USANA Technical Bulletin

Niacin and Niacinamide

Technical Background

- Niacin (nicotinic acid) and its derivative niacinamide (nicotinamide or nicotinic acid amide) are two forms of a water soluble vitamin that has been referred to as vitamin B_3. (Note that the name "niacin" is often used in the generic sense to refer to both nicotinic acid and its nicotinamide derivative.)[1]
- Both compounds are precursors for the active forms of important enzyme cofactors (coenzymes) that assist in catalyzing oxidation-reduction reactions in the cell.[2] Such oxidation-reduction reactions are fundamental to cellular energy production and utilization, steroid biosynthesis, DNA replication and repair, cellular differentiation, and the general metabolism of carbohydrates, amino acids, and fatty acids.[1]
- Niacin is also a structural component of Glucose Tolerance Factor (GTF), a complex that improves the binding of insulin to cell surface receptors.[3]
- Consequently, niacin has been found to address symptoms of diabetes. In patients with dyslipidemia, niacin decreased triglycerides, LDL cholesterol, and lipoproteins.[4]
- Niacin may also have non-invasive cancer-fighting abilities.[5,6]
- Deficiencies in niacin and niacinamide cause pellagra, a disorder with broad spectrum symptoms including inflammation of the skin and mucous membranes, diarrhea, and neurological dysfunction (confusion, disorientation, anxiety, and insomnia).

Sources and Recommended Intake

- Niacin is provided by most foods. Good sources include meats, cereals, legumes, milk, green leafy vegetables, fish, and yeast products.
- In general, free niacin and niacinamide are readily absorbed in the intestines, even in gram quantities. In plant-based foods, however, some niacin may be bound to macromolecules in forms unavailable for intestinal absorption.[1]
- Most animals, including humans, can synthesize niacin from biochemical precursors, particularly the amino acid tryptophan. Additional niacin may be synthesized by intestinal bacteria.[1]
- The Recommended Dietary Allowance (RDA) for niacin and niacinamide is expressed in niacin equivalents (NE: 1 NE = 1 mg niacin or niacinamide). RDA's have been set at 14 mg per day of NE for women and 16 for men.[7] Part of this requirement can be met by dietary tryptophan, although it takes 60 mg of tryptophan to provide 1 NE.[8]
- Average diets contain significant quantities of niacin and tryptophan, and they typically provide adults with 30-40 NE per day.[8]

- Ingestion of niacin (nicotinic acid), even at moderate doses, may produce vascular dilation or flushing. This reaction does not occur with niacinamide.[1]

Abstracts

Wieneke H, Schmermund A, Erbel R. [Niacin--an additive therapeutic approach for optimizing lipid profile]. Med Klin (Munich). 2005 Apr 15;100(4):186-92. BACKGROUND: Large interventional studies have shown that the reduction of total cholesterol and low-density lipoprotein cholesterol (LDL-C) is one of the cornerstones in the prevention of coronary artery disease. However, in up to 40% of patients the recommended target of LDL-C is not reached with a monotherapy. Furthermore, risk stratification only by LDL-C disregards a substantial number of patients with dyslipidemia with increased triglycerides and decreased high-density lipoprotein cholesterol (HDL-C). EFFECT OF NIACIN ON LIPID METABOLISM: In consequence, niacin has gained attention as a component of a combined therapeutic approach in patients with dyslipidemia. Niacin substantially increases HDL-C and decreases triglycerides, LDL-C and lipoprotein (a). By this mechanism of action niacin exhibited, in combination with statins or bile acid-binding resins, favorable effects on the incidence of cardiovascular events in selected patients. Side effects like flush and hepatotoxicity seem to be in part dependent on the niacin formulations used. However, niacin has been shown to be a well-tolerated and safe therapy in controlled studies. CONCLUSION: On the basis of current data niacin should be considered a valuable therapy component in patients with dyslipidemia, in which a monotherapy fails to optimize an increased risk of coronary artery disease.

References

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