

Uauy, R., M. Olivares, and M. Gonzalez. 1998. Essentiality of copper in humans. *Am.J Clin.Nutr.* 67:952S-959S.

Abstract: The biochemical basis for the essentiality of copper, the adequacy of the dietary copper supply, factors that condition deficiency, and the special conditions of copper nutriture in early infancy are reviewed. New biochemical and crystallographic evidence define copper as being necessary for structural and catalytic properties of cuproenzymes. Mechanisms responsible for the control of cuproprotein gene expression are not known in mammals; however, studies using yeast as a eukaryote model support the existence of a copper-dependent gene regulatory element. Diets in Western countries provide copper below or in the low range of the estimated safe and adequate daily dietary intake. Copper deficiency is usually the consequence of decreased copper stores at birth, inadequate dietary copper intake, poor absorption, elevated requirements induced by rapid growth, or increased copper losses. The most frequent clinical manifestations of copper deficiency are anemia, neutropenia, and bone abnormalities. Recommendations for dietary copper intake and total copper exposure, including that from potable water, should consider that copper is an essential nutrient with potential toxicity if the load exceeds tolerance. A range of safe intakes should be defined for the general population, including a lower safe intake and an upper safe intake, to prevent deficiency as well as toxicity for most of the population.