Niacin

Niacin		Women	Men	Children		
				2-5 y	6-9 y	10-13 y
	NE/d					girls / boys
Recommended intake	RI	15	18	9	12	14 / 16
Average requirement	AR	12	15			
Lower intake level	LI	9	12			
Upper intake level	UL	35*	35*			

*as nicotinic acid

Introduction

Niacin is the common term for nicotinic acid and nicotinamide and derivates that exhibit the biological activity of nicotinamide. Niacin has its main function in the form of the coenzymes NAD (nicotinamide adenine dinucleotide) and NADP (nicotinamide adenine dinucleotide phosphate), which are involved in a number of redox reactions in the metabolism of glucose, amino acids and fatty acids.

Dietary sources and intake

Preformed niacin occurs in foods such as meat, fish, and pulses. Protein-rich foods also contribute to the niacin intake through conversion from tryptophan. The diet in the Nordic countries provides 30-40 NE/10 MJ (see Chapter XX Dietary intake in Nordic countries).

Physiology and metabolism

In foods, niacin mainly occurs as NAD and NADP, which are effectively hydrolysed and absorbed in the intestine (SCF2002; Said2011). Data from human studies indicate near complete absorption of up to 3 grams of nicotinic acid. In cereals, e.g. maize, niacin can be present in a form considered to be less available, e.g. esterified to polysaccharides (van den Bergh 1997). Alkaline treatment during preparation releases much of the niacin.
In the body niacin is formed from tryptophan. On average, 60 mg of dietary tryptophan is estimated to give rise to 1 mg niacin (= niacin equivalent, NE). The body has a limited capacity for storing niacin nucleotides and deficiency symptoms can occur after 50-60 days of consumption of a low-niacin corn-based diet (National Research Council 1989).
Niacin status can be measured by urinary excretion of certain metabolites, e.g. N'-methyl-nicotinamide and methyl pyridone carboxamides.

Niacin deficiency results in pellagra, mainly observed in populations consuming a diet predominantly based on maize or other cereals with a low protein content and low bioavailability of niacin. Few controlled studies including small numbers of subjects, have investigated

effects of niacin-restricted diets (see National Research Council 1989, Powers 1999). In one controlled study, pellagra developed at an intake of 8.8 NE/d (National Research Council 1989). In two other studies no clinical symptoms were seen in subjects with an intake of 9.2-12.3 NE per day, equivalent to about 1 NE/MJ (National Research Council 1989).

Requirement and recommended intake

In the absence of new scientific data the reference values for niacin given in NNR 2004 are kept unchanged. The average requirement is set to 1.3 NE/MJ based on studies in which niacin status has been assessed using urinary excretion of niacin metabolites, which is considered to be an appropriate marker (Powers 1999). The recommended intake is set to 1.6 NE/MJ. This corresponds to an intake of 17-19 NE/d for adult men and 14-15 NE/d for adult women. However, when planning diets the niacin content should not be lower than 13 NE/d, even at energy intake below 8 MJ/d. For pregnant women an extra 1-2 NE/d, and for lactating women an extra intake of 4-5 NE/d is recommended, which is based on the niacin content of breast milk and the increased energy requirement.

For infants and children over 6 months of age, the recommended intake for adults is applied. The lower limit of intake is estimated to be 1 NE/MJ. At energy intakes below 8 MJ/d the lower limit is estimated to be 8 NE/d.

Reasoning behind the recommendation

The focus of interest for niacin requirements over the last decade has been as a ‘drug’ for the treatment of various dyslipidemias. The reference values for niacin given in NNR 2004 are kept unchanged as there are no new scientific data.

Upper intake levels and toxicity

There are no studies indicating adverse effects of consumption of naturally occurring niacin in foods. Intakes of nicotinic acid, but not nicotinamide, as a supplement or fortificant in the range 30-1000 mg/d can result in mild symptoms such as flushing. Higher intakes have been reported to induce liver damage. The US Food and Nutrition Board (National Research Council 1989) set an upper limit of 30-35 mg/d for adolescents and adults, based on the risk of flushing. For children 1-3 years, FNB set the UL to 10 mg/d, for 4-8 years 15 mg/d and for 9-13 years 20 mg/d.

The EU Scientific Committee for Food (2002) has proposed an upper limit for nicotinic acid of 10 mg/d and for nicotinamide of 900 mg/d for adults. These levels are also used in NNR.

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