

COPPER AND THE HEART

There are numerous anatomical, chemical, and physiological similarities between animals deficient in copper and people with ischemic heart disease. Ischemic heart disease is the leading cause of death in the industrialized world. Copper deficiency is a leading cause of *ischemic heart disease* (IHD), a condition of recurring chest pain or discomfort that occurs when a part of the heart does not receive enough blood. Ischemic means “restriction of blood supply, as is found in heart disease. It does not mean a specific kind of heart disease, it’s just the general, normal kind of heart disease you know about.

This condition occurs most often during exertion or excitement when the heart requires greater blood flow. Ischemic means that an organ (the heart) is not getting enough blood and oxygen. Ischemic heart disease is also called coronary heart disease, or simply “heart disease.”

Copper is an essential trace element that has been an overlooked factor in IHD. Numerous animal and human studies have demonstrated that copper deficiency can cause IHD and that copper supplementation or adequate dietary copper can improve many of the risk factors for IHD. Copper deficiency is driving much of the current burden of IHD in the population.

The heart is one of the main organs affected by copper deficiency, causing a reduction in metabolism and energy supply in the heart. Insufficient dietary copper can produce almost every risk factor for IHD. There are many important similarities between copper deficiency in animals and IHD in humans, including glucose intolerance, hypercholesterolemia, abnormal ECG, hyperuricemia and hypertension, all of which are risk factors for IHD. Much of the lipid hypothesis of heart disease can be seen in the light of copper deficiency.

Lack of a recommended dietary allowance for copper may be hazardous to your health. Ischemic heart disease and osteoporosis are the likely consequences of diets low in copper. A search for “copper deficiency heart disease” reveals the exact opposite of what the experts say. Articles claim that copper deficiency is rare, not common. And that copper toxicity is a bigger danger. They claim that chronic copper excess can lead to liver problems, but they provide no evidence. And no mention of heart disease at all. You might think the newer article would be better. But it’s all misinformation.

The lack of news around how copper deficiency causes heart disease means that this particular issue is being censored. It’s not that Big Pharma-funded news agencies are simply unaware of this shocking news, especially given that the science is clear from the years 1975, 1990, 2000, 2007, and 2018, and especially so, given the strength of the science on this.

Rats that were made copper-deficient had a >50% increase in total cholesterol. The group of copper-deficient animals that were fed high-iron diets either died of ruptured hearts or developed severe anemia, enlarged hearts and livers, hypercholesterolemia, and elevated triglycerides. Copper deficiency ties in with the iron hypothesis of heart disease, in which excess levels of stored body iron promote IHD.

Dietary copper deficiency causes a variety of cardiovascular deficits. Systemic effects include high blood pressure, enhancement of inflammation, anemia, reduced blood clotting, and possibly arteriosclerosis. Effects on specific organs or tissues include weakened structural integrity of the heart and blood vessels, impairment of energy usage by the heart, reduced ability of the heart to contract, altered ability of blood vessels to control their diameter and grow, and altered structure and function of circulating blood cells.

In some instances, the cause of a defect can be directly attributed to reduced activity of a specific copper-dependent enzyme. However, there nonspecific mechanisms of damage have been implicated in cardiovascular defects of copper deficiency. They are peroxidation, the interaction of oxygen-derived free radicals with lipids and proteins (possibly DNA); glycation, the nonenzymatic glycosylation of proteins and nitration, the interaction of nitric oxide and its metabolites with peptides and proteins. Though independently these mechanisms present great potential for added reason for concern. At least aging suggests that copper deficiency may exacerbate deficits associated with these two conditions.

THE HARMS OF COPPER DEFICIENCY:

Increased cholesterol, decreased glucose tolerance and abnormal ECGs.

Increased LDL and triglycerides, and decreased HDL.

Increased susceptibility of lipoproteins and tissues to oxidation. Copper deficiency causes oxidation, not copper, as is sometimes falsely claimed.

Increased apolipoprotein B.

Increased blood pressure.

Increased plasminogen activator inhibitor type I.

Increased early and advanced glycation end-products.

Increased inflammation and increase in the expression of genes involved in inflammation and fibrinogenesis.

Ultrastructural irregularities of elastin and abnormal endothelial cells, subendothelial space, collagen fibers and smooth muscle cells.

Increased atherosclerosis.

Myelodysplastic syndrome (poorly formed blood cells).

Hepatic iron overload.

Fatty liver disease.

Cardiac hypertrophy, cardiomyopathy.

Optic neuropathy, myelopathy, anemia, and neutropenia.

Atrial thrombosis, abnormal ECGs and sudden death.

Substantial evidence exists to show that marginal copper deficiency is common.

Copper supplementation in humans leads to lower levels of markers of oxidative stress.

With reductions in copper levels in nearly all foods over the last several decades along with widespread adoption of ultra-processed food, low average copper consumption, and extensive subclinical insufficiency or deficiency of copper may be more common than once thought.

Copper deficiency is the only nutritional insult that elevates cholesterol, blood pressure, homocysteine, and uric acid, had adverse effects on electrocardiograms and arteries, impairs glucose tolerance, and promotes thrombosis and oxidative damage. More than 80 anatomical, chemical, and physiological similarities between animals deficient in copper and people with ischemic heart disease have been identified. Copper deficiency in animals can induce cardiac enlargement, pleural effusion, and heart failure that are reversible with copper supplementation.

In severely deficient animals, the myocardium is hypertrophied and may rupture. Coronary artery resistance is decreased in copper-deficient animals, but it is increased in ischemic heart disease.

Any disease that makes your heart work harder to pump blood through your body can cause an enlarged heart. Just as the muscles of your arms and legs get bigger when you work them, your heart gets bigger when you work it. The most common causes of an enlarged heart are ischemic heart disease and high blood pressure. Ischemic heart disease occurs when narrowed arteries, caused by fatty deposits that build up in your arteries, prevent blood from getting to your heart muscle.

Copper deficiency results in many diverse health problems, maybe up to 100. Copper deficiency also causes osteoporosis, diabetes, and neuropathy. People often have diverse multiple health problems all at once, it's rarely just heart disease alone. So obese people also often have diabetes, neuropath, heart disease, and weak bones all at the same time, and all of that is copper deficiency. Some people might not complain of relatively minor complaints such as neuropathy and back pain, compared to their larger problems of heart disease and diabetes that they are actively working on managing. There are so many folks that are fat, have heart problems, diabetes, neuropathy, back pain, all at once. This is typical.