

A microscopic image showing a dense field of small, irregularly shaped particles. The particles exhibit a range of colors, including bright orange, red, and yellow, set against a dark blue background. The overall appearance is that of a complex, textured surface, possibly a biological or mineral structure.

A NOTE ON THE BIOLOGY OF COPPER

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Review Note: Biochemistry; Toxicology

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A note on the biology of copper

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Copper is often cited as an essential mineral. Copper-containing preparations are frequently employed in multi-mineral and multi-vitamin supplements. However, in what follows, we contend that the so-called ‘biological need for copper’ is a gratuitous and unfounded commonplace medical belief that is responsible (1) for the current lack of interest in researching the real biological effects of copper - which, in fact, are detrimental to health - and, likely, (2) for the slow and unwitting poisoning of people that consume such ill-advised supplements, placing them at risk for insidious neurological and sclerotizing diseases like Alzheimer’s disease or atherosclerosis. Indeed, no studies exist of the real effect of intake of copper via these supplements, and it is likely that copper is not the only heavy ion to which a caution of this nature applies.

There is a class of anemias that is often cited as evidence for the biological need for copper; they are referred to as copper-deficiency anemias precisely to indicate this assumed fact. However, the presumed etiology of these anemias stands upon an “inductive deduction”; and it happens to be the wrong one. For, in fact, what is assumed to be a lack of copper needed for nutrition has never been proven; it is inferred as such, when the evidence only permits one to state that *lack of the proper copper-binding proteins* needed to keep blood *free of copper*, leads to such anemias. They are, for the most part, and in effect, anemias caused by the resulting lack of defense - or protection of blood marrow - against poisoning by copper. Thus, the false notion that there is a nutritional requirement for copper is proof of the bogus nature of the national dietary requirements of the USDA. There is no such nutritional requirement. As an example of insidious pseudoscience, this imaginary requirement is inferred from *in vitro* studies of the electron transport chain in oxidative phosphorylation. *There - in the test tube* and *not* in a cellular system - completion of the respiratory chain and the remainder of the

required potential needs the addition of free copper, so that cytochrome a3 may bind oxygen noncovalently, and reduce it to water. The assumption has thus been made that the same applies *in vivo*, to the mitochondrion, where the electron shuttle is located. Nothing could be further from the truth, as our own work has suggested [1]: that end-point potential is precisely provided by ambipolar radiation, and it is the oxygen-cytochrome complex that functions as a resonant antenna capable of capturing ambipolar radiation of solar origin, in the 45.5 keV range. In the cell, we contended [1], the initiator and energy injector of the respiratory chain would be oxygen itself, once it is activated by ambipolar radiation - with no biological need for copper.

If we remove this apparent *ratio essendi* of the (imaginary) need for copper, then it is plain to see that copper is - like arsenic - simply a poison [2]. Indeed, this much is well-established - that copper is a heavy metal whose unbound ions are toxic, whether they are absorbed gastro-intestinally, through the skin or breathed in. Likely, substantial exposure to copper since the Mesolithic period has promoted biological, enzyme-mediated responses geared to bind copper and direct it to excretion via the bile. The rationale of this biological response is obvious - by binding to copper, its biological toxicity can be decreased. If copper - like aluminum - has no role in normal cellular metabolism, then these adaptive responses are strictly defenses against the toxicity of copper.

Little wonder, then, that the centennial edition of the Merck Manual could only conclude with a verbose euphemism: "In genetically-normal people, acquired, environmental or dietary abnormalities rarely cause clinically significant copper-deficiency" [3]. It is so rare that it has never been documented, demonstrated or encountered - other than in the imagination of unquestioning physicians or crackpot nutritionists. The imaginary etiology of so-called 'kwashiorkor disease' is not acquired copper deficiency - as its original reports suggested - but *excess intake of zinc salts*. Treatment with copper ions is merely a poor man's exchange of copper for the excess zinc. So-called inherited copper deficiency is another bogus construct of uncritical medicine. Patients with this diagnosis (Menke's syndrome) essentially suffer from deficient copper-binding proteins - such as ceruloplasmin and lysyl oxidase. Cytochrome c oxidase deficiency is also classified as a cause of copper-deficiency, but it is plain to see that if copper is not involved in the respiratory chain, the genetic deficiency in cytochrome c oxidase is simply a deficiency in an enzyme that is critical to complete the respiratory chain - critical, in fact, for the proper cyclic redox alteration of cytochrome a3, and thus the completion of the electron chain's last three steps (and not just the last step which is the step that supposedly would require copper). Wilson's disease (Inherited Copper Toxicosis) is also just a genetic deficiency of ceruloplasmin that, if diagnosed in time, precisely requires lifelong treatment with zinc salts to leach out any internal copper.

In the absence of copper-binding proteins that leach copper from blood and direct it towards

excretion, copper toxicity is all the more acute and intense. No treatment exists for Menke's syndrome, and the same Merck Manual acknowledges that it is unlikely that copper histidinate has any therapeutic value. But even in the presence of copper-binding proteins, copper toxicosis can and does result, reaching lethal doses when fractional gram quantities are ingested. Early stages of copper toxicosis present nausea, vomiting and diarrhea. These are brought about by milligram ingestion of copper, either in food or drink, or by inhalation or contact. Copper contamination of food is commonly the result of employing copper cookware, in particular to heat and cook with milk, milk products and fats. Likewise, copper plumbing leads to the contamination of tap water used for drinking or cooking. Copper contamination of wines and spirits is also frequently encountered (a common source are copper distillers). Inhalation of copper is commonly the result of copper aerosols present in paints, or produced during welding and soldering. Cigarette paper and mark labels are often contaminated with copper. Contact with copper piping, copper sheets, etc, leads to absorption by the skin - repeated contact leading to toxicosis. Copper toxicity progresses from gastrointestinal symptoms to more serious symptoms, such as hemolytic anemia and anuria. Thus, it is not the deficiency of copper that causes anemia; rather, deficiency of copper-binding proteins results in intoxication with copper - which happens faster than it would in the presence of such proteins. It is one and the same copper intoxication that leads to hemolytic anemia.

Moreover, in the past 2 decades, there has been a slow awakening to the toxic effects of copper in the brain and the CNS. In half the cases diagnosed with Wilson's disease, the toxicosis affects the CNS, causing motor impairment, tremors, incompetence and discoordination. Psychotic states indistinguishable from severe manic-depression are often early signs of the advanced degeneration. In the late 1980's, histopathology studies had suggested a link between Alzheimer's disease (AD) and copper [4] and aluminum [5] toxicities. Current views have largely discarded these results in what concerns aluminum ingestion [6], concentrating instead on the existence of genetic factors that present an increasing risk of AD [7]. But, precisely, while these studies eliminated aluminum from causation of AD, they failed to do so for copper.

Recent research has now confirmed the etiological role of copper in causing AD, by experimentally inducing beta-amyloid accumulations in the CNS neurons of rabbits fed with cholesterol and trace amounts of copper in tap water. The affected rabbits also presented senile plaque-like structures in the hippocampus and the temporal lobe, identical in all respects to those found in AD [8]. By 2005, the main mechanism of plaque production characteristic of AD had been identified: Cu^{2+} forms a redox-active complex with the amyloid beta-peptide, and this catalyzes the *in situ* formation of H_2O_2 from oxygen and cholesterol, and mimics the action of cholesterol oxidase [9]. Undoubtedly, copper will play a similar or parallel role in other degenerative diseases related to high cholesterol,

such as hypertension and vascular diseases. In neuronal cultures, the toxicity is inhibited by copper chelators, and the pathogenic mechanism is likely common to AD and atherosclerosis [9]. Cholesterol-oxide derivatives are even more reactive to the presence of copper, resulting in accelerated amyloidogenesis in AD. Lactate dehydrogenase (LDH) sensitivity to copper has also been implicated in Huntington's disease (HD), where increased brain copper correlates with decreased levels of the amyloid precursor protein [10].

From the preceding, one can conclude that the evidence for copper being a poison is now established beyond doubt, whereas the evidence for the nutritional requirement of copper remains devoid of any actual foundation. Obviously, consumers of health products should be made aware of the tremendous risks they are incurring when consuming preparations that contain copper. This is one more instance where medicine and medical research has lagged behind for no good reason, other than the social strength of irrational belief. Once again, this proves to be the greatest enemy of science and public health.

Post Scriptum: Aside from the wanton and unwarranted decades-old practices of wilfully poisoning wines with metabisulfites (to "give body" and "taste"), there are even more insidious practices of contamination with heavy metals, in particular, copper and lead. Thus the relevance of the following article, linked below, to the present review note -

http://www.aetherometry.com/External/newsmax_on_wine.html

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