Zinc deficiency in human subjects.

During the past two decades, the essentiality of zinc for man has been established. Deficiency of zinc in man due to nutritional factors and several diseased states has been recognized. High phytate content of cereal proteins decreases availability of zinc; thus the prevalence of zinc deficiency is likely to be high in a population subsisting mainly on cereal proteins. Alcoholism is known to cause hyperzincuria and thus may play a role in producing zinc deficiency in man. Malabsorption, cirrhosis of the liver, chronic renal disease and other chronically debilitating diseases may similarly induce zinc deficiency in human subjects. A severe deficiency of zinc has recently been recognized to occur in patients with sickle cell anemia and a beneficial effect of zinc therapy in such patients has been reported. Growth retardation, male hypogonadism, skin changes, poor appetite, mental lethargy and delayed wound healing are some of the manifestations of chronically zinc-deficient human subjects. Taste abnormalities, correctable with zinc supplementation, have been observed in uremic subjects. Recently, abnormal dark adaptation related to zinc deficiency in patients with cirrhosis of the liver and sickle cell disease has been reported. In severely zinc-deficient patients, dermatological manifestations, diarrhea, alopecia, mental disturbances and intercurrent infections predominate and if untreated the condition becomes fatal. Zinc deficiency is known to affect testicular functions adversely in man and animals. This effect of zinc is at the end organ level and it appears that zinc is essential for spermatogenesis and testosterone steroidogenesis. Zinc is involved in many biochemical functions. Several zinc metalloenzymes have been recognized in the past decade. Zinc is required for each step of cell cycle in microorganisms and is essential for DNA synthesis. Thymidine kinase, RNA polymerase, DNA-polymerase from various sources and RNA-dependent DNA polymerase from viruses have been shown to be zinc-dependent enzymes. Zinc also regulates the activity of RNase; thus the catabolism of RNA appears to be zinc-dependent. The effect of zinc on protein synthesis may be attributable to its vital role in nucleic acid metabolism. The activities of many zinc-dependent enzymes have been shown to be affected adversely in zinc-deficient tissues. Three enzymes, alkaline phosphatase, carboxypeptidase and thymidine kinase, appear to be most sensitive to zinc restriction in that their activities are affected adversely within three to six days of institution of a zinc-deficient diet to experimental animals.