31. Molybdenum

Physiology

Molybdenum has several oxidation states. The redox potential between Mo(V) and Mo(VI) is appropriate for electron exchange with flavinmononucleotides and this is exploited biochemically in the activities of sulphite oxidase, xanthine oxidase and aldehyde dehydrogenase, for which molybdate (Mo_4^{2-}) linked with a pterin is a cofactor. Intestinal absorption of dietary molybdenum is highly efficient (approximately 80 %). The element is metabolised as an anion (molybdate) and systemic homoeostatic excretion is attained by renal excretion [1,2].

Molybdenum deficiency in man has occurred with prolonged parenteral feeding [3], and in a fatal autosomal recessive syndrome affecting infants in whom the hepatic synthesis of the molybdenum-pterin cofactor is probably defective [2]. With both conditions the metabolism of sulphur amino acids and nucleotides was impaired, and neurological and encephalopathies developed. The inborn error of metabolism presents in neonates: they have abnormal faeces, feeding difficulties, and severe neurological and developmental abnormalities leading to encephalopathy and death.

Requirements

Molybdenum-responsive defects have been observed in adults fed about 50µg daily [4]. Reported dietary intakes in adults are 44-460 µg/d in USA, 48-96 µg/d in New Zealand, 44-260 µg/d in Sweden and mean intakes of 128 and 120 µg/d in the United Kingdom and Finland respectively [1]. A requirement cannot be established reliably, and, in the absence of evidence to the contrary, current intakes appear to be adequate and safe.

References

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