Hypertension and Lead
The toxic effects of lead poisoning are well known; however, the effects of lower levels of lead exposure are uncertain. Although the finding is controversial, chronic lower level lead exposure has been linked to hypertension in both clinical and experimental studies.

Several cross-sectional epidemiologic studies have demonstrated an association between blood lead levels and hypertension in individuals without known occupational exposure and in men with occupational risk. In most of these studies, the association persists after correction for other variables. A similar number of studies have shown no association or only a very weak association between lead and hypertension, after adjustment for age and body mass index, in populations with or without known lead exposure. A study from the United States demonstrated an association only in African-Americans, who had higher lead levels (Sokas et al., 1997).

In the United States and Western countries, mild blood pressure elevations due to moderated increases in lead blood levels translate into potentially large numbers of patients dying with coronary artery disease. This link between lead and coronary artery disease is corroborated by data showing a direct association between blood lead and electrocardiographic changes of left ventricular hypertrophy.

Hypertension and Calcium Deficiency
Several studies have shown an inverse relation between dietary calcium intake and blood pressure. Meta-analysis comprising nearly 40000 people has shown that a high calcium intake lowered both systolic and diastolic blood pressure (Cappucio et al., 1995).

Calcium also induces natriuresis, which has been shown to lower blood pressure in postmenopausal hypertensive women (Johnson et al., 1985). Also, previous ecologic studies have shown an inverse relation between calcium level in drinking water and CVD.

Calcium deficiency increases blood pressure which can lead to stroke.
Cardiovascular Diseases and Water Hardness
A study was presented by Schroeder (1999), comprising the 153 largest cities in the United States. He found inverse correlations between water hardness and CVD in both men and women. He also studied the relation between different water constituents and coronary heart disease (CHD) among 45-64-year-old men. He found significant correlations between death rate from CHD and sulfate and bicarbonate, respectively.

Cardiomyopathy and Aluminum
Aluminum levels may be elevated in patients on hemodialysis. Although multifactorial, the mechanism of cardiac hypertrophy in patients with end-stage renal disease may involve direct aluminum cardiotoxicity (Reusch et al., 1998). Aluminum may be demonstrated by special stains in the myocardium of such patients.

Cardiomyopathy and Mercury
Evidence suggests that in cases of sporadic dilated cardiomyopathy, there are thousand-fold elevations of mercury in heart tissue (Frascati et al., 1999).

Dilated Cardiomyopathy and Selenium Deficiency
Selenium is an essential nutrient in trace quantities. It combines with cysteine as a component of selenoproteins, many of which have antioxidant properties. Rare endemic diseases occur where the soil is extremely low in selenium, specifically parts of China. These diseases include Keshan cardiomyopathy and Kashin-Beck disease, a deforming arthritis. High prevalence diseases, such as cancer and heart disease, may have a risk factor a relatively mild deficiency of selenium, such has been reported in Europe. The role of mildly low selenium intake in the development of reproductive disease, mood disorders, thyroid function, inflammatory disease, and cancer has been recently reviewed (Rayman, 2000).

Epidemiological studies have provided some evidence for the role of selenium deficiency in etiology of atherosclerotic disease. Angiographic studies suggest that low levels of selenium are associated with coronary stenosis (Yegin et al., 1997), and patients with acute myocardial infarction tend to have lower serum selenium compared to controls (Navarro-Alarcon et al., 1999). Plasma, red blood cell, and urine selenium concentrations have been shown to be decreased in patients with acute myocardial infarction.

A large prospective study in a Danish population demonstrated that men with the lowest tertile of blood selenium had a mildly increased risk for developing acute ischemic events of stroke or myocardial infarct.

Cardiovascular Diseases and Iron Overload
Epidemiologic observations have linked high dietary iron intakes or high iron stores with increased risk of coronary heart disease (Salonen et al., 1992). Clinical signs appear when body iron accumulates to about tenfold excess of normal: these include hepatic cirrhosis, diabetes, heart failure, arthritis and sexual dysfunction.

Iron overload may adversely affect the heart in patients with hemosiderosis or primary hemochromatosis. The most common cause of death in patients with hemochromatosis is from cardiac failure and arrhythmias followed by cirrhosis and hepatocellular carcinoma. Histologically, these changes are similar to dilated cardiomyopathy with abundant stainable iron present within myocytes. Most patients with hemochromatosis-related cardiomyopathy have a genetic predisposition to iron overload (primary hemochromatosis); significant cardiac failure in patients with hemosiderosis secondary to blood transfusions is rare.
Stroke Mortality Map
March 2003 - March 2004

1- Stroke high mortality and calcium low occurrence
2- Stroke high mortality and calcium low occurrence

Stroke & Calcium Deficiency

Calcium deficiency is common among the elderly, especially women. The absorption and renal conservation of calcium decreases with age. Several studies conducted in the United States have shown an intake lower than recommended, especially among women (Fleming & Heimbach, 1994). For individuals with a deficiency, the additional calcium from water could be crucial to prevent this. Along with the contribution of drinking water, cooking food in calcium-rich water has been shown not only to prevent leaching but even to increase calcium levels in food (Haring & van Delft, 1981).

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Calcium deficiency increases blood pressure which can lead to stroke.

Death of heart tissue due to blocked coronary artery

Heart Attack

Calcium Geochemical Map (Raw Data Distribution)
Anemia and Iron
Iron is an essential element in human nutrition but it can be toxic. Estimates of the minimum daily requirement of iron for humans depend on age, sex, physiological status, and iron bioavailability. The range is 10-18mg per day, 30 mg per day if pregnant (U.S. recommended daily allowance; RDA), and 14mg per day (EU RDA). Iron toxicity can occur at high levels of intake. The average lethal dose of iron is 200-250 mg kg^{-1} of body weight. However, even an oral intake as low as 40mg kg^{-1} of body weight has been lethal.

Manifestation of Iron Deficiency
Iron deficiency is manifested as hypochromic, normocytic anemia; lethargy; apathy; listlessness, fatigue, impaired non-shivering thermogenesis; impaired immune function; impaired cognitive development; and reduced physical performance. In pregnancy, iron-deficiency increases the risk of premature delivery, low birth weight, and infant and maternal mortality.

Copper
The human body contains approximately 120 mg of copper, which is widely distributed in many tissues and fluids at mgkg^{-1} or µgkg^{-1} concentrations. Copper absorption/retention varies inversely with the level of copper intake, and tends to be moderate (e.g., 50-60%) even at low copper intakes. Copper deficiency is manifested as hypochromic, normocytic, or macrocytic anemia; bone abnormalities resembling osteoporosis or scurvy; increased susceptibility to infection; and poor growth. The ingestion of high amounts of copper can cause nausea. Chronic high copper intake can lead to the hepatic accumulation of copper, which has been suspected in juvenile cases of hepatic cirrhosis in India.

Cobalt
Cobalt incorporates into vitamin B12 for normal methionine synthesis and energy metabolism. Two different forms of vitamin B12 are responsible for these two metabolic pathways: methylcobalamin for methionine synthesis and adenosylcobalamin for energy metabolism. Methylcobalamin is of importance for both protein and lipid synthesis, and in humans a vitamin B12 deficiency typically causes a specific megaloblastic anemia (pernicious anemia) and neurological disorders due to progressive demyelination. Vitamin B12 and neurological problems among elderly humans have attracted considerable interest lately.
**Diabetes and Chromium**

Chromium (Cr) potentiates the action of insulin and has been shown to restore glucose tolerance in malnourished infants. Several studies have shown that chromium supplementation lowers circulating glucose levels, increases plasma insulin, and produces a favorable profile of plasma lipids (Offenbacher et al., 1997).
Central Nervous System Diseases Mortality Map
March 2003 - March 2004

Central Nervous System and Mercury

It should be recognized that a number of metallic elements, such as iron, copper, and manganese, are essential to life and play an important role in the functioning of the central nervous system (CNS); nevertheless, that certain metals, such as lead and mercury, have neurotoxic properties has been acknowledged since ancient times. Compound in which the metal is linked to a lipophilic organic compound tend to be particularly neurotoxic as they can readily cross the lipid membranes that comprise the blood-brain barrier. One example is mercury, which in its inorganic form, is relatively nontoxic to the CNS; however, when mercury is methylated to form methylmercury, the compound rapidly crosses the blood-brain barrier, is readily taken up by neurons, and produces massive cellular destruction. This results in the severe parenchymal damage that occurred in the outbreak of severe CNS damage in Minamata, Japan. Finally it should be recognized that the effects of toxins that damaged neurons are particularly serious because of the inability of these cells to regenerate. Nevertheless, oral mercury-containing calomel medicinals have been used relatively safely over the centuries, and inorganic mercury has a relatively low level of toxicity. On the other hand, exposure to organic mercury, such as methylmercury, causes dramatic nervous system destruction (Hunter & Russel, 1954).

Central Nervous System and Tin

A phenomenon similar to that of enhanced neurotoxicity of methylmercury also occurs following exposure to tin-containing compounds. Metallic tin and its inorganic salts have been included in medicinal preparations since the 16th century. These were widely administered in relatively high doses without any apparent adverse health effects; however, organic tin compounds, are highly lipid soluble and are also highly toxic to the nervous system (Cavanagh & Nolan, 1994). Experimental works have shown that triethyltin produces a characteristic and selective white matter edema, presumably through its toxic effects on the functioning of oligodendroglial membranes.
Iron & Aluminum and Alzheimer

The accumulation of metals in the brain has also been associated with several of the age-related neurodegenerative disorders. The two metals most often cited are aluminum and iron. Excess amounts of both metals have been identified in microprobe studies within the characteristic neurofibrillary tangles of cases of Alzheimer’s disease (Perl & Brody, 1980; Good et al., 1992a). Evidence for oxidative damage in the brains of Alzheimer’s disease victims is well established (Markesbery, 1997). Aluminum is a highly charged element that binds strongly to proteins comprising the intraneuronal inclusions that characterize the neurodegenerative diseases (the neurofibrillary tangle being the most prominent example) are highly cross-linked and thus resistant to degradation. Whether aluminum serves this role in the natural history of the disorder remains unclear, although recent evidence appears to support this concept.
Encephalitis and Lead Poisoning

Lead is perhaps the most important metallic neurotoxin. Adults are relatively resistant to its effects, and only in high doses is a peripheral neuropathy encountered. In children, however, the effects of relatively low doses can be much more devastating. High doses in children can cause acute lead encephalopathy, a life-threatening condition characterized by generalized cerebral edema with increased intracranial pressure, which leads to transtentorial and cerebellar tonsillar herniation. Clinical features of acute lead encephalopathy may include ataxia, seizures, stupor, coma, and often death. Almost always other associated systemic signs of lead exposure, such as anemia and the presence of lead lines on x-rays of the long bones, are noted in affected children. At autopsy, the brain is markedly swollen with compressed gyri, obliterated sulci, and collapsed lateral ventricles. Largely through the work of Needleman and colleagues (1979, 1988, 1990), the long-term effects of lower doses of lead exposure on children have been increasingly recognized. This relates to the adverse effects of lead on intellectual functioning as well as its association with behavioral problems in children.
**Calcium & Skeletal-Muscular Disorders**

Impaired bone mineralization in young children results in deformities of the growing bones and is called rickets; in adults with formed bones it is called osteomalacia and is characterized by increase fracture risk and loss of stature. Only in very severe deficiency, when bone mineral has largely been exhausted, does calcium deficiency result in impaired nervous conduction and muscular contraction.