates to seasons of maximal fungal growth and mycotoxin production. It will be shown in this presentation that hyperlipidemia is a protective-toxin binding mechanism that is seen in a number of complex infections and returns to normal with antibiotic therapy and/or toxin bind-agents including charcoal. Atherosclerotic lesions are characteristic by lipid deposition, foam cells, endothelial cell damage, smooth muscle cell proliferation, activation of all of the cellular and humoral elements of delayed hypersensitivity, and fibrosis/calcifications. All of these lesions are induced in animals and humans by fungi/mycotoxins. Cyclosporine, a mycotoxin (an immuno-toxic fungal antibiotic) causes accelerated atherosclerosis & hyperlipidemia in the vast majority of transplant patients. Primates developed hyperlipidemia and atherosclerosis when fed Fusarium toxins (corn). Hyperlipidemia associated with lipid-containing vascular lesions are found in sheep ingesting the mycotoxin sporidesmin. In humans, ergots induce spasm, stenosis and/or thrombosis of the coronary, carotid, aortic, renal, and peripheral arteries. Ergot-induced entities include angina, myocardial infarction, arrhythmia, carotid artery occlusion, stroke, intermittent claudication & gangrene. All drugs and dietary factors effective in treating atherosclerosis and/or hyperlipidemia share only antifungal or anti-toxicity activity (lovastatin, griseofulvin, ketoconazole, neomycin, fibers, etc.).
Medical data is for informational purposes only. You should always consult your family physician, or one of our referral physicians prior.

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