13. Pantothenic acid

Physiology and metabolism

Pantothenic acid is required for the synthesis of coenzyme A (CoA), which is required in the metabolism of carbohydrates, amino acids and fatty acids and the synthesis of steroids and other metabolites, and for the synthesis of the prosthetic group of acyl carrier protein required for fatty acid synthesis. It thus has a central role in a wide variety of metabolic pathways [1].

About 85% of dietary pantothenic acid is as CoA or phosphopantotheine. In the intestine these undergo hydrolysis to pantothenic acid, which is absorbed by diffusion throughout the small intestine.

All tissues are capable of forming CoA and the prosthetic group of acyl carrier protein from pantothenic acid. This synthesis requires the amino acid cysteine, and deficiency of the sulphur amino acids, methionine and cysteine, can result in impaired synthesis and hence secondary functional pantothenic acid deficiency. Phosphopantotheine, arising from the catabolism of CoA and acyl carrier protein, can be reused for CoA synthesis and new synthesis of acyl carrier protein. Phosphopantotheine which is not re-utilised is dephosphorylated, and the resultant pantotheine is cleaved to pantothenic acid and cysteamine.

Pantothenic acid is well conserved in the body; over a week after administration of tracer doses of [^{14}C] pantothenic acid only some 38% of the dose is recovered in the urine, all as the free vitamin. There is no information on body reserves of pantothenic acid.

Deficiency

Pantothenic acid is widely distributed in foods, and it is possible that intestinal bacterial synthesis also makes a contribution to intake. Deficiency has not been unequivocally reported in human beings except in specific depletion studies, which have frequently also used the antagonist methyl pantothenic acid.

Prisoners of war in the Far East, who were severely malnourished, showed, among other signs and symptoms of vitamin deficiency diseases, a new condition of paraesthesia and severe pain in the feet and toes, which was called the 'burning foot'

syndrome or nutritional melalgia. Although it was attributed to pantothenic acid deficiency, no specific trials of pantothenic acid were carried out; rather the subjects were given yeast extract and other rich sources of all vitamins as part of an urgent programme of nutritional rehabilitation.

Experimental pantothenic acid depletion, normally together with the administration of the antagonist methyl pantothenic acid, results in neuromotor disorders, including paraesthesia of the hands and feet, hyperactive deep tendon reflexes and muscle weakness, as well as mental depression, gastrointestinal complaints and metabolic abnormalities which can be attributed to changes in lipid metabolism [2].

Requirements

From the limited studies which have been performed it is not possible to establish requirements for pantothenic acid. Average intakes in adults are about 4-7 mg/d, but individuals consume 3-12 mg/d. Such intakes are obviously adequate to prevent deficiency; much higher amounts are harmless. There is no evidence on which to base estimates of changed pantothenic acid requirements in pregnancy or lactation.

Summary

Average intakes of pantothenic acid are adequate to meet requirements. There is no information on intakes below which deficiency is likely, nor adequate evidence to determine Population Reference Intakes.

The acceptable range of intakes in adults is the observed range 3 - 12 mg/d.

References

1. Bender DA. (1992). *Nutritional Biochemistry of the Vitamins.* Cambridge: Cambridge University Press, 341-360.

2. Fry PC, Fox HM, Tao HG. (1976). Metabolic responses to a pantothenic acid deficient diet in humans. *J Nutr Sci Vitaminol (Tokyo),* **22:** 339-346.