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HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

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Lipodystrophy has become a prominent feature within the HIV population. Abnormal glucose and lipid metabolism are the predominant risk factors for coronary vascular disease (CVD) described in people living with HIV infection (PLWHIV) on highly active antiretroviral therapy (HAART) and partial immune reconstitution. Central visceral adiposity appears to be a distinctive feature of lipodystrophy associated with the metabolic changes described above. To date, the cause is not fully understood. Acknowledging the possible role however of dietary components in decreasing the risk of CVD may help limit the incidence of this disease. The manipulation of numerous dietary components may help to lessen the occurrence or severity of CVD.

The purpose of this two-part article is to make a statement on the current state of knowledge about the risk for CVD in PLWHIV or AIDS on HAART. Secondly, for an historical perspective, we have chosen to review the research on food, nutrition and heart disease in the general population. Since there is no published data on the outcome of nutrition interventions in PLWHIV or AIDS with these risk factors, the final component of this article presents the results of a survey conducted on selected HIV nutritionists across the country. The endpoint arrives at a preliminary set of nutrition guidelines (based on expert opinion and anecdotal evidence) to help walk people through still another step in the survival process in the battle against HIV/AIDS.

(Continued on page 2)

Inside This Issue

heart healthy food choices in the era of haart	1
alternative focus: part ii: botanical use among people living with hiv	1
med watch: doxorubicin	5
resource corner	5
glossary-- definition of words noted with this symbol *	9
program spotlight: food for life network - miami, fl	10
handout: selected complementary treatments	23



ALTERNATIVE FOCUS- Part II: botanical use among people

By Nancy Spaulding-Albright
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This two-part article provides

(Continued on page 13)



HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

(Continued from page 1)

As people on effective HAART survive AIDS, heart disease is a new health hazard on the horizon. Since 1997, multiple risk factors have been reported in PLWHIV and AIDS. Those who have achieved undetectable viral loads and escalating CD4 T-cells* seem to be prime targets for one or more of these heart disease markers: hypercholesterolemia (high total cholesterol with low high density lipoproteins {HDL} and high low density lipoproteins {LDL}); hypertriglyceridemia and glucose abnormalities. Outwardly, one might demonstrate fat maldistribution with central visceral truncal obesity and peripheral fat wasting. Collectively the condition is labeled lipodystrophy (see Nov/Dec 1998 and Jan/Feb 1999 Review issues for more on lipodystrophy). To date, we have not identified the etiology nor have we arrived at solutions.

ERA OF HAART

In the year 2000, for many people, HIV has evolved into a chronic disease. Major events between 1995 to 1997 are given credit for changing the destinies of numerous AIDS patients who were almost certain to die of opportunistic infections and cancers. Events include the advent of protease inhibitors (PIs) and HAART^(1, 2) as well as the introduction of an HIVRNA assay^(3, 4) to measure levels of virus in the blood. This gave providers the tools to evaluate their patient's viral activity and an

armamentarium* of medical expertise with the potential to save their lives. Death rates plummeted while PLWHIV settled into major pill burdens and the era of undetectable viral loads and escalating CD4 T-cells.⁽⁵⁾



Early signs of metabolic complications of therapy were released from the Food and Drug Administration (FDA) in the form of a Public Health Advisory to physicians in June of 1997. The FDA had 83 reports of new or exacerbated cases of diabetes or hyperglycemia in HIV-positive patients on PI therapy. Twenty-seven of these cases required hospitalizations, five were life threatening. Average time of onset was seventy-six days after start of treatment, but in some patients symptoms began in as little as four days.⁽⁶⁾

In late 1997, early 1998 we saw the first published reports of a phenomenon seen in HIV-positive people on HAART that included increased abdominal girth, otherwise called central visceral obesity, breast enlargement in women and buffalo hump. In addition to hyperglycemia and diabetes, hyperlipidemia was also seen as reason for concern because of the link to atherosclerosis.⁽⁷⁾ The condition was described as lipodystrophy (an abnormality in the use of fats in the body).⁽⁸⁾

Dube and others estimated a 6% incidence of diabetes associated with the new PI therapy.⁽⁹⁾ He speculated that pancreatic β -cell destruction by PIs was probably not the cause. The fact that the hyperglycemia responds to sulfonylureas (oral agents) pointed to diabetes type 2, which is characterized by insulin resistance.⁽¹⁰⁾ Behrens and colleagues reported that glucose intolerance (a disease state in which fasting plasma glucose level is less than 140 milligrams (mg) per deciliter (dl) and successive plasma glucose concentrations following a glucose tolerance test exceeds 200 mg/dl.) was apparent in 18 patients and diabetes in 5 of 38 patients on PIs.⁽¹¹⁾ Insulin resistance was reported in this study and has also been reported by others.⁽¹²⁾ Insulin resistance is usually not associated with glucose intolerance or diabetes and is described as a calculated value that assesses the amount of insulin required to maintain normal blood sugar levels.⁽¹³⁾ It is thought that glucose metabolism, because of insulin resistance, is probably altered in a substantial number of patients taking PIs, but diabetes develops rapidly only among patients with a family history of the disease⁽¹⁴⁾

At the 6th Annual Conference on Retroviruses and Opportunistic infections in 1999, Walli and colleagues and Glesby and others presented new findings of insulin resistance with and without PIs.^(15, 16) Contrary to Dube's belief that pancreatic β -cell destruction was

(Continued on page 3)



HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

(Continued from page 2)

not the cause of PI associated diabetes, new speculation notes there is pancreatic β -cell dysfunction in some PLWHIV on PI containing HAART and hyperproinsulinemia is an indicator. ⁽¹¹⁾ Diabetes is an independent risk factor for large vessel atherosclerosis and myocardial infarction (MI) in people with both insulin and non-insulin dependent diabetes. Coronary artery disease (CAD) is the most common cause of mortality in adults with diabetes. The risk of CAD increases with the duration of diabetes. ⁽¹⁷⁾

In an early study of central obesity in PI users, Miller and colleagues compared HIV-positive patients who had been using indinavir for at least 6 months to indinavir-naïve (had not tried PIs), HIV-positive patients. ⁽¹⁸⁾ The indinavir users were divided into two groups, symptomatic versus non-symptomatic. Measurements were made of the VAT: TAT (visceral adipose tissue: total adipose tissue) ratio, weight gain, body mass index (BMI) and triglyceride (TG) levels.

Symptomatic indinavir users had a higher VAT: TAT ratio and elevated serum TGs that correlated with the initiation of the drug and the VAT: TAT ratio. BMI and weight gain did not change after the drug was started, but the accumulation of intraabdominal fat was apparent from CT examination. The authors refer to central obesity and this over abundance of visceral fat as a marker for various metabolic and endocrine disorders

including hyperlipidemia and glucose intolerance that had been reported with the use of PIs.

In another study, Lo and colleagues investigated the appearance of buffalo hump in eight HIV-positive patients. ⁽¹⁹⁾ Four of these patients were on drug regimens that included PIs and four were on protease sparing protocols. Since a buffalo hump is a classic marker for Cushing's syndrome one of the goals of this study was to rule out the possibility of a cushingoid* type disease. Plasma cortisol* levels were normal in all eight subjects, and 24-hour urinary free cortisol was within the normal range in all but one patient. Compared to HIV-positive patients without a buffalo hump, TGs were elevated but not significantly. There was no difference in glucose and cholesterol levels. Yanovski and others also ruled out hypercortisolism in a group of patients on PI therapy who demonstrated intraabdominal obesity, insulin resistance and hypertriglyceridemia. ⁽²⁰⁾

At the 5th annual Conference on Human Retroviruses and Opportunistic Infections, Carr and colleagues reported on a symptomatic group of HIV-positive patients in Australia who were taking HAART with PIs. ⁽²¹⁾ The study group consisted of 195 patients including 116 on PIs. Seventy-four of the PI treated patients reported changes in body

shape that included an increase in abdominal girth and thinning of the legs and face. Blood samples revealed elevations in TGs, and total cholesterol with decreased HDL cholesterol. The ratio of insulin to glucose was also elevated (a measure of insulin resistance). The alteration in body shape took from 10 months to more than a year to develop in some patients.

At the International AIDS Conference in Geneva, Carr and colleagues presented the first theory on how PIs might cause lipodystrophy. ⁽²²⁾ Searching for answers, they speculated that since the PIs bind to the HIV protease they might bind to similar looking proteins involved in lipid metabolism. They targeted lipoprotein receptor- like protein (LRP) and cytoplasmic (cellular) retinoic acid-binding protein type I (CRABP-1). [Editor's Note: See Nov/Dec 1998 Review issue for more information on this protein.]

"The ratio of insulin to glucose was also elevated (a measure of insulin resistance)."

The researchers were relying on the fact that if the PIs were inhibiting these two proteins it might explain the changes in serum lipid concentrations as well as the destruction of peripheral adipocytes, thus the fat wasting in the extremities. This theory has not been proven or disproved.

(Continued on page 4)



HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

(Continued from page 3)

At the 6th Annual Conference on Retroviruses and Opportunistic Infections, Dr. Carl Grunfeld spoke about a longitudinal study that he conducted on 41 patients; 20 patients on protease-containing HAART, 9 on 3TC without PI and 12 on antiretrovirals (ARV) without PI or 3TC. ⁽²³⁾ The only subjects with lipid elevations including TGs, total cholesterol, LDL and very low-density lipoprotein (VLDL) were those on PI therapy.

Kotler first presented his theory at the 1998 Geneva conference. ⁽²⁴⁾ He described patients that he had seen before PI and HAART with features of lipodystrophy. His findings were later published in a retrospective study of 430 “previous” patients (studied since 1984) and 96 “current” patients (studied since January 1996). Features of lipodystrophy, primarily gain of visceral fat, were seen in patients with and without PIs. In his “current” group, Kotler found the best correlation to be plasma HIVRNA and fat redistribution. He did not attribute lipodystrophy to a direct effect of drugs but an indirect effect, more likely partial immune reconstitution. Aldeen and colleagues described a group of 58 patients who were PI naïve, but had been treated with nevirapine based HAART for more than a year. ⁽²⁵⁾ Nine of the patients had symptoms of lipodystrophy that included abdominal obesity and peripheral fat wasting.

Kotler theorizes that lipodystrophy is similar to Syndrome X. ⁽²⁴⁾ He notes lipodystrophy-like syndromes occur in other chronic infections and are associated with stress. This syndrome includes fat redistribution, elevated blood levels of lipid, insulin and glucose. The syndrome also includes elevated urinary cortisol secretions that Kotler has noted

“The syndrome also includes elevated urinary cortisol secretions that Kotler has noted in many of his patients with lipodystrophy.”

in many of his patients with lipodystrophy. He believes that the PIs uncover this underlying phenomenon by dramatically reducing viral load and affording partial immune reconstitution.

In summer 1999, another theory on the etiology of lipodystrophy made news. Most notably, that it was not only PIs causing the problem but the nucleoside reverse transcriptase inhibitors (NRTI) are also suspect. St Marc and colleagues presented data on lipodystrophy (body fat redistribution) in protease naïve patients on nucleoside therapy. ⁽²⁶⁾ Polo and others presented information on 150 patients receiving different combinations of NRTI and protease containing HAART. ⁽²⁷⁾ Body composition changes were significantly different depending on the combination of drugs prescribed.

Nucleosides can cause mitochondrial damage resulting in high levels of lactic acid.

Brinkman and colleagues propose that the mitochondrial toxicity with associated lactic acidosis caused by all the NRTI agents may be a cause of lipodystrophy.

⁽²⁸⁾ It is the mitochondrial DNA that is vulnerable to damage by the NRTI, and has limited ability to repair mutations. Mitochondrial damage is linked to both heart muscle and kidney

damage. Mitochondrial DNA is fundamentally important to the function of every human tissue and toxicity is related to not only lactic acidosis, but polyneuropathy, myopathy, steatosis, pancreatitis and pancytopenia as well. Clinical symptoms include nausea, vomiting, abdominal pain and hyperventilation. Diagnosis is made on the detection of abnormal elevations of serum lactate.

The HIV-related mitochondrial toxicity lipodystrophy theory is based on its’ similarity to another lipodystrophy syndrome, Multiple Symmetrical Lipomatosis that also involves an abnormality in mitochondrial DNA. In vitro, NRTIs are shown to lead to reductions of mitochondrial DNA. Multiple Symmetrical Lipomatosis is genetically determined.

(Continued on page 5)



HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

(Continued from page 4)

Ellen Engelson, Ed.D, a close associate and co-investigator with Dr. Donald Kotler believes that metabolic abnormalities and fat redistribution definitely occur independently of each other. ⁽²⁹⁾ The question is why. We still do not know if they are a single syndrome or several. The researchers are also uncertain as to whether some medications may cause one abnormality but not another. According to Engelson, many questions remain since fat redistribution was also seen prior to HAART and elevated TG levels have always been associated with HIV.

In spite of intense international research around the clock and conferences devoted to the discussion and debate of lipodystrophy the cause remains a mystery. What is clear is that hyperlipidemia, glucose abnormalities and visceral adiposity are linked to CVD in the general population and coronary events have been reported in PLWHIV on HAART.

In Geneva 1998, the first poster was presented to really bring home the fact that these changes in blood lipids are putting people at high risk of CVD. Henry and others described three HIV-positive men treated with PIs who had angiographically proven CAD, two with MIs. ⁽³⁰⁾ All three were 40 years old or younger with no family history of heart disease. Two of the patients had high total serum cholesterol and TGs; one had very low HDL cholesterol.

Jutte and colleagues retrospectively studied patients to determine the incidence of MI with and without PI therapy. ⁽³¹⁾ Group one included 958 patients receiving non-protease HAART and group two involved 373 patients on PI containing HAART. The researchers found three cases of MI in cohort one (0.21 per 100 patients) and five cases in cohort number two (1.06 per 100 patients), a statistically significant difference.

According to one European autopsy study, cardiac disease was a problem before PIs and estimated to be the cause of death in 9.1% of persons with HIV. ⁽³²⁾ Opportunistic infections – fungal, viral, protozoan and HIV itself can infect the hearts of PLWHIV. ⁽³³⁾ Myocarditis, myocardial necrosis, cardiomyopathy, arteriopathy, endocarditis, pericarditis, pericardial effusion and cardiac neoplasm have been described in the literature.

⁽³⁴⁾ Heart disease is also a feature of aging and will increase as PLWHIV advance into their 50's. Other risk factors for heart disease besides lipid levels are family history of diabetes or heart disease, smoking and sedentary life style. ⁽³⁵⁾

Dr. Carl Grunfeld tried to put into perspective the risk of CVD on PI containing therapy versus the risk of dying from AIDS related causes in the absence of HAART. ⁽²³⁾ He notes if we assume that total cholesterol is from 140 to 170 and that HDL cholesterol changes little (25-27 mg/dl) as a result of PI

therapy, the risk of CAD rises by 0.19% per year. Compared to a decrease in the risk of dying averaging about 50% and as high as 85% in some centers, the benefits of PI therapy clearly appears to outweigh the risk. For now, it appears that people will remain on PI-containing HAART, so lifestyle changes will be necessary to decrease the risk of CVD and lessen the signs of lipodystrophy. Before we note what HIV-savvy nutritionists have to say about what they tell their people living with the syndrome, it will help to review the research on food, nutrition and heart disease.

FOOD AND HEART DISEASE: A HISTORICAL PERSPECTIVE



Today, the link between diet and chronic disease is considered a well-established medical fact.

Excessive intakes of fat, saturated fat, cholesterol, salt and alcohol, and low intakes of fiber contribute to five of the 10 leading causes of death in the United States: coronary heart disease (CHD), cancer, stroke, diabetes and liver cirrhosis. ⁽³⁶⁾ Yet just 50 years ago, scientists had only begun to explore the connection between diet and chronic illness as CVD replaced infectious disease as the leading cause of mortality in the United States. In 1948, a group of researchers under the direction of the U.S. Public Health Service

(Continued on page 6)



HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

(Continued from page 5)

launched a groundbreaking epidemiological study: The Framingham Heart Study. The initial group consisted of 5,209 men and women between 30 and 62 years of age who lived in Framingham, Massachusetts. Over the years, Framingham researchers have released more than 1,000 studies, many of which were among the first to recognize risk factors for CVD such as elevated serum TGs, LDLs and total cholesterol, and low levels of HDLs.

The Framingham Heart Study researchers were also among the first to demonstrate the relationship between vascular disease and non-insulin dependent diabetes mellitus (NIDDM). A study published in 1979 suggested blood pressure tended to be elevated in people with NIDDM, and these people were two- to three-times as likely to develop clinical atherosclerotic disease as people without NIDDM.⁽³⁷⁾ Another Framingham study described the link between elevated blood sugar and NIDDM by demonstrating that hyperglycemia is an independent risk factor for CVD in non-diabetic women.⁽³⁸⁾ For obese people with NIDDM, moderate weight loss has been shown to reduce hyperglycemia, dyslipidemia and hypertension.⁽³⁹⁾ Based on the scientific evidence that has accumulated over the years, the American Diabetes Association recommends that goals of medical nutrition therapy include the



achievement of optimal lipid levels and HDL cholesterol, and the maintenance of a reasonable weight.

In addition to the landmark studies that emerged from Framingham, data from the Seven Countries Study helped to define how diet

could lower the risk of CVD. In the early 1950s, Ancel Keys began the first of several investigations into the dietary habits of people inhabiting seven countries: Finland, The

Netherlands, Italy, Croatia (former Yugoslavia), Serbia (former Yugoslavia), Greece and Japan. His research revealed that morbidity and mortality from chronic diseases were lower in these countries than in the United States due largely to the influence of diet. Since that time, the many beneficial health effects of “the Mediterranean diet” http://www.oldwayspt.org/html/p_med.htm have been described in great detail. The diet, named for the food patterns typical of countries surrounding the Mediterranean Sea, is rich in fruit, vegetables, legumes and whole grains, moderate in alcohol and low in animal products, saturated fat, trans fatty acids and refined carbohydrates.

Many other studies including the Nurses’ Health Study, the Chicago Workers Study, the Ireland-Boston study, the Leiden study and the Multiple Risk Factor Intervention Trial (MRFIT) have contributed to our understanding of how diet and nutrition can lower the risk of heart

disease. These findings are supported by epidemiological data that document rising rates of CVD and certain types of cancer in countries that have adopted a Western-style diet rich in animal fat and low in fruits, vegetables and grains.

The accumulating body of evidence of how diet can protect against chronic disease has driven federal dietary recommendations since the 1970s. Current policy is expressed in the U.S. Department of Agriculture’s (USDA) food guide pyramid that recommends a diet based on grains and plant foods, and in the Dietary Guidelines for Americans. The dietary guidelines are: eat a variety of foods; maintain or improve weight; choose a diet with plenty of grain products, vegetables and fruits; choose a diet low in fat, saturated fat and cholesterol; choose a diet moderate in sugars, choose a diet moderate in salt and sodium; if you drink alcoholic beverages, do so in moderation.⁽⁴⁰⁾ The next research frontier will be to investigate the role that certain micronutrients and other dietary components might play. Already, evidence has emerged suggesting that high intakes of oleic acid, certain antioxidants, monounsaturated fatty acids and fiber, and moderate intakes of alcohol and high-fat dairy products, may correlate with a reduced risk of heart disease and some forms of cancer.

Fiber: The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study found that a cohort of

(Continued on page 7)



HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

(Continued from page 6)

middle-aged, smoking men with a median fiber intake of 34.8 grams (gm) per day were significantly less likely to suffer from major coronary events or die from CHD than men with a median fiber intake of 16.1 gm/day. ⁽⁴¹⁾ Epidemiological evidence, including recent data from the Nurses' Health Study, has supported the hypothesis that fiber may be cardioprotective. Researchers looked at fiber intake in women for up to ten years and reviewed incidence of acute MI or death from CHD. Results show that the age-adjusted relative risk for major CHD event was 0.53 for women who consumed the most fiber, compared with women who consumed the least fiber. Cereal fiber was strongly associated with a reduced risk of CHD. ⁽⁴²⁾ A significantly greater decrease in total cholesterol and LDL cholesterol has been observed with oat bran compared with wheat fiber. ⁽⁴³⁾

Cardioprotective effects have been observed in other sources of fiber. The Iowa Women's Health Study found a clear inverse association between whole-grain intake and risk of ischemic heart disease ⁽⁴⁴⁾, while a European study found the risk of ischemic heart disease was 15% lower among people whose fruit and vegetable intake was in the 90th percentile versus the 10th percentile of consumption. ⁽⁴⁵⁾

Folate: Elevated plasma levels of homocysteine, a breakdown product of the amino acid methionine, have been found to correlate with

increased risk of CVD. Data indicate that supplementation with the B vitamin folate can lower plasma homocysteine levels. However, the effect of folate supplementation on cardiovascular outcome remains unclear. Several recent studies have confirmed that high serum total homocysteine is related to increased coronary risk ⁽⁴⁶⁾ and that folic acid supplementation and dietary folic acid-rich foods reduce plasma homocysteine. ^(47, 48) Although lowering homocysteine levels through increased folate intake seems promising, it remains an unproven treatment. Still, many physicians and nutritionists support the folate/CVD hypothesis and recommend daily supplementation as well as high intakes of folate-rich foods such as citrus fruit and fortified grains.

Some prospective and epidemiological data support the hypothesis that supplementation with folate can lower rates of heart disease. Rimm and others looked at a cohort from the Nurses' Health Study to investigate intakes of folate and vitamin B6 in relation to the incidence of nonfatal MI. After 14 years of follow-up, researchers concluded that intake of folate (and vitamin B6) above the current Recommended Dietary Allowances of 180 mcg per day might contribute to lower rates of CHD among women. ⁽⁴⁹⁾

Olive Oil: Olive oil has traditionally been the main source of dietary fat for people living in the Mediterranean region. Its perceived cardioprotective effects are associated with oleic acid, a substance that has been shown to interfere directly with the inflammatory response that characterizes early atherogenesis, ⁽⁵⁰⁾ poly-phenolic compounds, which may contribute to lower rates of CHD, ⁽⁵¹⁾ and antioxidants, which appear to prevent lipid oxidation. ⁽⁵²⁾ A study comparing the effects of diets rich in three types of fatty acids on serum lipids and lipoproteins demonstrates how oleic acid may benefit hypercholesteremic patients. Subjects consumed diets in which fat accounted for 40% of total energy, of which 28% was supplied



by lauric, palmitic or oleic acid. There was a significant rise in serum cholesterol levels with lauric acid compared with oleic acid, and in total cholesterol concentrations

with palmitic acid compared with oleic acid. The authors concluded that, both lauric and palmitic acids are hypercholesterolemic compared with oleic acid. ⁽⁵³⁾

It should be noted that many of the studies suggesting that olive oil is

(Continued on page 8)



HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

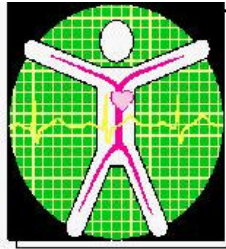
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cardioprotective have been small and not all demonstrate that it can influence risk factors such as high blood pressure and plasma lipid and cholesterol levels. In a randomized, double-blind, crossover study on the effects of dietary olive oil and fish oil supplementation on ten hypertensive patients, olive oil (9 gm daily for 6 weeks) was found to have no significant effect on diastolic blood pressure, systolic blood pressure or intracellular free platelet calcium.⁽⁵⁴⁾

Omega-3 Fatty Acids: Early studies in Greenland Eskimos, a population whose diet is rich in fish that contain essential omega-3 fatty acids, prompted researchers to investigate the potential role this nutrient may play in protecting against CAD. Some studies have noted a significant decrease in serum TGs in those consuming high levels of fish oils containing docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA). Studies have also shown a correlation between fish oil supplements and lower rates of mortality from cardiac disease, and fewer manifestations of cardiac symptoms.⁽⁵⁵⁾ A recent trial found an association between fish consumption and a lower risk from all-cause, ischemic heart disease and stroke mortality at the population level.⁽⁵⁶⁾

Not all research, however indicates that omega-3 fatty acids exert a cardioprotective effect. A case-

control study conducted in eight European countries and Israel found no association between adipose tissue levels of DHA and lower risk of MI in men.⁽⁵⁷⁾ A randomized, double-blind, placebo-controlled trial found only modest and statistically insignificant positive effects on the course of coronary atherosclerosis in humans.⁽⁵⁸⁾



Editors Note: As reported in the Jan/Feb 1999 Review issue, it is interesting to know that increasing intake of fish increases the importance of vitamin E supplementation. Also, more than once a week consumption of certain fish found in the Everglades, tuna and swordfish may be harmful in some regions of the U.S. because of its increased mercury content. Review the Jan/Feb 1999 Review issue for additional information on essential fatty acids.

Soy: Soy products such as soymilk, tofu and tempeh have been linked to lower rates of heart disease through the effects of isoflavones and phytoestrogens. The incidence of CHD, the major cause of morbidity and mortality in women, increases after menopause when serum levels of estrogen drop. Phytoestrogens may lower the risk of CHD by affecting lipids, carbohydrate metabolism, body fat distribution and blood pressure.⁽⁵⁹⁾

A recent study suggests that high intakes of soy foods, particularly those rich in isoflavones, may

contribute to lower total blood cholesterol and LDL cholesterol levels. After nine weeks, subjects who ate a standard low-cholesterol diet plus soy with 62 mg of isoflavones reduced total cholesterol by 4% and LDL cholesterol by 6%, compared with subjects who did not consume soy. Participants whose blood cholesterol exceeded 164 mg/dl were able to reduce total cholesterol by 9% and LDL cholesterol by 10%.⁽⁶⁰⁾

Some research shows that phytoestrogens can protect against oxidative DNA damage in lymphocytes. One case-control study suggests supplementation with soy may decrease oxidative damage to DNA but has no significant effect on plasma cholesterol or TG levels.⁽⁶¹⁾ The possible cholesterol-lowering properties of soy were demonstrated in another trial, however. This trial found that the National Cholesterol Education Program (NCEP) Step 1 diet combined with soy protein was associated with a statistically significant decrease in LDL cholesterol and the LDL to HDL ratio in men with normal and high cholesterol levels. The hypocholesterolemic effect of soy was independent of age, body weight, pretreatment plasma lipid levels and the sequence of dietary treatment during the trial.⁽⁶²⁾

Trans Fatty Acids: Trans fatty acids, formed by the hydrogenation of vegetable oils, have been linked

(Continued on page 9)



HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

(Continued from page 8)

to CHD. Studies have shown that high intakes of trans fatty acids correlate with risk factors such as high serum lipids and lipoprotein concentrations. Trans fatty acids have also been shown to increase the ratio of plasma LDL to HDL cholesterol. Dietary sources include stick margarine, shortenings, frying fats, commercial baked goods and certain crackers.

The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study observed a significant positive association between intake of trans fatty acids and the risk of coronary death. For example, the multivariate relative risk of men whose median trans fatty intake was 6.2 gm/day was 1.39 compared with men whose median intake was 1.3 gm a day. ⁽⁶³⁾ These studies suggest that lowering intakes of trans fatty acids may lower the risk of CHD.

Willett and others found that trans fatty acids were directly related to CHD risk by reviewing the dietary records of 85,095 women participating in the Nurses' Health Study. During eight years of follow-up, intakes of margarine, cookies, cake and white bread were each significantly associated with higher risk of CHD. ⁽⁶⁴⁾

Vitamin E: The Seven Countries Study demonstrated the protective role of antioxidants by showing that populations consuming a diet low in saturated fat and rich in antioxidants, combined with no smoking, are associated with a low risk of CHD. ⁽⁶⁵⁾ Among the

antioxidant vitamins, vitamin E may be the most promising in its potential to inhibit the development of CHD, however, its role remains controversial. Some studies indicate that supplementation with vitamin E and consuming vitamin E-rich foods can inhibit LDL oxidation and reduce deposition on arterial walls. According to results of one prospective cohort study, subjects who ate more than five ounces of nuts a week had a significantly lower risk of total CHD than women who ate fewer servings after adjusting for age, smoking and other known risk factors. The magnitude of risk reduction was similar for both fatal CHD and non-fatal MI, and adjustments for intake of dietary fats, fiber, vegetables and fruits did not change the results. ⁽⁶⁶⁾ The Cambridge Heart Antioxidant Study found that vitamin E supplementation (800 IU daily for the first 546 patients and 400 IU daily for the remainder of 2,002 patients with angiographically proven coronary atherosclerosis) significantly reduced the risk of both cardiovascular death and non-fatal MI, and non-fatal MI alone. ⁽⁶⁷⁾

Other research suggests that vitamin E when taken with beta-carotene may have detrimental effects in certain groups. In a randomized trial of the effects of alpha-tocopherol and beta-carotene supplements on male smokers with previous MI, the risk of fatal CHD

increased significantly in the groups that received beta-carotene or a combination of beta-carotene and alpha-tocopherol. ⁽⁶⁸⁾ Another study looked at the primary preventive effect of alpha-tocopherol supplementation on major coronary events in the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. The incidence of primary major coronary events decreased 4% among male smokers with no history of MI who received 50 mg/day vitamin E, but increased 1% among subjects who received beta-carotene (20 mg/day) compared with subjects who did not receive any supplements. ⁽⁶⁹⁾ Other studies have found no association between vitamin E and decreased risk of heart disease. Researchers agree that more trials are needed to define the potential role that vitamin E may play in the prevention of CHD.

Editors Note: Review the Jan/Feb '99 Review issue for additional information on vitamin E and its relationship with lipodystrophy.

Wine: Researchers with the Seven Countries Study were among the first to observe that moderate alcohol intake and consumption of wine in particular, may be associated with lower rates of chronic disease. Epidemiological studies have since demonstrated an inverse relationship between moderate alcohol intake and morbidity and mortality from CHD.

"Other research suggests that vitamin E when taken with beta-carotene may have detrimental effects in certain groups."

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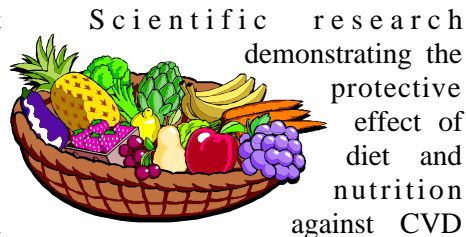
HEART HEALTHY FOOD CHOICES IN THE ERA OF HAART

(Continued from page 9)

⁽⁷⁰⁾ Wine has also been implicated in the “French Paradox,” a term that refers to the traditionally low rates of heart disease in France despite a diet that is seemingly rich in fat.

The beneficial components of red wine are attributed to certain antioxidants (resveratrol, catechin, epicatechin and proanthocyanidins), tannins, flavonoids and polyphenols. Studies suggest that moderate amounts of wine can raise HDL cholesterol, inhibit the formation of blood clots due to decreased platelet activity, reduce the proliferation of smooth muscle cells inside the arteries and reduce serum levels of lipoprotein (a), also known as Lp(a). (Lp(a) is a cholesterol-related complex related to the risk of heart disease and stroke.) Recently, the cardioprotective effects of wine and other alcoholic beverages have come under fire as researchers have hypothesized that a diet low in animal fat and cholesterol is responsible for the lower rates of heart disease in France. As the French have adopted a more American-style diet that contains increasing amounts of saturated fat and cholesterol, rates of heart disease are expected to rise. ⁽⁷¹⁾ Others suggest that people who drink wine have a healthier lifestyle than people who drink other alcoholic beverages. According to one recent study, wine drinkers were more likely to drink moderately, refrain from smoking, maintain a healthy body weight, exercise and work in “white collar” jobs. ⁽⁷²⁾

TREATING HEART DISEASE



Scientific research demonstrating the protective effect of diet and nutrition against CVD has forever changed the way the medical community treats heart disease. In the 1940s, CVD was considered to be a genetically determined disease and one of the inevitable consequences of old age. Framingham Heart Study researchers shattered these myths when they began to uncover the risk factors associated with CVD. In the 50 years that have followed, thousands of other scientists have contributed to our understanding of how dietary factors influence risk. Therefore, patients need not resign themselves to the debilitating and often deadly consequences of CVD. Rather, they can choose to fight heart disease by consuming a diet rich in whole grains, fruits and vegetables; moderate in alcohol and sugar; and low in fat, saturated fat, trans fatty acid and cholesterol. Studies have shown that exercise and quitting smoking can also lower CVD risk. There may be important lessons in the role that diet has come to play in the management of CVD that can be applied to other disease states such as lipodystrophy, which is also characterized by atherogenic plaque and high serum levels of fatty acids.

Editors Note: This two-part article provides information on the possible nutritional considerations

of CVD in those with lipodystrophy. Part Two of this article will detail dietary considerations in the era of HAART and relate what HIV-savvy nutritionists across the country are doing to face this issue.

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(Continued on page 14)



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PART TWO: FOOD CHOICES

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**HEART HEALTHY FOOD CHOICES
IN THE ERA OF HAART**

(Continued from page 13)

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