

Cardiovascular Disease from Copper Deficiency—A History

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ABSTRACT Although the nutritional essentiality of copper was established in 1928, a preoccupation with hematology delayed the discovery of cardiovascular disease from copper deficiency for more than a decade. Anatomical studies of several species of deficient animals revealed, interalia, aortic fissures and rupture, arterial foam cells and smooth muscle migration, cardiac enlargement and rupture, coronary artery thrombosis and myocardial infarction. Abnormal biochemistry in deficiency probably contributes to these lesions, e.g., decreased activities of lysyl oxidase and superoxide dismutase which result in failure of collagen and elastin crosslinking and impaired defense against free radicals. Copper deficiency also decreases copper in hearts and other organs and cells and increases cholesterol in plasma. Abnormal physiology from deficiency includes abnormal electrocardiograms, glucose intolerance and hypertension. People with ischemic heart disease have decreased cardiac and leucocyte copper and decreased activities of some copper-dependent enzymes. Copper depletion experiments with men and women have revealed abnormalities of lipid metabolism, blood pressure control, and electrocardiograms plus impaired glucose tolerance. The Western diet often is as low in copper as that proved insufficient for these people. Knowledge of nutritional history can be useful in addressing contemporary nutritional problems.

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