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## 11 Thiamin

Thiamin mg/d		Women	Men	Children		
				2-5 y	6-9 y	10-13 y girls/boys
Recommended intake	RI	1.1	1.4	0.6	0.9	1.0/1.2
Average requirement	AR	0.9	1.2			
Lower intake level	LI	0.5*	0.6*			
Upper intake level	UL	ND	ND			

\*0.8 mg at energy intakes <8 MJ/d

12  
13

### 14 Introduction

15 Thiamin (vitamin B<sub>1</sub>) is essential for the utilisation of carbohydrates and branched-  
16 chain amino acids in the body. In the metabolism thiamin participates in the form of  
17 thiamin pyrophosphate (TPP, or TDP thiamine diphosphate) as a coenzyme for  
18 pyruvate dehydrogenase, transketolase and  $\alpha$ -ketoglutarate dehydrogenase in the  
19 oxidative decarboxylation of  $\alpha$ -keto acids to aldehydes and in the utilisation of  
20 pentoses (Butterworth 2006, Bender 1999). TPP is also a coenzyme for keto acid  
21 dehydrogenase in the metabolism of branched chain amino acids (Bender 1999).  
22 Thiamin triphosphate (TTP) is involved in nerve and possibly muscle function (Bender  
23 1999).

24

### 25 Dietary sources and intakes

26 Major food sources of thiamin in the Nordic diet are cereals and cereal products, meat  
27 and meat products and milk products. The dietary supply of thiamin in the Nordic  
28 countries is 1.3-1.6 mg/10 MJ (see Chapter XX: Intake of vitamin and minerals in the  
29 Nordic countries).

30

### 31 Physiology and metabolism

32 In vegetable foods, thiamin occurs mainly in the free form and in animal foods mainly  
33 in phosphorylated forms, which are converted to free thiamine prior absorption  
34 (Gregory 1997, Said 2011). Absorption takes place in the small intestine, generally via  
35 an active, carrier mediated system involving phosphorylation. At high intakes passive  
36 diffusion also takes place (Zielinska Dawidziak et al 2008, Smithline et al 2012).  
37 Thiamin is also obtained from bacterial sources, provided by the normal microflora of  
38 the large intestine and it is absorbed in that region of the gut (Said 2011), although the  
39 quantitative importance of this source is uncertain. Studies with <sup>14</sup>C labelled thiamin in  
40 young men (Ariaey Nejad et al 1970) showed that more than 95 % of the vitamin was

41 absorbed at intakes of 1-2 mg/d. At intakes above 5 mg/d the relative absorption  
42 rapidly decreases.

43  
44 After absorption thiamin is transported to the liver. In the cells thiamine is converted to  
45 its biologically active form, TPP (1). The main proportion of the total body pool of  
46 about 30 mg in an adult is found in the muscles and liver (Butterworth 2006, Ariaey  
47 Nejad et al 1970). The metabolism of thiamin in the body is relatively fast, and the half  
48 life of <sup>14</sup>C labelled thiamin is estimated to be 9-18 days (Ariaey Nejad et al 1970).

49  
50 Thiamin deficiency causes beri-beri. In adults, symptoms include disturbances in the  
51 peripheral nervous system and heart function. Early deficiency symptoms may include  
52 anorexia, weight loss, mental changes and muscle weakness. In alcoholics, conditions  
53 such as Wernicke's encephalopathy and Korsakoff's psychosis occur, which are strong-  
54 ly related to insufficient thiamin intake and/or malabsorption (Sriram et al. 2012).  
55 Among children symptoms appear more instantly and are generally more severe, e.g.  
56 heart failure.

57  
58 Commonly used indicators of thiamin status include the activity of the enzyme trans-  
59 ketolase in the erythrocytes (ETK<sub>AC</sub>). In NNR recommendations for thiamin consider  
60 urinary excretion relative to ETK<sub>AC</sub> and thiamin intakes. The activity coefficient  
61 represents the degree of enzyme activity stimulation in vitro. The activity of this  
62 enzyme depends not only on TPP availability but also on glucose phosphate  
63 availability. An activity coefficient below 1.15 is regarded as an indicator of sufficient  
64 status, while a coefficient of 1.15-1.25 indicates marginal status (Finglas 1993). The  
65 concentration of free thiamin and its phosphate esters in blood or erythrocytes has been  
66 shown to be a good indicator of thiamin status (Talwar et al 2000), especially among  
67 subjects at risk for thiamin deficiency (Talwar et al 2000, Tallaksen et al 1992). The  
68 usefulness of the activity coefficient as an indicator of thiamin status in population  
69 surveys has been questioned, mainly due to its low correlation with e.g. erythrocyte  
70 thiamin (Baily et al 1994).

### 71 72 **Other health effects**

73 Several epidemiological studies have investigated the relationship between intake of  
74 thiamin, and other B-vitamins (folate, riboflavin, vitamin B6 and B12), and various  
75 cancers, mainly colorectal and breast cancer. However, no clear evidence for a relation  
76 between thiamin intake and different cancer forms have been found (e.g. Kabat et al  
77 2008, Key et al 2012, Pelucchi et al 2009). Thiamin has also been related to neuro-  
78 degenerative disorders in elderly such as Alzheimer's disease (Lu'o'ng 2011), but  
79 evidence for a role in preventing neurological disorders is lacking or limited (Balk et  
80 al. 2006).

81

### 82 **Requirement and recommended intake**

83 The requirement of thiamin has been related to the energy and carbohydrate intake  
84 (WHO/FAO 2004, DACH 2000). A clear relationship was shown by Sauberlich and  
85 co-workers (1979). The current US dietary reference values are, however, based on  
86 absolute intakes (Food and Nutrition Board 1998). Generally, thiamin intakes are  
87 related to energy and protein intakes at normal intake ranges of populations such as  
88 those of the Nordic countries.

89

90

91 Clinical signs of deficiency have been observed at intakes below 0.5 mg/d, correspond-  
92 ing to 0.05 mg/MJ (0.2 mg/1000 kcal) (WHO/FAO 2004, Food and Nutrition Board  
93 1998). In other studies thiamin excretion in urine and erythrocyte transketolase activity  
94 coefficients were normalised at intakes of 0.07 0.08 mg/MJ (0.30 0.33 mg/1000 kcal).  
95

96 In the absence of new data, the reference intakes set in NNR 2004 are kept unchanged.  
97 The average requirement for adults and children is thus set at 0.10 mg/MJ and the re-  
98 commended intake at 0.12 mg/MJ. However, when planning diets with energy levels  
99 below 8 MJ/d, the thiamin content should be at least 0.8 mg/d. The recommended  
100 intake for infants 0-12 months is set to 0.10 mg/MJ. The lower limit of intake is esti-  
101 mated at 0.05 mg/MJ.  
102

103 Studies on pregnant and lactating women indicate a higher requirement as assessed  
104 with biochemical parameters (Food and Nutrition Board 1998). An additional intake of  
105 0.4 mg/d during pregnancy and 0.5 mg/d during lactation is recommended.  
106

107 A few studies indicate that thiamin utilisation is impaired among elderly subjects (e.g.  
108 Nichols and Basu 1994). Therefore, when planning diets with energy levels below 8  
109 MJ/d, the thiamin content should be at least 1.0 mg/d.  
110

### 111 Reasoning behind the recommendation

112 The reference intakes set in NNR 2004 are kept unchanged. Few studies have explored  
113 relationships between thiamin intake and function. A few studies have examined  
114 effects of supplements on various clinical or biochemical outcomes but they do not  
115 make a useful contribution to understanding requirements in healthy population (e.g.  
116 Page et al 2011). Thus, there is no strong evidence to support a need for revision of the  
117 recommendation.  
118

### 119 Upper intake levels and toxicity

120 The EU Scientific Committee for Food (2001) concluded that it was not possible to set  
121 a safe upper intake level for thiamine, due to lack of data. Habitual thiamin intakes up  
122 to 6-7 mg/d have not been associated with negative effects. Oral intakes up to 500  
123 mg/d for periods up to one month have not been associated with toxic effects (SCF  
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125

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