The Fungal/Mycotoxin Etiology of Atherosclerosis
Foods That Cause & Foods that Prevent
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INTRODUCTION-AN OVERLOOK
Atherosclerosis Is A Stored Food-Related Disease

Atherosclerosis is the leading cause of death. Despite intensive research, its etiology remains unknown but there is no question that the etiology of atherosclerosis is in some way related to food. However, and quite obviously, it is not the food itself which is poisonous but rather something which man has done to food in his development and management of his food chain as a part of the creation of a complex urban industrialization.

Man had to store food to make that social machine function and in so-doing, he created that for which there was no plan built into the Garden of Eden, food storage and food fermentation.

It was this manipulation of food which brought into the food equation the increased presence of the toxicogenic fungi whose toxins (mycotoxins) now permeate our food and our bodies and has given rise to the mycotoxin-induced diseases.

Atherosclerosis may be one of those diseases. Atherosclerosis is apparently food-related but not to the natural fresh food itself but possibly rather to the mycotoxins related to food storage. The techniques include the creation of toxicogenic fungal growth promoters such as concentrated oil and sugar products; grain fermentation such as making bread and beer; fermenting (curing) tobacco leaves; fermenting milk to make cheeses; etc. To make matters even worse, the Western diet lacks sufficient vegetables and fish, both of which are protective against toxicogenic fungi, and in the case of vegetable fiber, binds mycotoxins.

Atherosclerosis-Linked Risk Factors Are Not Etiologic.

Epidemiological studies have shown an association with such factors as hypertension, diabetes, hyperlipidemia, hyperuricemia, smoking, the "Western diet", and obesity. However, these positive associations are referred to as "risk factors" and not as etiological because, with the exception of what is vaguely described as the Western diet, over half of atherosclerotic patients have none of these risk factors linked to the pathogenesis of their particular arterial lesions.

Cholesterol/Lipids Are Not The Cause Of Atherosclerosis.

Atherosclerosis and hyperlipidemia are clinical entities generally accepted to be of unknown etiology. The postulated causative role of cholesterol and lipids has failed to be proven and, as Steinberg and Witztum (1990) point out, there is increasing movement away from viewing atherosclerosis as a primary lipid/metabolic process and towards finding a nonlipid etiology for its complex cellular nature.

It is a long overdue conclusion for as pointed out by Keys (1963), dietary cholesterol has absolutely no adverse effect in humans. Furthermore, cholesterol is a normal constituent of every living cell; cells can not be injured by their normal components.

The Atherosclerotic Lesion Is Granulomatous-Not Metabolic.

What Steinberg and Witzum see in their contemporary research, and Keys reported 30 years ago, is that lipids, cholesterol, and metabolic processes do not explain the complex inflammatory nature of the atherosclerotic lesion and there has to be something else to account for the "complex cellular nature of the atherosclerotic plaque".

As Virchow did over a hundred years ago, one must look at the underlying cellular pathology of the atherosclerotic lesion in order to climb out of the state of absolute metabolic confusion which now exists regarding the true nature of atherosclerosis.

The underlying cellular pattern of atherosclerotic lesions is one of delayed hypersensitivity. The cardinal finding of delayed hypersensitivity is the immunological granuloma, a type of host response which occurs only against microbes or microbial products such as Freund's adjuvant (dead mycobacteria). No such response has ever been encountered or induced in metabolic entities.

What the atherosclerotic plaque actually constitutes is a granuloma-like lesion flattened by the effect of the intraluminal arterial pressure exerted upon a soft tissue lesion located in the tight confines of the vascular wall.

There is a zone of central necrosis surrounded by macrophages many of which become foam cells, lymphocytes, plasma cells, mast cells, proliferated smooth muscle cells producing collagen which becomes the fibrous tissue encasement

complexed with calcific deposition.

Plaque rupture with discharge of the necrotic centrum into the vascular lumen is complicated by thrombus formation with the dire consequence of obstruction of blood flow with resulting infarction of the tissue supplied by the particular artery.

Virchow's view that atherosclerosis is an inflammatory disease is in retrospect obviously quite correct. He perceived in his microscope that cholesterol deposits were secondary phenomena and not the cause of the inflammation.

Lipid/Cholesterol-Oriented Research Proves That Neither Lipids Nor Cholesterol Causes Hyperlipidemia or Atherosclerosis

The hyperlipidemic risk factor dominates atherosclerosis research which has been directed towards attempting to prove that excessive dietary intake of cholesterol and/or lipids causes the disease.

None of this research effort has provided proof that either cholesterol or lipids, or any other particular food, consistently causes atherosclerosis either in animals or in humans. Conversely, severe atherosclerosis occurs in cholesterol-free/fat-free dietary experiments in animals. Also, African tribes whose adults characteristically consume up to 8 liters of whole milk direct from the udder of their cows have the lowest blood lipids in the world and no atherosclerosis.

What the sum total results of the dietary experiments actually prove is that dietary cholesterol and/or dietary fats per se are not the causes of atherosclerosis.

The Fungal/Mycotoxin Nature of The Atherogenic Western Diet

However, there is something in the Western diet which is absent from the "Far East" and the "native" diets and which is the cause of atherosclerosis. The missing link is some items which has been overlooked even in the face of their obvious presence in the Western diet and relative absence in the Far East/native diets. These exogenous atherogenic factors present in the Western diet, which have been documented to cause hyperlipidemia, hyperuricemia and atherosclerosis both in humans and animals, are the various fungi and their unique toxic metabolites, the mycotoxins.

Fungi and mycotoxins are present in variable amounts in the food products of Western civilization such as the stored grains, particularly corn, fat-laddened meat of stored grain-fed animals, yeast-fermented beer, wine, bread, cheeses, and cured (fermented) meats and tobacco leaf.

The recent Harvard study of the dietary habits of 90,000 American nurses followed for 5 years revealed increased atherosclerosis occurred in those nurses who were heavy consumers of two items characteristic of the Western diet, cookies and bread (yeast bread) (Lancet 1993).

The so called native diets and the wartime restricted diets, which are known not to be atherogenic, lack these specific types of fungal/mycotoxic connections. The missing items of the diet are bread, beer, cheese and other fermented foods. Similarly, successfully employed anti-atherosclerosis dietary measures significantly reduce the degree of exposure to these fungal/mycotoxic factors.

The Protective Aspects of Hyperlipidemia

The fungal/mycotoxin observations also may contribute a different meaning of the relationship of hyperlipidemia to atherosclerosis; it is protective mechanism which binds mycotoxins and thereby greatly reduces the degree of their cytotoxicity. Hyperlipidemia also provides an effective degree of antimicrobial activity.

Hyperlipidemia is actually a protective host response to microbial invasion, particularly if associated with microbial toxins. The indiscriminate lowering of one's lipoproteins without understanding their purpose may be a disadvantage such as seen in the several major medical reports (WHO and Finland Long Term Study) which have documented that some lipid-lowering measures may actually result in increased atherosclerosis.

Hyperlipidemia Is A Signal Of An Impending Disaster:

Quickly Find The Cause and Quickly Remove It.

Hyperlipidemia is indeed an ominous finding and needs to be properly investigated as to its specific cause in each individual patient. If it is of fungal/mycotoxin etiology, eliminate that cause and not only do the plasma lipids return to their normal levels, regression of active atherosclerotic lesions

will occur. (End stage fibrosis and calcification will not regress any more than they regress in the other immunological granulomatous diseases such as mycoses and tuberculosis).

The Antifungal/Anti-Mycotoxicity Nature Of Effective Anti-Atherosclerosis Agents

Critical to the documentation of the validity of a fungal/mycotoxin etiology of hyperlipidemia/atherosclerosis is the authors' finding that every single one of the quite heterogenous non-surgical measures proven to be effective in treating patients with hyperlipidemia and/or atherosclerosis share nothing in common except antifungal and/or antimycotoxin activity.

The Antifungal/Anti-Mycotoxicity Nature Of Effective Anti-Atherosclerosis Dietary Measures

Lastly, rather paradoxically for it is actually of prime importance, the atherogenic dietary factors to be avoided can now be clearly defined as those which contain mycotoxins, toxicogenic fungi, and fungal growth promoters such as cholesterol, lipids, yeasts, and sugars. The anti-atherogenic foods can be similarly defined in terms of their being antifungal and/or anti-mycotoxic such as green vegetables, beans-particularly soya, garlic, fiber, herbs, spices, fish, fatty acids, proteins and vitamins.

A Unified Etiologic and Therapeutic Concept Leads to more Effective Treatment and Cost Containment.

This unifying concept of etiology. preventive dietary measures and pharmacotherapeutics, provides both the patients and their physicians with a logical approach for both the prevention and the treatment of atherosclerosis as well as significant cost-reduction for those who are charged with paying the bill for the treatment of this common disease.

TABLE OF CONTENTS

PART I
FUNGI AND MYCOTOXINS

CHAPTER 1
FUNGALBIONIC: FUNGAL TOXINS CAUSE
DEGENERATIVE AND CANCEROUS DISEASES

CHAPTER 2
THE TROJAN HORSE: FUNGI AND
MYCOTOXINS ARE IN THE FOOD CHAIN

PART II
FUNGI/MYCOTOXINS ARE THE
CAUSE OF ATHEROSCLEROSIS

CHAPTER 3
THE FUNGAL/MYCOTOXIN ETIOLOGY OF
ATHEROSCLEROSIS AND HYPERLIPIDEMIA

CHAPTER 4
THE FABLE OF THE CHOLESTEROL/LIPID
ETIOLOGY OF ATHEROSCLEROSIS

CHAPTER 5
THE MICROBIAL INFLAMMATORY
NATURE OF ATHEROSCLEROSIS

CHAPTER 6
A NEW POSTULATE: MYCOPLASMA
ETIOLOGY OF ATHEROSCLEROSIS

CHAPTER 7
MYCOTOXIN-INDUCED
ATHEROSCLEROSIS

CHAPTER 8
THE MUSHROOM CONNECTION
TO ATHEROSCLEROSIS

CHAPTER 9
BAKER'S/BREWER'S YEAST
CONNECTION TO ATHEROSCLEROSIS

CHAPTER 10
FUNGI/MYCOTOXINS

CAUSE CORONARY ARTERITIS

PART III
HYPERLIPIDEMIA PROTECTS
AGAINST ATHEROSCLEROSIS

CHAPTER 11
MYCOTOXINS CAUSE HYPERLIPIDEMIA

CHAPTER 12
PROTECTIVE ROLE OF
HYPERLIPIDEMIA IN MICROBIAL INJURY

CHAPTER 13
PROTECTIVE ROLE OF LIPOPROTEINS
AGAINST MICROBIAL TOXINS

CHAPTER 14
THE PROTECTIVE ROLE OF CHOLESTEROL
IN MICROBIAL-RELATED INJURY

PART IV
MYCOTOXINS CAUSE ALL OF THE
ATHEROSCLEROSIS RISK FACTORS

CHAPTER 15
GOUT/HYPERURICEMIA: RISK
FACTORS FOR ATHERODCLEROSIS

CHAPTER 16
HYPERTENSION IS A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 17
DIABETES IS A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 18
ABDOMINAL OBESITY IS A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 19
TOBACCO IS A RISK

FACTOR FOR ATHEROSCLEROSIS

CHAPTER 20
ALCOHOL IS A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 21
HYPERLIPIDEMIA IS A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 22
OXIDATIVE STRESS IS A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 23
HOMOCYSTEINE IS A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 24
MENOPAUSE IS A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 25
STRESS IS NOT A RISK
FACTOR FOR ATHEROSCLEROSIS

CHAPTER 26
GENES AND ATHEROSCLEROSIS

PART V
ANTIFUNGAL DRUGS ARE EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 27
THE ANTIFUNGAL/ANTIMYCOTOXIN NATURE
OF AGENTS EFFECTIVE IN ATHEROSCLEROSIS

CHAPTER 28
GRISEOFULVIN EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 29
ANTIFUNGAL "STATINS" EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 30
NEOMYCIN EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 31
FIBRIC ACIDS EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 32
COLCHICINE EFFECTIVE
AGAINST ATHEROSCLEROSIS 122

CHAPTER 33
RIFAMYCIN EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 34
KETOCONAZOLE EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 35
FLUCONAZOLE EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 36
PROBUCOL EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 37
SALICYLATES EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 38
CALCIUM BLOCKERS EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 39
CHOLESTYRAMINE EFFECTIVE
AGAINST ATHEROSCLEROSIS

CHAPTER 40
SITOSTEROL EFFECTIVE
AGAINST ATHEROSCLEROSIS

PART VI
FUNGAL/MYCOTOXIN CONTAMINATED
FOODS & DRINKS WHICH CAUSE
ATHEROSCLEROSIS

CHAPTER 41
ALCOHOL CAUSES ATHEROSCLEROSIS

CHAPTER 42
BEER CAUSES ATHEROSCLEROSIS

CHAPTER 43
WINE CAUSES ATHEROSCLEROSIS

CHAPTER 44
BREAD CAUSES ATHEROSCLEROSIS

CHAPTERS 45-58
OTHER FOODS AND HABITS
WHICH CAUSE ATHEROSCLEROSIS

PART VII
VITAMINS AND MINERALS WHICH
PREVENT ATHEROSCLEROSIS

CHAPTER 59
VITAMIN A PREVENTS
ATHEROSCLEROSIS

CHAPTER 60
THIAMINE PREVENTS
ATHEROSCLEROSIS

CHAPTER 61
RIBOFLAVIN PREVENTS
ATHEROSCLEROSIS

CHAPTERS 62-80
OTHER VITAMINS AND MINERALS
WHICH PREVENT ATHEROSLEROSIS

PART VIII
PLANT COMPONENTS
PREVENT ATHEROSCLEROSIS

CHAPTER 81
PLANT CHLOROPHYLL
PREVENTS ATHEROSCLEROSIS

CHAPTER 82
PLANT FIBER
PREVENTS ATHEROSCLEROSIS

CHAPTER 83
PLANT PECTIN
PREVENTS ATHEROSCLEROSIS

CHAPTERS 83-88
OTHER PLANT COMPONENTS
WHICH PREVENT ATHEROSCLEROSIS

PART IX
FOODS WHICH PREVENT
ATHEROSCLEROSIS

CHAPTER 89
VEGETABLES PREVENT
ATHEROSCLEROSIS

CHAPTER 90
GARLIC PREVENTS
ATHEROSCLEROSIS

CHAPTER 91
TOMATOES PREVENT
ATHEROSCLEROSIS

CHAPTERS 92-115
OTHER FOODS WHICH
PREVENT ATHEROSCLEROSIS

PART X
EXERCISE AND SUNLIGHT

CHAPTER 116

EXERCISE PREVENTS
ATHEROSCLEROSIS vs.
EXERCISE INEFFECTIVE IN
PREVENTING ATHEROSCLEROSIS

CHAPTER 117
SUNLIGHT PREVENTS
ATHEROSCLEROSIS

PART XI
EGGS ON OUR FACES

CHAPTER 118
EGGS PREVENT ATHEROSCLEROSIS
and
EGGS DO NOT CAUSE
ATHEROSCLEROSIS

CHAPTER 119
CONCLUSION

REFERENCES

INDEX