DIETS DEFICIENT IN COPPER AND ZINC?

Leslie M. Klevay. United States Department of Agriculture, Science and Education Administration, Human Nutrition Laboratory, P.O. Box 7166, University Station, Grand Forks, North Dakota, 58202, U.S.A.

ABSTRACT

Hospital diets, and, perhaps, diets in general may be low in copper and zinc, with the diets being lower in copper than zinc in comparison to requirements. Several human diseases or pathologic conditions of unknown etiology have similarities to findings in deficient animals. Thus nutritional status and metabolism of copper may be important in anemia, ischemic heart disease, rheumatoid arthritis, osteoporosis, multiple sclerosis and seborrheic dermatitis. Zinc may be involved in growth failure, acne, difficulties of labor, congenital abnormalities and wound healing. Whether or not dietary amounts of copper and zinc are involved in the etiology or the pathogenesis of these conditions can be determined by clinical investigation.

Key Words acne, copper, labor complications, multiple sclerosis, osteoporosis, trace elements, wound healing, zinc

INTRODUCTION

Hospital diets and, perhaps, diets in general may be low in copper and zinc, with the diets being lower in copper than zinc in comparison to requirements. This conclusion is based on measurements of the daily amount of copper and zinc in diets prepared in a local hospital and on analyses of diets done in several other States (1). For 20 diets, the median daily amounts of copper and zinc, 0.78 and 12.0 mg, were lower than the respective adult requirements, 2.0 mg and 12.5 mg (2,3). Current estimates of requirements are minimal estimates: the amounts needed to replace loss via feces and urine.
If diets frequently are low in copper and/or zinc, it is important to consider the possibility that common dietary amounts of these elements may cause health problems. Natural and controlled experiments with animals can serve as guides for human pathology; however, one should not expect anatomical and chemical lesions of animals deficient in copper or zinc to be identical with the findings in human diseases of unknown etiology. One must allow for differences among species and for the myriad environmental factors that may influence the development of human pathology.

ANIMAL DEFICIENCIES AND HUMAN DISEASES

Several human diseases or pathologic conditions of unknown etiology have similarities to findings in deficient animals. Among the more important abnormalities produced in animals by copper deficiency are (4): (a) anemias, (b) defects in connective tissue leading to abnormalities of arteries and bone and (c) degeneration of brain and spinal cord secondary to abnormal myelinization, (d) myocardial degeneration. Zinc deficiency has produced (4): (a) growth impairment and delayed maturity, (b) skin lesions and (c) reproductive failure and fetal abnormalities.

Anemia is common; most anemia is thought to be due to iron deficiency. Apparently no one has investigated the role of copper in anemia of ambulatory adults since Cartwright (5) stated, in 1947, that "The value of copper in the treatment of hypochromic microcytic anemia in adults is controversial".

The etiology of ischemic heart disease remains obscure. An imbalance in zinc and copper metabolism has been associated with many apparently dissimilar observations on the epidemiology of ischemic heart disease and the metabolism of cholesterol (6,7). Copper deficiency produces hypercholesterolemia and myocardial fibrosis in rats (8); myocardial fibrosis also is found in cattle deficient in copper (4).

Although many hypotheses have been proposed and many potentially causal agents have been studied, the origins of rheumatoid arthritis and of osteoporosis are unknown. Sorensen (9) has suggested that the active forms of antiarthritic compounds used clinically are copper chelates formed in vivo.

Multiple sclerosis is of unknown etiology, although several hypotheses have been proposed. The metabolism of unsaturated fatty acids in multiple sclerosis seems to be abnormal (10,11). Experiments with rats have implicated copper in the metabolism of these acids (4). Similarly, in the ceroid-lipofuscinoses an abnormality of superoxide dismutase (10), an enzyme containing copper, has been suggested. Interestingly, in Menkes' syndrome, in which intestinal absorption of copper is impaired, abnormalities of myelination are not prominent (12).
Whether or not this finding is suggestive of a lack of involvement of copper in multiple sclerosis or the ceroid-lipofuscinoses remains to be determined.

Hambidge et al. (13) found laboratory evidence of zinc deficiency in some small children. After treatment with zinc sulfate, test results moved toward or into the normal range.

A symmetrical, vesiculopustulobulbous eruption is characteristic of acrodermatitis chronica enteropathica, an apparently hereditary disease which can be treated successfully with zinc sulfate (14). Treatment of acne with oral zinc sulfate has produced improvement (15). Seborrheic dermatitis is found in neonatal copper deficiency (16) and Menkes' syndrome (13).

Low serum zinc values early in pregnancy are associated with difficulties of labor and congenital abnormalities (17). Low serum zinc values are associated with impaired wound healing; healing improved with zinc therapy (18).

CONCLUSIONS

Thus there are several diseases and pathological conditions in which one may suspect the involvement of copper or zinc. Whether or not dietary amounts of these elements are involved in the etiology or the pathogenesis of these conditions remains to be seen. Perhaps dietary amounts of these elements are important only as modifiers of abnormalities of absorption, transport or utilization. Once these possibilities are considered, experiments using the usual methods of clinical investigation will provide the answers.

REFERENCES


