Vitamin B5 | Pantothenic acid

Summary of benefits:

Required for red blood cell production (87-89).

Supports the production of sex and stress-related hormones in the adrenal glands (90-96). Helps form coenzyme A (CoA) which is involved in metabolism and energy production. Since Pantothenic acid supports fat metabolism, it might aid in reducing cholesterol levels.

Since Pantothenic acid supports fat metabolism, it might aid in reducing cholesterol levels in people with dyslipidemia (131).

Functions as an antioxidant and reduces low-grade inflammation (131).

Pantothenic acid, also know as vitamin b5, plays a crucial role in the synthesis of coenzyme A (CoA) and acyl carrier protein [33,34]. CoA is a molecule essential for the proper functioning of numerous enzymes in the body and for overall energy production.

CoA plays a crucial role in fatty acid synthesis (primarily via acyl carrier protein (34)) and degradation, transfer of acetyl and acyl groups, and numerous anabolic and catabolic reactions [35,36].

Pantothenic acid plays a vital role in the manufacturing of red blood cells, as well as sex and stress-related hormones produced in the adrenal glands (small glands that sit atop the kidneys) (87-96).

Albeit, deficiencies are relatively uncommon, The recommended dietary allowance (RDA) for vitamin b5 is 5 milligrams (mg) for adult men and women 19 years and older.

Pantothenic acid is found in a wide range of plant and animal food sources. Some include...

- Fortified cereals
- Organ meats (liver, kidney)
- Beef
- Chicken breast
- Mushrooms
- Avocados
- Nuts, seeds
- Dairy milk
- Yogurt
- Potatoes
- Eggs
- Brown rice
- Oats
- Broccoli (33, 131, 132)

Approximately 85% of dietary Pantothenic acid is present in the form of CoA or phosphopantetheine [34,36]. Digestive enzymes (such as nucleosidases, peptidases, and phosphorylases) in our cells and those of the intestinal lumen convert these forms into Pantothenic acid. The absorption of pantothenic acid occurs in the intestines where it enters the bloodstream via active transport (and possibly simple diffusion at higher doses) [33,34,36]. Before entering the bloodstream however, pantetheine, the dephosphorylated form of phosphopantetheine, is taken up by intestinal cells and converted to pantothenic acid [34]. The intestinal flora also produces pantothenic acid, but its contribution to the total amount absorbed by the body remains uncertain [36]. Pantothenic acid is carried by red blood cells throughout the

body, primarily as CoA, while smaller amounts exist in the form of acyl carrier protein or free pantothenic acid [33,36].

Concentration Measurement and Deficiency:

Routine measurement of pantothenic acid status is not typically performed in healthy individuals. Methods such as microbiologic growth assays, animal bioassays, and radioimmunoassays can be utilized to measure pantothenic acid concentrations in blood, urine, and tissues. Among these, urinary concentrations are considered the most reliable indicators due to their close association with dietary intake [33,36]. In individuals following a typical American diet, the average urinary excretion rate of pantothenic acid is approximately 2.6 mg/day [35,37]. Excretion of less than 1 mg of pantothenic acid per day suggests a deficiency [33,38]. Whole-blood concentrations of pantothenic acid correlate with pantothenic acid intake but require enzyme pretreatment to release free pantothenic acid from CoA [33]. Normal blood concentrations of pantothenic acid range from 1.6 to 2.7 mcmol/L, and levels below 1 mcmol/L are considered low and indicative of deficiency [33,36]. Plasma levels of pantothenic acid, unlike whole-blood concentrations, do not exhibit a strong correlation with changes in intake or status [32,33].

Dosage Rationale:

Although pantothenic acid is extremely important for its aforementioned purposes, deficiency is unlikely and obtaining ample amounts from the diet should render additional supplementation unnecessary. That being said, we provide the daily recommended intake of pantothenic acid in our Super U formula to reduce the likelihood of deficiency. Additionally, since we recommend it be taken without food first thing in the morning, it will ensure the daily intake and necessary component of energy metabolism has been replenished in the fasted state prior to feeding.

It's important to note that while these statements are based on available information in the scientific literature, it is always advisable to consult with a healthcare professional before making any changes to your supplementation or health routine.

REFERENCES:

33. Miller JW, Rucker RB. Pantothenic acid. In: Erdman JW, Macdonald IA, Zeisel SH, eds. Present Knowledge in Nutrition. 10th ed. Washington, DC: Wiley-Blackwell; 2012:375-90.

34. Sweetman L. Pantothenic acid. In: Coates PM, Betz JM, Blackman MR, et al., eds. Encyclopedia of Dietary Supplements. 2nd ed. London and New York: Informa Healthcare; 2010:604-11.

35. Institute of Medicine. Food and Nutrition Board. Dietary Reference Intakes: Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. Washington, DC: National Academy Press; 1998.

36. Trumbo PR. Pantothenic acid. In: Ross AC, Caballero B, Cousins RJ, et al., eds. Modern Nutrition in Health and Disease. 11th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2014:351-7.

87. Ellinger S, Stehle P. Efficacy of vitamin supplementation in situations with wound healing disorders: results from clinical intervention studies. Curr Opin Clin Nutr Metab Care. 2009 Nov;12(6):588-95. Review.

88. Joncyzk R, Ronconi S, Rychlik M, Genschel U. Pantothenate synthetase is essential but not limiting for pantothenate biosynthesis in Arabidopsis. Plant Mol Biol. 2008;66(1-2):1-14.

89. Konings EJ; Committee on Food Nutrition. Water-soluble vitamins. JAOAC Int. 2006 Jan-Feb;89(1):285-8.

90. McCarty MF. Inhibition of acetyl-CoA carboxylase by cystamine may mediate the hypotriglyceridemic activity of pantethine. Med Hypotheses. 2001;56(3):314-317.

91. McPherson. Henry's Clinical Diagnosis and Management by Laboratory Methods, 22nd ed. Philadelphia, PA: Saunders, An Imprint of Elsevier. 2011.

92. Naruta E, Buko V. Hypolipidemic effect of pantothenic acid derivatives in mice with hypothalamic obesity induced by aurothioglucose. Exp Toxicol Pathol. 2001;53(5):393-398.

93. National Academy of Sciences. Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Vitamins. Accessed June 1, 2011.

94. Nutrients and Nutritional Agents. In: Kastrup EK, Hines Burnham T, Short RM, et al, eds. Drug Facts and Comparisons. St. Louis, Mo: Facts and Comparisons; 2000:4-5.

95. Pins JJ, Keenan JM. Dietary and nutraceutical options for managing the hypertriglyceridemic patient. Prog Cardiovasc Nurs. 2006 Spring;21(2):89-93. Review.

96. Scheurig AC, Thorand B, Fischer B, Heier M, Koenig W. Association between the intake of vitamins and trace elements from supplements and C-reactive protein: results of the MONICA/KORA Augsburg study. Eur J Clin Nutr. 2007 Feb 21; [Epub ahead of print]

131. Pantothenic acid – vitamin B5. The Nutrition Source. (2023b, March 7). https://www.hsph.harvard.edu/nutritionsource/pantothenic-acid-vitamin-b5/

132. U.S. Department of Health and Human Services. Pantothenic Acid Fact Sheet for Health Professionals. <u>https://ods.od.nih.gov/factsheets/PantothenicAcid-HealthProfessional/</u>. Accessed 2/3/20.