

Documentation of Common Cardiovascular Health Effects from Mercury
from Amalgam

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1 Documentation of common cardiovascular health effects from mercury from amalgam

Documentation of Common Cardiovascular Health Effects from Mercury from Amalgam

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1.1 Introduction

Cardiovascular disease affects more people and causes more deaths each year than any other chronic condition. Atherosclerosis (buildup of plaque deposits in arteries) is the most common type of heart disease. Atherosclerosis is a significant factor in many types of cardiovascular disease: coronary heart disease (CHD), myocardial infarction (MI), angina pectoris, cerebral vascular disease (CVD), peripheral artery disease (PAD), thrombotic stroke, transient ischemic attacks (TIAs), insufficient blood supply to lower limbs (cludication), organ damage, and vascular complications of diabetes.

Stroke is the third leading cause of death in the U.S. but millions also suffer silent strokes (TIAs) each year that cause memory loss, neurologic disorders, etc. Ischemic stroke is where a blood clot blocks the flow of oxygenated blood to a portion of the brain (83% of all strokes). The majority of these are related to atherosclerosis. Hemorrhagic stroke is where a blood vessel in the brain ruptures (17%). Irregular heartbeat and tachycardia is another common type of heart disease that has become more common (580, 584).

Other types of cardiovascular problems include hypertension, thrombosis, thrombocytopenia, peripheral artery disease (PAD), anemia, and Leukopenia. Hypertension is high blood pressure and may be caused by atherosclerosis or other factors including mercury toxicity. Supplementation with chlorella has been found to result in beneficial effects when used in patients chronic conditions such as hypertension, ulcerative colitis, or Fibromyalgia (304). Doctors such as D. Klinghardt (303) have suggested that the mechanism by which chlorella improves treatment of such conditions is metals detoxification, which is the main mechanism of action of chlorella and has been found to greatly improve intestinal function.

Factors underlying atherosclerosis include inflammation, free-radical assault, nutrient deficiency, “thick blood”, and ability to activate B vitamins such as vit B12 and vit B6. (30) Plaque buildup in arteries can cause dying of heart muscle cells, weakening of the heart muscle, irregular heart beat, angina, etc. Vit C is an essential factor in building and maintaining collagen and elastin, primary factors in connective tissues, so Vit C deficiency is a major factor in leaking veins and plaque buildup.

Supplementation with vit C has been found to significantly reduce such plaque buildups and leaking veins (30ab). Other factors in cardiovascular disease include imbalances of Lipoprotein A, C-reactive protein, Homocysteine, and Fibrinogen. See the section on treatment of cardiovascular conditions for more factors and tests to determine which factors need consideration.

Anemia is a decrease in the number of red blood cells. Anemia can be related to iron deficiency, Vitamin B12 deficiency, folate deficiency, etc. When one of these factors is present, supplementation can often resolve the problem, though B12 deficiency can also be related to reduced ability to absorb B12. In this case weekly injections may be required. Methylcobalamin is the preferred form of B12. Thrombosis is an abnormal blood clot inside a blood vessel, causing an obstruction of blood flow. Thrombocytopenia is usually microvascular leakage with platelet aggregation, often induced by drugs. Leukopenia is an abnormal decrease in the number of white blood cells. Chronic mercury exposure such as from amalgam dental fillings commonly has significant effects on levels and function of both red and white blood cells (35, 303) and reduction of mercury exposure often results in improvement of these conditions. Peripheral artery disease (PAD) is a lesser-known condition marked by blockages in the arteries leading to your extremities, most commonly your feet and legs. The damaging process begins when low-density lipoprotein cholesterol (commonly known as LDL or "bad cholesterol") encounters free radicals on the walls of your arteries. Free radicals are a factor in most chronic inflammatory process-and the development of atherosclerosis is no exception. When the production of free radicals exceeds your body's ability to remove them-a condition that can result from stress, smoking, drugs, environmental toxins, and even extreme sports-it results in oxidative stress. Unstable free radicals meeting with LDL cholesterol in the lining of arteries, causes a reaction called lipid peroxidation. The constant inflammatory assault that takes place at the site of these lesions can eventually take its toll on the fibrous cap that the immune system forms to keep it intact. Macrophages will secrete enzymes that weaken the cap, which can cause it to rupture-and once ruptured, platelets will be activated, causing thrombosis (the formation of a clot). In cases of advanced atherosclerosis, coronary arteries have become significantly narrowed over the years, allowing a clot to block blood flow to the heart-resulting in cell death (known as myocardial infarction) and heart failure. Likewise, a clot in your neck can block blood flow to your brain, resulting in a stroke. Lastly, the potential exists for embolism, in which the clots break off to enter your circulation, where they can obstruct blood flow to any number of your vital organs. All of these risks are increased by a condition known as hyperviscosity or hypercoagulation-an innate tendency toward clotting. Certain blood markers can reveal this condition: High levels of the amino acid homocysteine or excess fibrinogen - a protein that plays a key role in your body's clotting mechanisms, have been linked to hypercoagulation. Any of these conditions if untreated commonly lead to other degenerative conditions or can lead to death (580).

The primary risk factors that have been identified for cardiovascular disease are: elevated C-Reactive Protein, elevated fibrinogen, elevated homocysteine, elevated Lipoprotein (a), elevated LDL cholesterol/low HDL cholesterol, elevated triglycerides, hyperinsulinemia (excess insulin), low testosterone levels in men (580). Anyone concerned about cardiovascular health should periodically get a blood test to monitor the levels of these risk factors, which all can be significantly controlled or improved by avoidance of toxic exposures, diet and supplementation. As will be seen in this paper, toxic metal exposure is a significant factor in cardiovascular disease, causing inflammation and oxidative damage to the cardiovascular system and increases in the noted risk factors.

The personal risk factors of cardiovascular disease, like smoking, alcohol consumption, a diet high in saturated fat and cholesterol, sedentary life style, obesity, glucose intolerance and diabetes, and high salt intake have been extensively studied as contributors to the vascular diseases of the heart, brain and peripheral circulation but can be controlled by lifestyle decisions.

Inflammation and inflammatory cytokines such as Tumor Necrosis Factor Alpha (TNF α), interleukin 1b (Il-1b), and interleukin 6 (Il-6) have been found to be major factors in most cardiovascular conditions (580, 598). Measures of inflammation such as C-reactive protein, fibrinogen, homocys-

teine, and level of immune cytokines have been found to be the best guides to assessing cardiovascular health since these generate high levels of free radicals and lipid peroxidation chemicals. Excess insulin levels (hyperinsulinemia) has been found to be a significant risk factor for cardiovascular disease, and causes reactive hypoglycemia due to blood glucose deficiency, causing chronic hunger feeling and is a factor in why obese people do not lose weight.

1.2 Mercury, toxic metals, and cardiovascular disease

Both ionic and organic mercury accumulate in the heart and has been associated with elevated blood pressure, abnormal heart rhythms including tachycardia and ventricular heart rhythms, and increased heart attacks (125, 276, 10, 19, 20, 59, 205, 303, 348, 539, 571) [125, NAS, p.168 & 276, U.S.EPA, p.3-20]. It is unknown to what extent cardiovascular effects of mercury are due to direct cardiac toxicity or to indirect toxicity caused by effects on the neural control of cardiac function (276). The researchers believe that mercury promotes heart disease in several ways: mercury promotes free radical generation; it inactivates the body's natural antioxidant glutathione; and it binds with selenium thus making it unavailable as an antioxidant and component of glutathione peroxidase; All these mechanisms would lead to an increased level of lipid peroxidation and subsequent heart disease. The researchers also point out that studies have discovered a clear correlation between the number of amalgam tooth fillings and the risk of heart attack. Selenium and vitamin E have both been found to have a protective effect against mercury toxicity. Mercury has also been found to promote overgrowths of pathogens including bacteria and viruses that are known to damage the heart (303, 577).

The clinical consequences of mercury toxicity include hypertension, coronary heart disease, myocardial infarction, increased carotid IMT and obstruction, cerebrovascular accident, generalized atherosclerosis, and renal dysfunction with proteinuria (539, 541, 571a, etc.). Mercury induces mitochondrial dysfunction with reduction in ATP, depletion of glutathione, and increased lipid peroxidation and oxidative stress. The endothelial lipid signaling enzyme, phospholipase D (PLD), which is an important player in the endothelial cell (EC) barrier functions. All three forms of mercury (inorganic mercury, methyl mercury, and thimerosal significantly activated pulmonary artery endothelial cells in a dose-dependent and time-dependent fashion (571c). Metal chelators significantly attenuated mercury-induced PLD activation, suggesting that cellular mercury-ligand interaction (s) is required for the enzyme activation and that chelators are suitable blockers for mercury-induced PLD activation. Sulfhydryl (thiol-protective) agents and antioxidants also significantly attenuated the mercury-induced PLD activation. All the three different forms of mercury significantly induced the decrease of levels of total cellular thiols. Methylmercury also activates the lipid signaling enzyme phospholipase A2 (PLA2) in vascular endothelial cells (ECs), causing upstream regulation of cytotoxicity. Methylmercury also induced the loss of thiols and increase of lipid peroxidation in BPAECs (571d).

Numerous studies have reported tachycardia, high blood pressure and heart palpitations after acute exposure to elemental mercury vapor (19, 571, 538, 539, 541, etc.) A positive correlation was found between heart palpitations and urinary Hg concentrations in workers from a chlor-alkali plant (538, 276). In addition, tachycardia and elevated blood pressure have been reported in numerous instances after children were exposed to a broken thermometer, elemental mercury vapor, mercury in vaccines, or treated with medicines containing mercurous chloride, such as calomel containing teething powder, worm medicine, or ammoniated mercury ointments used for diaper rash (539, 541, 542). In children, tachycardia associated with the inhalation of elemental mercury vapor might be related to a non-allergenic hypersensitivity reaction to mercury (ATSDR, 539f). It should be noted that both blood and urine measure very recent exposures and are *not reliable indicators of mercury*

body burden¹ or mercury toxicity, as in (539b).

KAWASAKI DISEASE IS THE LEADING CAUSE of acquired heart disease in children in the developed world. Kawasaki disease is an acute systemic vasculitis that primarily affects children under 5 years of age. Many patients with Kawasaki's Disease have presented with elevated urine mercury levels compared to matched controls (542). Most symptoms and diagnostic criteria which are seen in children with acrodynia, known to be caused by mercury, are similar to those seen in Kawasaki's Disease. Coinciding with the largest increase (1985-1990) of thimerosal (49.6% ethyl mercury) in vaccines, routinely given to infants in the U.S. by 6 months of age (from 75 microgram to 187.5 microgram), the rates of Kawasaki's Disease increased ten times, and, later (1985-1997), by 20 times. Since 1990 88 cases of patients developing Kawasaki's Disease some days after vaccination have been reported to the Centers of Disease Control (CDC) including 19% manifesting symptoms the same day (542).

A recent review study found that toxic metals are a significant factor in cardiovascular disease (571). Mercury, cadmium, and other heavy metals have a high affinity for sulfhydryl (-SH) groups, inactivating numerous enzymatic reactions, amino acids, and sulfur-containing antioxidants (NAC, ALA, GSH), with subsequent decreased oxidant defense and increased oxidative stress (13, 571, 576). Such metal exposures are common and have additive or *synergistic effects*². Oxidative stress and lipid peroxidation have been found to be factors in metabolic syndrome and causes of inflammation (596, 598). Both metals bind to metallothionein and substitute for zinc, copper, and other trace metals reducing the effectiveness of metalloenzymes (571, 576). Mercury induces mitochondrial dysfunction with reduction in ATP, depletion of glutathione, and increased lipid peroxidation; increased oxidative stress is common (13, 571, 576, 303). Selenium antagonizes mercury toxicity. The overall vascular effects of mercury include oxidative stress, inflammation, thrombosis, vascular smooth muscle dysfunction, endothelial dysfunction, dyslipidemia, immune dysfunction, and mitochondrial dysfunction (571). The clinical consequences of mercury toxicity include hypertension, CHD, MI, increased carotid IMT and obstruction, CVA, generalized atherosclerosis, and renal dysfunction with proteinuria. Pathological, biochemical, and functional medicine correlations are significant and logical. Mercury diminishes the protective effect of fish and omega-3 fatty acids. Mercury, cadmium, and other heavy metals inactivate COMT, which increases serum and urinary epinephrine, norepinephrine, and dopamine. This effect will increase blood pressure and may be a clinical clue to heavy metal toxicity. Cadmium concentrates in the kidney, particularly inducing proteinuria and renal dysfunction; it is associated with hypertension, but less so with CHD. Renal cadmium reduces CYP4A11 and PPARs, which may be related to hypertension, sodium retention, glucose intolerance, dyslipidemia, and zinc deficiency. Dietary calcium may mitigate some of the toxicity of cadmium.

Adverse cardiovascular effects have been associated with exposure to MeHg. A retrospective study of cord-blood levels on 1000 children in the Faeroe Islands at age seven who had been exposed prenatally to MeHg was conducted. After body weight adjustments, as the cord-blood levels of MeHg increased from 1-10 micrograms/ liter, the diastolic and systolic pressures increased by 13.9 and 14.6 mm Hg. In boys, as cord-blood levels increased from 1-10 micrograms/liter their heart rate variability decreased by 47%. Heart rate variability is a reflection of cardiac autonomic control (308). Children with lower birth weights experienced blood pressure increases about 50% higher than normal birth weight children having similar mercury levels. At seven years of age, clear dose-response relationships were observed for deficits in attention, language, and memory (d). Thus a levels of exposure below current Government health safety limits, mercury is documented to have significant cardiovascular effects and the recommended limit for mercury has been decreased from the former limit of 10 $\mu\text{g/L}$ in blood.

A cohort of 1833 Finnish men were followed over 7 years (201), to compare dietary intake of

¹**Internet:** "<http://www.flcv.com/damspr17.html>".

²**Internet:** "<http://www.flcv.com/synergis.html>".

fish, and MeHg concentrations in hair and urine with the incidence of cardiovascular disease. All participants were free of clinical heart disease, stroke, claudication, and cancer at the onset of the study. Fish intake correlated with hair Hg and daily urinary Hg excretions. Men who consumed at least 30 grams of fish per day had a 2.1 fold greater risk of acute myocardial infarction. For each additional 10 grams of fish consumed there was an incremental 5% increase in the five-year risk of acute myocardial infarction. The fish consumed by this population was mostly fresh water fish, as differentiated from populations that eat mostly fatty fish like salmon and tuna and may factors that factors that partially counteract the effects of mercury (201c).

A large U.S. Centers for Disease Control epidemiological study, NHANES III, found that those with more amalgam fillings (more mercury exposure) have significantly higher levels of chronic health conditions (543a). A 2009 study found that inorganic mercury levels in people have been increasing rapidly in recent years (543b). It used data from the U.S. Centers for Disease Control and Prevention's National Health Nutrition Examination Survey (NHANES) finding that while inorganic mercury was detected in the blood of 2 percent of women aged 18 to 49 in the 1999-2000 NHANES survey, that level rose to 30 percent of women by 2005-2006. Surveys in all states using hair tests have found dangerous levels of mercury in an average of 22 % of the population, with over 30% in some states like Florida and New York (543c).

1.3 High levels of Mercury Exposure from Dental Amalgam

*Dental amalgam*³ has been documented by peer-reviewed studies, government studies, and scientific panels to be the largest source of mercury in most people (575), including methyl mercury since elemental and inorganic mercury are commonly methylated in the body. But many also get significant exposure to *methyl mercury from fish*⁴, and ethyl mercury from vaccines. The number of amalgam surfaces has a statistically significant correlation to blood plasma mercury level (17, 22, 23, 49, 79, 89, 133, 211). Much mercury in saliva and the brain is also organic (220, 272, 506), since mouth bacteria and other organisms in the body methylate elemental and inorganic mercury to organic mercury (51, 81, 225, 503b, 506, 512). Studies and clinical tests have found amalgam to be the largest source of methyl mercury in most people (506, 220, 79, 386, 575). Bacteria also oxidize mercury vapor to the water soluble, ionic form Hg (II) (431). A clinical study found that methyl mercury in saliva is significantly higher in those with amalgam fillings than those without, and correlated with the number of amalgam fillings (506). The average level of methyl mercury in the blood of a group with amalgam was more than 4 times that of groups without amalgam or that had amalgam replaced. Total mercury in those with amalgams was over 10 times that of those without amalgam. Other studies have found similar results (512, 575).

As is known from autopsy studies for those with chronic mercury exposure such as amalgam fillings, in addition to accumulating in the brain, CNS, and hormone glands, mercury also bioaccumulates in the heart (59, 85, 205, 348). Significant levels are able to cross the blood brain barrier, placenta, and also cellular membranes into major organs such as the heart since the oxidation rate of Hg⁰ though relatively fast is slower than the time required by pumped blood to reach these organs (290, 370). Thus the level in the brain and heart is higher after exposure to Hg vapor than for other forms (360, 370). The upper level of mercury exposure recommended by the German Commission on Human Biomonitoring is 10 micrograms per liter in the blood, but adverse effects such as increases in blood pressure and cognitive effects have been documented as low as 1 $\mu\text{g/L}$ cord blood, with impacts higher in low birth weight babies (308) and commonly in adults with levels below 10 $\mu\text{g/l}$ (540).

³**Informativo:** "Dental Amalgam Mercury Solutions".

⁴**Internet:** "<http://www.flcv.com/fishhg.html>".

1.4 Effects of Mercury Exposure on the Cardiovascular System

Mercury vapor is lipid soluble and has an affinity for red blood cells and CNS cells (21a).

Both mercury and methyl mercury have been shown to cause depletion of calcium from the heart muscle and to inhibit myosin ATPase activity by 50% at 30 ppb (59), as well as reducing NK-cells in the blood and spleen. The interruption of the ATP energy chemistry results in high levels of porphyrins in the urine (260) and stresses the major organs. The fractionated porphyrin test is approved by the FDA for diagnosis of mercury toxicity. Mercury also inhibits aquaporin-mediated water transport in red blood cells (479a), and has been found to cause significant heart damage (479b). Mercury accumulates in all hormone glands and adversely affects hormonal function, which controls all bodily processes, at very low levels of exposure.

Na(+), K(+)-ATPase is a transmembrane protein that transports sodium and potassium ions across cell membranes during an activity cycle that uses the energy released by ATP hydrolysis. Mercury is documented to inhibit Na (+), K (+)-ATPase function at very low levels of exposure (288). Studies have found that in patients with mucoid angiopathy, endomyocardial fibrosis and syndrome X there was a reduction in serum magnesium and RBC membrane Na (+)-K+ ATPase activity (263, 260d) and an elevation in plasma serum digoxin. This inhibition leads to depletion of intracellular magnesium and an increase in intracellular calcium load. This underlying magnesium-related insulin resistance and the consequence of this intracellular magnesium and calcium alteration in the pathogenesis of these disorders along with the inhibition of Na+-K+ ATPase can result in 1) defective neurotransmitter transport mechanism, 2) neuronal degeneration and apoptosis, 3) mitochondrial dysfunction, 4) defective golgi body function and protein processing dysfunction. It is documented that mercury is a cause of most of these conditions (13a, 43, 111, 288, 521b, 263, etc).

Mercury causes cardiovascular damage and disease: including damage to vascular endothelial cells, damage to sarcoplasmic reticula, sarcolemma, and contractile proteins, increased white cell count, decreased oxyhemoglobin level, high blood pressure (539, 541), tachycardia (539), inhibits cytochrome P450/heme synthesis (84, 35, 201, 538, 539), increased reactive oxygen species (13, 137), and increased risk of acute myocardial infarction (35, 59, 201, 202, 205, 212, 232, 306, 310, 351, 510, 50/201, 308).

Studies have demonstrated that low concentrations of mercury (HgCl_2 , ie, 10^{-9} - 10^{-15} M) significantly enhanced chemiluminescence, as well as stimulated H_2O_2 production by polymorphonuclear leukocytes (137). These studies clearly demonstrate the ability of extremely low levels of HgCl_2 not only to suppress various PMN leukocyte functions involved in host defense, but also to stimulate oxygen metabolism (137, 13). In vivo, these HgCl_2 effects would not only compromise host defense but also promote tissue injury via the local production of oxygen metabolites. This has been demonstrated increase effects of factors in cardiovascular disease and neurological disease. Melatonin, vitamin E, and vitamin C have been found to partially alleviate these conditions (13a).

Mercury has been found to accumulate in the pineal gland and reduce melatonin levels, which is thought to be a significant factor in mercury's toxic effects (569). Melatonin has found to have a significant protective action against methyl mercury toxicity, likely from antioxidative effect of melatonin on the MMC induced toxicity (567). Melatonin is documented to be effective at prevention of stroke and cardiovascular damage, as well as seizures and other neurological damage in patients that are prone to such conditions, and to be important in getting a good nights sleep in patients with many chronic conditions, which is important to both cardiovascular and neurological health (570).

Mercury binds to hemoglobin oxygen binding sites in the red blood cells thus reducing oxygen carrying capacity (232, 35) and adversely affects the vascular response to norepinephrine and potassium. Mercury's effect on pituitary gland vasopressin is a factor in high blood pressure (35, 201). Mercury also increases cytosolic free calcium levels in lymphocytes in a concentration-dependant manner causing influx from the extracellular medium (270c), and blocks entry of calcium ions into

the cytoplasm (16, 17, 21, 33, 35, 333), and at 100 ppb can destroy the membrane of red blood cells (35, 22, 17, 270c) and damage blood vessels - reducing blood supply to the tissues (34, 202, 306). Amalgam fillings have been found to be related to higher blood pressure (539, 541), hemoglobin irregularities, tachycardia (539), chest pains, etc. (201, 202, 205, 212, 222, 306, 310, 35, 59). Mercury also accumulates in the heart and damages myocardial and heart valves (Turpayev, in (35) & 59, 201, 205, 306, 351, 370).

Mercury has been found to be a cause of atherosclerosis, hypertension (539, 541), and tachycardia (539) in children and adults (59, 201, 205, 306, 308, 538, 571, 35) and heart attacks in adults (59, 201, 310).

Thyroid imbalances, which are documented to be commonly caused by mercury (369, 382, 459, 35, 50, 91, 212, 10b), have been found to play a major role in chronic heart conditions such as clogged arteries, myocardial infarction, and chronic heart failure (510). In a recent study, published in the *Annals of Internal Medicine*, researchers reported that *subclinical hypothyroidism is highly prevalent in elderly women and is strongly and independently associated with cardiac atherosclerosis and myocardial infarction* (510c). People who tested hypothyroid usually have significantly higher levels of homocysteine and cholesterol, which are documented factors in heart disease. 50% of those testing hypothyroid, also had high levels of homocysteine (hyperhomocysteinemic) and 90% were either hyperhomocysteinemic or hypercholesterolemic (510a). These are also known factors in developing atherosclerotic vascular disease. Homocysteine levels are significantly increased in hypothyroid patients and normalize with treatment (510efg, 511).

Studies have also established a connection between subclinical maternal thyroid disease and babies born with heart (509g), brain and neurological effects (509a-f), kidney defects, etc. Mercury reduces the blood's ability to transport oxygen to fetus and transport of essential nutrients including amino acids, glucose, magnesium, zinc and Vit B12 (43, 55, 96, 198, 263, 264, 338, 339, 347, 427); depresses enzyme isocitric dehydrogenase (ICD) in fetus, causes reduced iodine uptake, autoimmune thyroiditis, & hypothyroidism. (50, 91, 212, 222, 369, 382, 459, 35).

Another study (59) found such impairment of neutrophils by mercury decreases the body's ability to combat viruses or bacteria such as those that cause heart damage, resulting in more inflammatory damage. Clinical experience has found that mercury exposure increases susceptibility to pathogen infections, including those that adversely affect the heart (303), and that such infections cannot be controlled or eliminated without reducing mercury levels. Another way that mercury may cause cardiovascular conditions is through its adverse effects on gum disease, which is known to cause inflammation and increased levels of C-reactive protein (572, 576). C-reactive protein is a known marker for increased cardiovascular damage and disease (561), along with fibrinogen and albumin. Researchers at Duke University Medical Center and other research have discovered that otherwise healthy people who are prone to anger, hostility and mild to moderate depressive symptoms produce higher levels of C-reactive protein, a substance that promotes cardiovascular disease and stroke (562). Mercury is documented to be a common cause of anger, hostility, depression, and anxiety (564).

There are extensive documented cases (many thousands) where removal of amalgam fillings and/or mercury detoxification led to cure or significant improvement of serious health problems such as tachycardia and heart problems (205, 35, 59, 94, 115, 212, 222, 232, 233, 271, 306, 310, 539, 541, 571), blood and circulatory conditions (212, 222, 232, 233, 271, 523, 35, 95).

1.5 Other factors in Cardiovascular Disease and Beneficial Treatments

Some drugs that can cause cardiac arrest include codeine, hydrocodone, oxycodone, viagra, triptan drugs for migraine, and diuretics. S

Inflammation, free-radical assault, nutrient deficiency, and "thick blood" are factors underlying

cardiovascular disease, affecting levels of Lipoprotein A, “high sensitivity” C-reactive protein, homocysteine, and fibrinogen - which are factors/indicators of heart disease that can be tested for through blood tests. (30) High cholesterol is the body’s defense against some of these other factors, and reducing cholesterol without dealing with the real underlying problem can be counterproductive and dangerous. (30) Statin drug use depletes the vital heart nutrient CoQ10, so anyone taking Statins should also take CoQ10. Likewise Red Yeast Rice has similar effects as statins, but less dangerous side effects, but also requires additional CoQ10 supplementation (30).

Fish oil (DHA, EPA), DHEA, and vitamin K have been documented to suppress inflammatory cytokines, TNFa, Il-1b, and Il-6, reducing inflammatory effects (580, 30). Green tea, ginkgo biloba, garlic, vitamin E, vitamin A, lumbrokinase, nattokinase, L-carnitine, hawthorn, forskolin, and beta-carotene have been found to lower fibrinogen levels and lower cardiovascular risk levels (580, 581, 30). Excess homocysteine blocks the natural breakdown of fibrinogen. Elevated homocysteine can be reduced through the remethylation process [tri-methyl glycine (TMG), vitamin B12, folic acid, garlic] or the trans-sulfuration process (vitamin B6). Methionine is the only amino acid that creates homocystiene, so people who eat a lot of methionine foods such as red meat, chicken, dairy products need more vitamin B6. The level of supplementation can be determined by blood tests to see if risk factors are under control. In people with elevated fibrinogen levels, high levels of fish or olive oil and vitamin C (=;2000 mg) have been found to break down excess fibrinogen levels (580). CRP levels can be reduced by supplementing with natural vit E, fish oil, CoQ10, and ginger (30). Vitamin C, hawthorn, and CoQ10 have also been found to be effective in reducing the effects of congestive heart failure (CHF) and other types of cardiovascular conditions. Ginger appears to increase the contractile strength of the heart and to increase ATP energy production in the heart. (580) Studies have found that policosanol supplemenatation decreases LDL cholesterol and increases HDL. Choline, lecithin, and creatine have been found to have beneficial effects on cholesterol levels. L-arginine promotes vasodilation, maintaining both healthy blood pressure and regulating angina symptoms and taurine lowers the risk of abnormal clots and regulates heartbeat). (581cd) Padma Basic is a combination of many of these natural substances that has been found to be effective at reducing factors involved in cardiovascular disease (581). Pantethine (B5) is useful to increase the good cholesterol, HDL. Fiber from foods or psyllium binds cholesterol, but psyllium should be taken 2 hours away from medications (30).

Hyperinsulinemia is extremely common, especially in overweight individuals, and a significant factor in cardiovascular disease and type 2 diabetes. (580) High insulin levels deplete glucose levels in the blood, causing “reactive hypoglycemia” which prevents breakdown of fat cells. This can bring about a condition where the individual is constantly “hungry” (low in blood glucose) making it difficult to lose weight. Consuming foods high in glycemic index is a factor in this. Studies indicate that attention should be given to consuming foods primarily low in glycemic index and regular exercise. Low testosterone level in men has also been found to be a risk factor of cardiovascular disease, causing higher levels of cholesterol, fibrinogen, triglycerides, and insulin, along with abdominal fat increases, human growth hormone decreases, blood pressure increase. (580) DHEA is a precursor hormone of testosterone produced by the adrenal glands. Low levels of DHEA have been to be significantly related to heart disease.

Thrombosis causes can include atherosclerosis; injury to endothelial cells lining the heart, arteries, veins; blood hypercoagulability, excess fibrinogen, excess platelet aggregation (580, 581). As previously noted mercury and toxic metals can be a factor in some of these conditions and improvement commonly occurs after treatment for mercury toxicity. For cardiovascular conditions related to atherosclerosis, etc. EDTA chelation has been found to usually be a safe and significantly beneficial treatment (585).

Aspirin or blood thinning drugs are often used to reduce platelet aggregation to prevent thrombosis or strokes. Polycosanol, aged garlic, and niacin have been found to improve cholesterol balance safely and can be beneficial in alleviating or preventing cardiovascular disease. (580) Natural platelet

aggregation inhibitors include ginkgo biloba, EFAs, Vitamin E (tocopherol). Anti-Inflammatories that have been found beneficial include: curcumin, DHEA, Nettle leaf. Antioxidants that have been found beneficial in thrombosis prevention include quercetin, green tea, lycopene, grape juice. N-acetyl-L-cysteine, onions, and exercise have also been found beneficial (580). Other heart healthy nutrients include D-ribose, L-Carnitine, Flaxseed, and L-Arginine (30).

Other factors that have been found to be significantly associated with cardiovascular disease include daily consumption of soda drinks, diet drinks, fried foods, or a “Western Diet” high in fried foods, refined grains, fast foods, soda, etc. and low in fruits and vegetables (590). These diet patterns all have been found to be significantly associated with metabolic syndrome, a cluster of cardiovascular disease and diabetes risk factors including elevated waist circumference, high blood pressure, elevated triglycerides, low levels of high-density lipoprotein (HDL or “good”) cholesterol, clogged arteries, and high fasting glucose levels. The presence of three or more of the factors increases a person’s risk of developing diabetes and cardiovascular disease. An elevated hemoglobin HbA1c level has been found to increase risk of cardiovascular related problems and deaths, and this test can be useful in assessing risk.(580) Avoiding processed food and food cooked at high temperatures, and consuming nutrients that block damaging glycation reactions such as carnosine, benfotaine, and pridoxamine reduce A1c levels. Good dietary habits and regular exercise have been found to reduce cardiovascular problems and promote cardiovascular health. (30, 580) Highly colorful vegetables and use of coconut and coconut oil are part of a heart healthy diet.

Higher levels of vit D reduce heart attacks and strokes, and supplementation with Ginko Biloba may also reduce strokes (580) and improve recovery. EGCG extract from green tea or theaflavins from black tea have also been shown to have a significant protective effect in reducing inflammation and preventing cardiovascular disease (580). Studies have shown theaflavin supplementation significantly reduces levels of inflammatory cytokines such as TNF-alpha, Il-6, Il-8, and C-reactive protein; and lowered rates of production of inflammation-generating transcription factor NF-kB, cytokine generating COX-2, and the adhesion molecule ICAM-1. Theaflavin supplementation or drinking multiple cups of tea has also been found to have beneficial effects to prevention of ischemia-reperfusion injury following strokes as well as in reduction of LDL cholesterol and endothelial vasomotor dysfunction in patients with coronary artery disease (580).

Normal aging usually involves calcification in soft tissues throughout the body, such as heart valves, glands, and blood vessels. A calcium deficient diet increases such calcification. Atherosclerosis is the leading cause of disability and death. Homocysteine or oxidized LDL cholesterol are two factors that increase such damage. Studies show that insufficient vitamin K2 accelerates arterial calcification and vitamin K2 supplementation can reverse such arterial calcification (580). Studies also have found that emotional factors such as chronic anxiety, anger, or depression as well as insufficient sleep promote inflammation and cardiovascular disease, and that measures that decrease these are beneficial to cardiovascular health (562). Melatonin supplementation has been found to be beneficial to promoting sleep and benefitting the heart (563).

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