6 Vitamins

6.1 Foreword

Vitamins are minor but essential constituents of food. They are required for the normal growth, maintenance and functioning of the human body. Hence, their preservation during storage and processing of food is of far-reaching importance. Data are provided in Tables 6.1 and 6.2 to illustrate vitamin losses in some preservation methods for fruits and vegetables. Vitamin losses can occur through chemical reactions which lead to inactive products, or by extraction or leaching, as in the case of water-soluble vitamins during blanching and cooking.

The vitamin requirement of the body is usually adequately supplied by a balanced diet. A defi-

Table 6.1. Vitamin losses (%) through processing/canning of vegetables

Processed/ canned product	Samples of vegetable analyzed	Vitamin losses as % of freshly cooked and drained product				
		A	B_1	B_2	Niacin	C
Frozen products (cooked and drained)	10[a]	12[c]	20	24	24	26
		0–50[d]	0–61	0–45	0–56	0–78
Sterilized products (drained)	7[b]	10	67	42	49	51
		0–32	56–83	14–50	31–65	28–67

[a] Asparagus, lima beans, green beans, broccoli, cauliflower, green peas, potatoes, spinach, brussels sprouts, and baby corn-cobs.
[b] As under a) with the exception of broccoli, cauliflower and brussels sprouts; the values for potato include the cooking water.
[c] Average values.
[d] Variation range.

Table 6.2. Vitamin loss (%) through processing/canning of fruits

Processed/canned product	Fruit samples analyzed	Vitamin losses as % of fresh product				
		A	B_1	B_2	Niacin	C
Frozen products (not thawed)	8[a]	37[c]	29	17	16	18
		0–78[d]	0–66	0–67	0–33	0–50
Sterilized products (including the cooking water)	8[b]	39	47	57	42	56
		0–68	22–67	33–83	25–60	11–86

[a] Apples, apricots, bilberries, sour cherries, orange juice concentrate (calculated for diluted juice samples), peaches, raspberries and strawberries.
[b] As under a) except orange juice and not its concentrate was analyzed.
[c] Average values.
[d] Variation range.

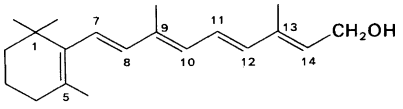
ciency can result in hypovitaminosis and, if more severe, in avitaminosis. Both can occur not only as a consequence of insufficient supply of vitamins by food intake, but can be caused by disturbances in resorption, by stress and by disease.
An assessment of the extent of vitamin supply can be made by determining the vitamin content in blood plasma, or by measuring a biological activity which is dependent on the presence of a vitamin, as are many enzyme activities.
Vitamins are usually divided into two general classes: the fat-soluble vitamins, such as A, D, E and K_1, and the water-soluble vitamins, B_1, B_2, B_6, nicotinamide, pantothenic acid, biotin, folic acid, B_{12} and C.
Data on the desirable human daily intake of some vitamins are presented by age group in Table 6.3.

6.2 Fat-Soluble Vitamins

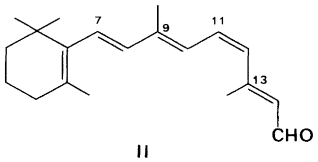
6.2.1 Retinol (Vitamin A)

6.2.1.1 Biological Role

Retinol (I, in Formula 6.1) is of importance in protein metabolism of cells which develop from the ectoderm (such as skin or mucouscoated linings of the respiratory or digestive systems). Lack of retinol in some way negatively affects epithelial tissue (thickening of skin, hyperkeratosis) and also causes night blindness.



I



II (6.1)

Furthermore, retinol, in the form of 11-cis-retinal (II), is the chromophore component of the visual cycle chromoproteins in three types of cone cells, blue, green and red (λ_{max} 435, 540 and 565 nm, respectively) and of rods of the retina.

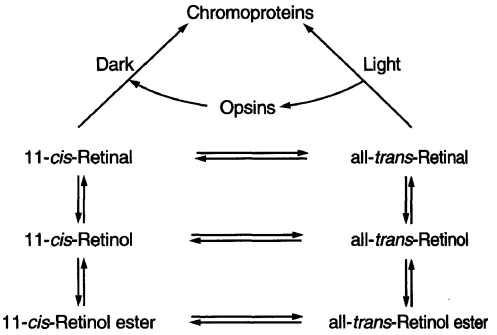


Fig. 6.1. Schematic representation of the visual cyle

The chromoproteins (rhodopsins) are formed in the dark from the corresponding proteins (opsins) and 11-cis-retinal, while in the light the chromoproteins dissociate into the more stable all-trans-retinal and protein. This conformational change triggers a nerve impulse in the adjacent nerve cell. The all-trans-retinal is then converted to all-trans-retinol and through an intermediary, 11-cis-retinol, is transformed back into 11-cis-retinal (see Fig. 6.1 for the visual cycle reactions).

6.2.1.2 Requirement, Occurrence

The daily requirement of vitamin A (Table 6.3) is provided to an extent of 75% by retinol intake (as fatty acid esters, primarily retinyl palmitate), while the remaining 25% is through β-carotene and other provitaminactive carotenoids. Due to the limited extent of carotenoid cleavage, at least 6 g of β-carotene are required to yield 1 g retinol. Vitamin A absorption and its storage in the liver occur essentially in the form of fatty acid esters. Its content in liver is 250 µg/g fresh tissue, i. e. a total of about 240–540 mg is stored. The liver supplies the blood with free retinol, which then binds to proteins in blood. The plasma concentration of retinol averages 1.78 µmol/l in women and 2.04 µmol/l in men.
A hypervitaminosis is known, but the symptoms disappear if the intake of retinal is decreased.
Vitamin A occurs only in animal tissue; above all in fish liver oil, in livers of mammals, in milk fat and in egg yolk. Plants are devoid of vitamin A but do contain carotenoids which yield vitamin A

Table 6.3. Recommended daily intake of vitamins

Age group (years)	A (mg Retinol[a])	D (μg)[b]	E (mg)[c]	K (μg)[d]	C (mg)	B_1 (mg)	B_2 (mg)	Niacin[e] (mg)	B_6 (μg)	Folic acid[f] (mg)	Pantothenic acid (mg)	Biotin (μg)	B_{12} (μg)
<1	0.5–0.6	10	3–4	4–10	50–55	0.2–0.4	0.3–0.4	2–5	0.1–0.3	60–80	2–3	5–10	0.4–0.8
1–4	0.6	5	6	15	60	0.6	0.7	7	0.4	200	4	10–15	1.0
4–10	0.7–0.8	5	8–10	20–30	70–80	0.8–1.0	0.9–1.1	10–12	0.5–0.7	300	4–5	15–20	1.5–1.8
10–15	0.9–1.1	5	10–14	40–50	90–100	1.0–1.3	1.2–1.6	13–18	1.0–1.4	400	5–6	20–35	2.0–3.0
15–25	0.9–1.1	5	15	60–70	100	1.0–1.3	1.2–1.5	13–17	1.2–1.6	400	6	30–60	3.0
25–51	0.8–1.0	5	14	60–70	100	1.0–1.2	1.2–1.4	13–16	1.2–1.5	400	6	30–60	3.0
52–65	0.8–1.0	5	13	80	100	1.0–1.1	1.2–1.3	13–15	1.2–1.5	400	6	30–60	3.0
>65	0.8–1.0	10	12	80	100	1.0	1.2	13	1.2–1.4	400	6	30–60	3.0
Pregnant women	1.1	5	13	60	100	1.2	1.5	15	1.9	600	6	30–60	3.5
Lactating women	1.5	5	17	60	150	1.4	1.6	17	1.9	600	6	30–60	4.0

[a] 1 mg retinol = 1 mg retinol equivalent = 6 mg all-trans-β-carotene = 12 mg other provitamin A carotinoids = 1.15 mg all-trans-retinyl acctate = 1.83 mg all-trans-retinyl palmitate (IU = 0.34 μg retinol).
[b] Ergocalciferol (D_2) or cholecalciferol (D_3) (1 IU = 0.025 μg).
[c] Tocopherol equivalent (cf. 6.2.3.1).
[d] Phylloquinone (cf. 6.2.4).
[e] 1 mg niacin equivalent = 60 mg tryptophan.
[f] 1 μg folate equivalent = 1 μg food folate = 0.5 μg folic acid (PGA, cf. 6.3.7.1).

by cleavage of the centrally located double bond (provitamins A). Carotenoids are present in almost all vegetables but primarily in green, yellow and leafy vegetables (carrots, spinach, cress, kale, bell peppers, paprika peppers, tomatoes) and in fruit, outstanding sources being rose hips, pumpkin, apricots, oranges and palm oil, which is often used for yellow coloring. Animal carotenoids are always of plant origin, derived from feed.
Table 6.7 gives the vitamin A content of some common foods. These values can vary greatly with cultivar, stage of ripeness, etc. An accurate estimate of the vitamin A content of a food must include a detailed analysis of its carotenoids.

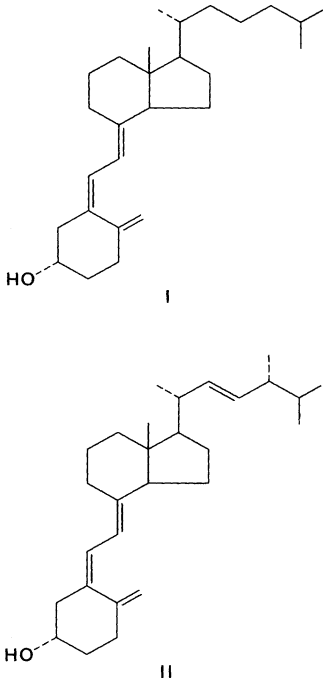
6.2.1.3 Stability, Degradation

Food processing and storage can lead to 5–40% destruction of vitamin A and carotenoids. In the absence of oxygen and at higher temperatures, as experienced in cooking or food sterilization, the main reactions are isomerization and fragmentation. In the presence of oxygen, oxidative degradation leads to a series of products, some of which are volatile (cf. 3.8.4.4). This oxidation often parallels lipid oxidation (cooxidation process). The rate of oxidation is influenced by oxygen partial pressure, water activity, temperature, etc. Dehydrated foods are particularly sensitive to oxidative degradation.

6.2.2 Calciferol (Vitamin D)

6.2.2.1 Biological Role

Cholecalciferol (vitamin D_3, I) is formed from cholesterol in the skin through photolysis of 7-dehydrocholesterol (provitamin D_3) by ultraviolet light ("sunshine vitamin"; cf. 3.8.2.2.2). Similarly, vitamin D_2 (ergocalciferol, II; cf. Formula 6.2) is formed from ergosterol.
Vitamin D_2 and D_3 are hydroxylated first in the liver to the prohormone 25-hydroxycholecalciferol (calcidiol) and subsequently in the kidney to the vitamin D hormone 1α,25-dihydroxycholecalciferol (calcitriol). Calcitriol acts as an inductor of proteins in various organs. It promotes calcium resorption in the intestine and an optimal calcium concentration in the kidney and in the bones, it induces the synthesis of proteins involved in the structure of the bone matrix and in calcification.
Vitamin D deficiency can result in an increased excretion of calcium and phosphate and, consequently, impairs bone formation through inadequate calcification of cartilage and bones (childhood rickets). Vitamin D deficiency in adults leads to osteomalacia, a softening and weakening of bones. Hypercalcemia is a result of excessive intake of vitamin D (>50 µg/day), causing calcium carbonate and calcium phosphate deposition disorders involving various organs.



(6.2)

6.2.2.2 Requirement, Occurrence

The daily requirement is shown in Table 6.3. Indicators of deficiency are the concentration of the metabolite 25-hydroxycholecalciferol in plasma and the activity of alkaline serum phosphatase, which increases during vitamin deficiency.
Most natural foods have a low content of vitamin D_3. Fish liver oil is an exceptional source

of vitamin D_2. The D-provitamins, ergosterol and 7-dehydrocholesterol, are widely distributed in the animal and plant kingdoms. Yeast, some mushrooms, cabbage, spinach and wheat germ oil are particularly abundant in provitamin D_2. Vitamin D_3 and its provitamin are present in egg yolk, butter, cow's milk, beef and pork liver, mollusks, animal fat and pork skin. However, the most important vitamin D source is fish oil, primarily liver oil. The vitamin D requirement of humans is best supplied by 7-dehydrocholesterol. Table 6.7 gives data on vitamin D occurrence in some foods. However, these values can vary widely, as shown by variations in dairy cattle milk (summer or winter), caused by feed or frequency of pasture grazing and exposure to the ultraviolet rays of sunlight.

6.2.2.3 Stability, Degradation

Vitamin D is sensitive to oxygen and light. Its stability in food is not a problem, because adults usually obtain a sufficient supply of this vitamin.

6.2.3 α-Tocopherol (Vitamin E)

6.2.3.1 Biological Role

The various tocopherols differ in the number and position of the methyl groups on the ring. α-Tocopherol (Formula 6.3; the configuration at the three asymmetric centers, 2, 4′ and 8′, is R) has the highest biological activity (Table 6.4). Its activity is based mainly on its antioxidative properties, which retard or prevent lipid oxidation (cf. 3.7.3.1). Thus, it not only contributes to the stabilization of membrane structures, but also stabilizes other active agents (e. g., vitamin A, ubiquinone, hormones, and enzymes) against oxidation. Vitamin E is involved in the conversion of arachidonic acid to prostaglandins and slows down the aggregation of blood platelets. Vitamin E deficiency is associated with chronic disordes (sterility in domestic and experimental animals, anemia in monkeys, and muscular dystrophy in chickens). Its mechanism of action is not fully elucidated.

Table 6.4. Biological activity of some tocopherols

Tocopherol (T)	Vitamin E activity	
	In IU/mg[a]	Conversion factor[b]
2R,4′R,8′R-α-T	1.49	1.00
2S,4′R,8′R-α-T	0.46	0.31
2R,4′R,8′S-α-T	1.34	0.90
2S,4′R,8′S-α-T	0.55	0.37
2R,4′S,8′S-α-T	1.09	0.73
2S,4′S,8′R-α-T	0.31	0.21
2R,4′S,8′R-α-T	0.85	0.57
2S,4′S,8′S-α-T	1.10	0.60
2R,4′R,8′R-α-Tocopheryl acetate	1.36	0.91
all-rac-α-T	1.10	0.74
all-rac-α-Tocopheryl acetate	1.00	0.67
all-rac-β-T	0.30	0.20
all-rac-γ-T	0.15	0.10
all-rac-δ-T	0.01	

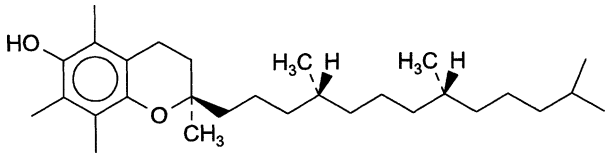
[a] International units (IU) per mg substance.
[b] Conversion factor from mg substance to mg α-tocopherol equivalents.

Table 6.5. Requirement of tocopherol equivalents (TE) on supply of unsaturated fatty acids

Fatty acid	TE (mg/g fatty acid)
Monoene acids	0.06
Diene acids	0.4
Triene acids	0.8
Tetraene acids	1.0
Pentaene acids	1.2
Hexaene acids	1.45

6.2.3.2 Requirement, Occurrence

The daily requirement is given in Table 6.3. It increases when the diet contains a high content of unsaturated fatty acids (cf. Table 6.5). A normal supply results in a tocopherol concentration of 12–46 μmol/l in blood plasma.
A level less than 0.4 mg/100 ml is considered a deficiency.
Section 3.8.3.1 and Table 6.7 provide data on the tocopherol content in some foods. The main sources are vegetable oils, particularly germ oils of cereals.



(6.3)

6.2.3.3 Stability, Degradation

Losses occur in vegetable oil processing into margarine and shortening. Losses are also encountered in intensive lipid autoxidation, particularly in dehydrated or deep fried foods (Table 6.6).

Table 6.6. Tocopherol stability during deep frying

	Tocopherol total (mg/100 g)	Loss (%)
Oil before deep frying	82	
after deep frying	73	11
Oil extracted from potato chips		
immediately after production	75	
after 2 weeks storage at room temperature	39	48
after 1 month storage at room temperature	22	71
after 2 months storage at room temperature	17	77
after 1 month kept at −12 °C	28	63
after 2 months kept at −12 °C	24	68
Oil extracted from French fries		
immediately after production	78	
after 1 month kept at −12 °C	25	68
after 2 months kept at −12 °C	20	74

6.2.4 Phytomenadione (Vitamin K_1 Phylloquinone)

6.2.4.1 Biological Role

The K-group vitamins are naphthoquinone derivatives which differ in their side chains. The structure of vitamin K_1 is shown in Formula 6.4. The configuration at carbon atoms 7′ and 11′ is R and corresponds to that of natural phytol. Racemic vitamin K_1 synthesized from optically inactive isophytol has the same biological activity as the natural product. Vitamin K is involved in the post-translational synthesis of γ-carboxyglutamic acid (Gla) in vitamin K-dependent proteins. It is reduced to the hydroquinone form (Formula 6.4) which acts as a cofactor in the carboxylation of glutamic acid. In this process, it is converted to the epoxide from which vitamin K is regenerated. Blood clotting factors (prothrombin, proconvertin, Christmas and Stuart factor) as well as proteins which perform other functions belong to the group of vitamin K-dependent proteins which bind Ca^{2+} ions at Gla. Deficiency of this vitamin causes reduced prothrombin activity, hypothrombinemia and hemorrhage.

6.2.4.2 Requirement, Occurrence

The activity is given in vitamin equivalents (VE): 1 VE = 1 µg phylloquinone. The daily requirement of vitamin K_1 is shown in Table 6.3. It is covered by food (cf. Table 6.7). The bacteria present in the large intestine form relatively high amounts of K_2. However, it is uncertain whether they appreciably contribute to covering the requirement.

Vitamin K_1 occurs primarily in green leafy vegetables (spinach, cabbage, cauliflower), but liver (veal or pork) is also an excellent source (Table 6.7).

6.2.4.3 Stability, Degradation

Little is known about the reactions of vitamin K_1 in foods. The vitamin K compounds are destroyed by light and alkali. They are relatively stable to atmospheric oxygen and exposure to heat.

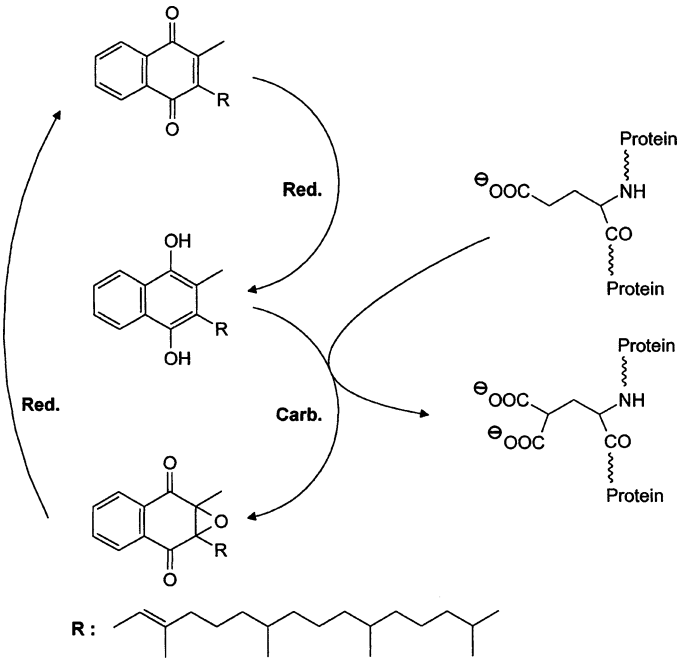
Table 6.7. Vitamin content of some food products[a]

Food product	Carotene[b] mg	A mg	D µg	E mg	K mg	B_1 mg	B_2 mg	NAM[c] mg	PAN[d] mg	B_6 mg	BIO[e] µg	FOL[f] µg	B_{12} µg	C mg
Milk and milk products														
Bovine milk	0.018	0.028	0.088	0.07	0.0003	0.04	0.18	0.09	0.35	0.04	3.5	8.0	0.4	1.7
Human milk	0.003	0.054	0.07	0.28	0.0005	0.02	0.04	0.17	0.21	0.01	0.6	8.0	0.05	6.5
Butter	0.38	0.59	1.2	2.2	0.007	0.005	0.02	0.03	0.05	0.005				0.2
Cheese														
Emmental	0.12	0.27	1.1	0.53	0.003	0.05	0.34	0.18	0.40	0.11	3.0	9.0	3.0	0.5
Camembert (60% fat)	0.29	0.50		0.77		0.04	0.37	0.95	0.7	0.2	2.8	38	2.4	
Camembert (30% fat)	0.1	0.2	0.17	0.30		0.05	0.67	1.2	0.9	0.3	5.0	66	3.1	
Eggs														
Chicken egg yolk	0.29	0.88	5.6	5.7		0.29	0.40	0.07	3.7	0.3	53	208	2.0	0.3
Chicken egg white						0.02	0.32	0.09	0.14	0.012	7	9.2	0.1	0.3
Meat and meat products														
Beef, muscle			0.02		0.48	0.013	0.08	0.26	7.5	0.31	0.24	3.0	3.0	5.0
Pork, muscle		0.006		0.41	0.018	0.90	0.23	5.0	0.70	0.4			0.8	
Calf liver		28.0	0.33	0.24	0.09	0.28	2.61	15.0	7.9	0.17	80	240	60	35
Pork liver		36		0.60	0.06	0.31	3.2	15.7	6.8	0.6	30	220	40	23
Chicken liver		33	1.3	0.5	0.08	0.32	2.49	11.6	7.2	0.8		380	20	28
Pork kidneys		0.06		0.45		0.34	1.8	8.4	3.1	0.6			20	16
Blood sausage	0.02					0.07	0.13	1.2					50	
Fish and fish products														
Herring		0.04	27	1.5		0.04	0.22	3.8	0.9	0.5	4.5	5	8.5	0.5
Eel		0.98	20	8		0.18	0.32	2.6	0.3			13	1	1.8
Cod-liver oil				3.26										
Cereals and cereal products														
Wheat, whole kernel	0.02			1.4		0.48	0.09	5.1	1.2	0.27	6	58		
Wheat flour, type 550				0.34		0.11	0.03	0.5	0.4	0.10	1.1	16		
Wheat flour, type 1050						0.43	0.07	1.4	0.63	0.24	1.1	30		
Wheat germ	0.06			27.6	0.13	2.01	0.72	4.5	1.0	0.5	17	520		
Rye whole kernel				2.0		0.35	0.17	1.8	1.5	0.23	5.0	35		
Rye flour, type 997						0.19	0.11	0.8				33		
Corn whole kernel	1.3			2.0	0.04	0.36	0.20	1.5	0.7	0.4	6	31		

Table 6.7. Continued

Food product	Carotene[b] mg	A mg	D µg	E mg	K mg	B_1 mg	B_2 mg	NAM[c] mg	PAN[d] mg	B_6 mg	BIO[e] µg	FOL[f] µg	B_{12} µg	C mg
Corn (breakfast cereal, corn flakes)	0.17			0.18		0.60		1.4	0.2	0.07		6		
Oat flakes				1.5	0.063	0.59	0.15	1.0	1.1	0.16	20	67		
Rice, unpolished				0.74		0.41	0.09	5.2	1.7	0.28	12	16		
Rice, polished				0.18		0.06	0.03	1.3	0.6	0.15	3.0	11		
Vegetables														
Watercress	4.9					0.09	0.17	0.7						96
Mushrooms (cultivated)	0.01		1.94	0.12	0.02	0.10	0.44	5.2	2.1	0.07	16	25		4.9
Chicory	3.4					0.06	0.03	0.24		0.05	4.8	50		8.7
Endive	1.7					0.05	0.12	0.4				109		9.4
Lamb's lettuce	3.9			0.6		0.07	0.08	0.4		0.25		145		35
Kale	5.2			1.7	0.82	0.1	0.25	2.1		0.3	0.5	187		105
Potatoes	0.005			0.05	0.002	0.11	0.05	1.2	0.4	0.31	0.4	15		17
Kohlrabi	0.2					0.05	0.05	1.8	0.1	0.1	2.7	70		63
Head lettuce	1.1			0.6	0.2	0.06	0.08	0.3	0.1	0.06	1.9	41		13
Lentils, dried	0.1					0.48	0.26	2.5	1.4	0.6		168		7.0
Carrots	12			0.47	0.015	0.07	0.05	0.6	0.3	0.27	5	17		7.1
Brussels sprouts	0.4			0.6	0.24	0.13	0.14	0.7		0.3	0.4	101		114
Spinach	4.8			2.5	0.4	0.09	0.20	0.6	0.3	0.22	6.9	145		52
Edible mushroom (*Boletus edulis*)			3.1	0.63		0.03	0.37	4.9	2.7					2.5
Tomatoes	0.59			0.81	0.006	0.06	0.04	0.5	0.3	0.1	4	33		19
White cabbage	0.07			1.7	0.07	0.05	0.04	0.3	0.3	0.19	3.1	31		48
Fruits														
Apple	0.05			0.49	0.004	0.035	0.032	0.3	0.1	0.1	0.0045	5		12
Orange	0.1			0.32		0.08	0.04	0.3	0.2	0.1	2.3	22		50
Apricot	1.8			0.5		0.04	0.05	0.8	0.3	0.1		4		9.4
Strawberry	0.02			0.12	0.02	0.03	0.05	0.5	0.3	0.06	4	43		64
Grapefruit	0.01			0.30		0.05	0.02	0.24	0.25	0.03	0.4	10		44
Rose hips	4.8			4.2		0.09	0.06	0.48		0.05				1250
Red currants	0.03			0.71		0.04	0.03	0.23	0.06	0.05	2.6	11		36
Black currants	0.08			1.9		0.05	0.04	0.28	0.4	0.08	2.4	8.8		177
Sour cherries	0.24			0.13		0.05	0.06	0.4				29		12
Plums	0.41			0.86		0.07	0.04	0.4	0.2	0.05	0.1	2		5.4
Sea buckthorn	1.5			3.2		0.03	0.21	0.3	0.2	0.11	3.3	10		450
Yeast														
Baker's yeast, pressed						1.43	2.31	17.4	3.5	0.68	33	293		
Brewer's yeast, dried						12.0	3.8	44.8	7.2	4.4	20			

[a] Values are given in mg or µg per 100 g of edible portion. [b] Total carotenoids with vitamin A activity. [c] Nicotinamide.
[d] Pantothenic acid. [e] Biotin. [f] Folic acid.



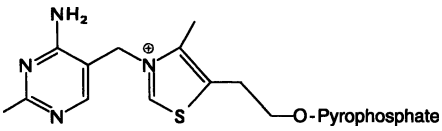
(6.4)

In the hydrogenation of oils, the double bond in residue R (cf. Formula 6.4) is attacked. Although hydrogenated vitamin K (2′,3′-dihydrophylloquinone) is absorbed, it is apparently no longer as active as the natural form.

6.3 Water-Soluble Vitamins

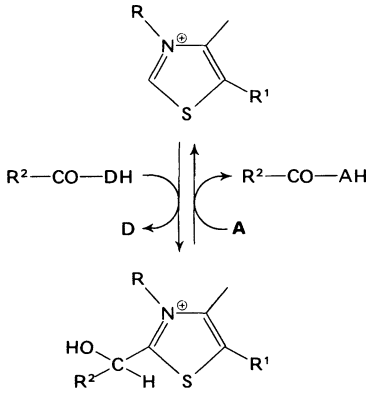
6.3.1 Thiamine (Vitamin B_1)

6.3.1.1 Biological Role



(6.5)

Thiamine, in the form of its pyrophosphate, such as pyruvate dehydrogenase, transketolase, phosphoketolase and α-ketoglutarate dehydrogenase, in reactions involving the transfer of an activated aldehyde unit (D: donor; A: acceptor):



(6.6)

Vitamin B_1 deficiency is shown by a decrease in activity of the enzymes mentioned above. The disease known as beri-beri, which has neurological and cardiac symptoms, results from a severe dietary deficiency of thiamine.

6.3.1.2 Requirement, Occurrence

The daily requirement is shown in Table 6.3. Since thiamine is a key substance in carbohy-

drate metabolism, the requirement increases in a carbohydrate-enriched diet. The assay of transketolase activity in red blood cells or the extent of transketolase reactivation on addition of thiamine pyrophosphate can be used as indicators for sufficient vitamin intake in the diet.

Vitamin B_1 is found in many plants. It is present in the pericarp and germ of cereals, in yeast, vegetables (potatoes) and shelled fruit. It is abundant in pork, beef, fish, eggs and in animal organs such as liver, kidney, brain and heart. Human milk and cow's milk contain vitamin B_1. Whole grain bread and potatoes are important dietary sources. Since vitamin B_1 is localized in the outer part of cereal grain hulls, flour milling with a low extraction grade or rice polishing remove most of the vitamin in the bran (cf. 15.3.1.3 and 15.3.2.2.1). Table 6.7 lists data on the occurrence of thiamine.

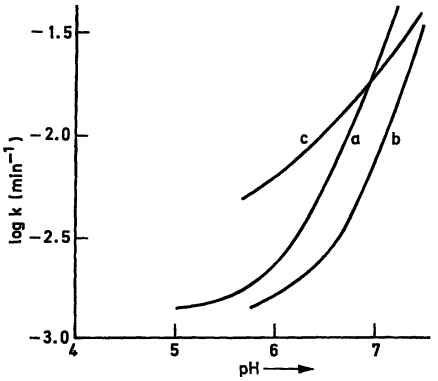


Fig. 6.2. Inactivation rate of thiamine as affected by pH **a** Thiamine in phosphate buffer, **b** thiamine in wheat or oat flour, **c** thiamine pyrophosphate in flour

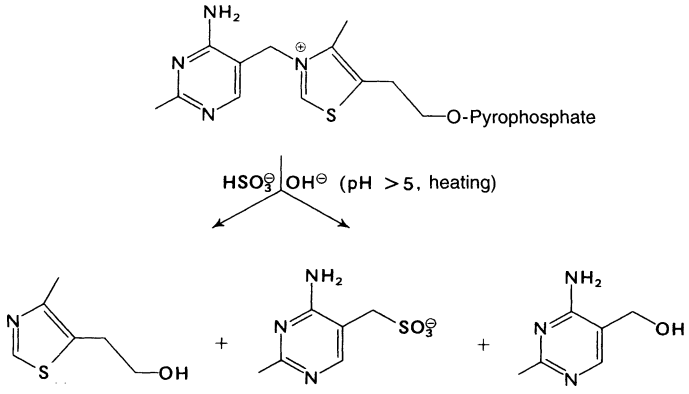
Table 6.8. Thiamine losses in food during storage (12 months)

Food	Thiamine loss, %	
	1.5 °C	38 °C
Apricots	28	65
Orange juice	0	22
Peas	0	32
Green beans	24	92
Tomato juice	0	40

6.3.1.3 Stability, Degradation

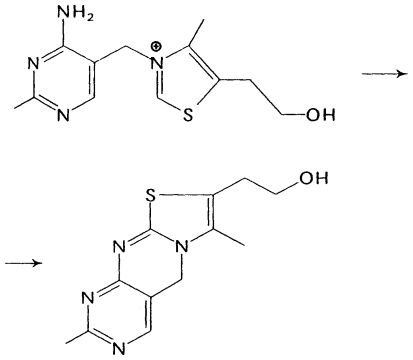
Thiamine stability in aqueous solution is relatively low. It is influenced by pH (Fig. 6.2), temperature (Table 6.8), ionic strength and metal ions. The enzyme-bound form is less stable than free thiamine (Fig. 6.2). Strong nucleophilic reagents, such as HSO_3^- or OH^-, cause rapid decomposition by forming 5-(2-hydroxyethyl)-4-methylthiazole and 2-methyl-4-amino-5(methylsulfonic acid)-pyrimidine, or 2-methyl-4-amino-5-hydroxymethylpyrimidine (see Reactions 6.7). Thermal degradation of thiamine, which also initially yields the thiazole and pyrimidine derivatives mentioned above, is involved in the formation of meat-like aroma in cooked food (cf. 5.3.1.4).

Thiamine is inactivated by nitrites, probably through reaction with the amino group attached to the pyrimidine ring.



(6.7)

Strong oxidants, such as H_2O_2 or potassium ferricyanide, yield the fluorescent thiochrome. This reaction is often used in chemical determination of the thiamine content in food (see Reaction 6.8).



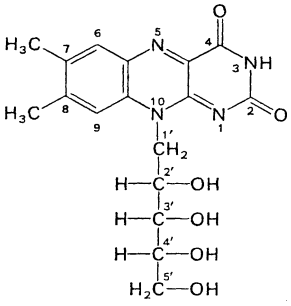
(6.8)

The following losses of thiamine can be expected: 15–25% in canned fruit or vegetables stored for more than a year; 0–60% in meat cooked under household conditions, depending on temperature and preparation method; 20% in salt brine pickling of meat and in baking of white bread; 15% in blanching of cabbage without sulfite and 40% with sulfite. Losses caused by sulfite are pH dependent. Practically no thiamine degradation occurs in a stronger acidic medium (e. g. lemon juice).

6.3.2 Riboflavin (Vitamin B_2)

6.3.2.1 Biological Role

Riboflavin (Formula 6.9) is the prosthetic group of flavine enzymes, which are of great importance in general metabolism and particularly in metabolism of protein.



(6.9)

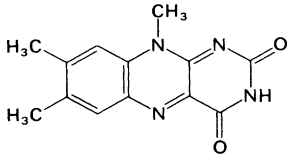
Riboflavin deficiency will lead to accumulation of amino acids. A specific deficiency symptom is the decrease of glutathione reductase activity in red blood cells.

6.3.2.2 Requirement, Occurrence

The daily requirement is given in Table 6.3. Deficiency symptoms are rarely observed with a normal diet and, since the riboflavin pool in the body is very stable, even in a deficient diet it is not depleted by more than 30–50%. The riboflavin content of urine is an indicator of riboflavin supply levels. Values above 80 µg riboflavin/g creatinine are normal; 27–79 µg/g are low; and less than 27 µg/g strongly suggests a vitamin-deficient diet. Glutathione reductase activity assay can provide similar information.
The most important sources of riboflavin are milk and milk products, eggs, various vegetables, yeast, meat products, particularly variety meats such as heart, liver and kidney, and fish liver and roe. Table 6.7 provides data about the occurrence of riboflavin in some common foods.

6.3.2.3 Stability, Degradation

Riboflavin is relatively stable in normal food handling processes. Losses range from 10–15%. Exposure to light, especially in the visible spectrum from 420–560 nm, photolytically cleaves ribitol from the vitamin, converting it to lumiflavin:



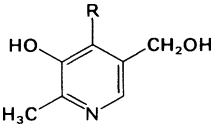
(6.10)

6.3.3 Pyridoxine (Pyridoxal, Vitamin B_6)

6.3.3.1 Biological Role

Vitamin B_6 activity is exhibited by pyridoxine (Formula 6.11) or pyridoxol (R = CH_2OH), pyridoxal (R = CHO) and pyridoxamine (R = CH_2NH_2). The metabolically active form,

pyridoxal phosphate, functions as a coenzyme (cf. 2.3.2.3) of amino acid decarboxylases, amino acid racemases, amino acid dehydrases, amino transferases, serine palmitoyl transferase, lysyl oxidase, δ-aminolevulinic acid synthase, and of enzymes of tryptophan metabolism. Furthermore, it stabilizes the conformation of phosphorylases.



(6.11)

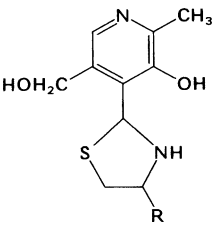
The intake of the vitamin occurs usually in the forms of pyridoxal or pyridoxamine.
Pyridoxine deficiency in the diet causes disorders in protein metabolism, e. g., in hemoglobin synthesis. Hydroxykynurenine and xanthurenic acid accumulate, since the conversion of tryptophan to nicotinic acid, a step regulated by the kynureninase enzyme, is interrupted.

6.3.3.2 Requirement, Occurrence

The daily requirement is given in Table 6.3. An indicator of sufficient supply is the activity of glutamate oxalacetate transaminase, an enzyme present in red blood cells. This activity is decreased in vitamin deficiency. The occurrence of pyridoxine in food is outlined in Table 6.8.

6.3.3.3 Stability, Degradation

The most stable form of the vitamin is pyridoxal, and this form is used for vitamin fortification of food. Vitamin B_6 loss is 45% in cooking of meat and 20–30% in cooking of vegetables. During milk sterilization, a reaction with cysteine transforms the vitamin into an inactive thiazolidine derivative (Formula 6.12). This reaction may account for vitamin losses also in other heat-treated foods.

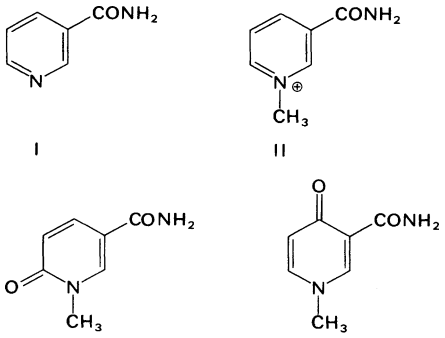


(6.12)

6.3.4 Nicotinamide (Niacin)

6.3.4.1 Biological Role

Nicotinic acid amide (I), in the form of nicotinamide adenine dinucleotide (NAD^+, cf. 2.3.1.1), or its phosphorylated form ($NADP^+$), is a coenzyme of dehydrogenases. Its excretion in urine is essentially in the form of N^1-methylnicotinamide (trigonelline amide, II), N^1-methyl-6-pyridone-3-carboxamide (III) and N^1-methyl-4-pyridone-3-carboxamide (IV):



(6.13)

Vitamin deficiency is observed initially by a drop in concentration of NAD^+ and $NADP^+$ in liver and muscle, while levels remain normal in blood, heart and kidney. The classical deficiency disease is pellagra, which affects the skin, digestion and the nervous system (dermatitis, diarrhea and dementia). However, the initial deficiency symptoms are largely non-specific.

6.3.4.2 Requirement, Occurrence

The daily requirement (cf. Table 6.3) is covered to an extent of 60–70% by tryptophan intake. Hence, milk and eggs, though they contain little niacin, are good foods for prevention of pellagra because they contain tryptophan. It substitutes for niacin in the body, with 60 mg L-tryptophan equalling 1 mg nicotinamide. Indicators of sufficient supply of niacin in the diet are the levels of metabolites II (cf. Formula 6.13) in urine or III and IV in blood plasma.
The vitamin occurs in food as nicotinic acid, either as its amide or as a coenzyme. Animal organs, such as liver, and lean meat, cereals, yeast and mushrooms are abundant sources of niacin. Table 6.7 provides data on its occurrence in food.

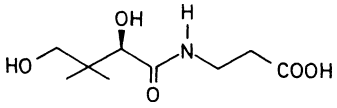
6.3.4.3 Stability, Degradation

Nicotinic acid is quite stable. Moderate losses of up to 15% are observed (cf. Tables 6.1 and 6.2) in blanching of vegetables. The loss is 25–30% in the first days of ripening of meat.

6.3.5 Pantothenic Acid

6.3.5.1 Biological Role

Pantothenic acid (Formula 6.14) is the building unit of coenzyme A (CoA), the main carrier of acetyl and other acyl groups in cell metabolism. Acyl groups are linked to CoA by a thioester bond. Pantothenic acid occurs in free form in blood plasma, while in organs it is present as CoA. The highest concentrations are in liver, adrenal glands, heart and kidney.



(6.14)

Only the R enantiomer occurs in nature and is biologically active. A normal diet provides an adequate supply of the vitamin.

6.3.5.2 Requirement, Occurrence

The daily requirement is 6–8 mg. The concentration in blood is 10–40 µg/100 ml and 2–7 mg/day are excreted in urine.
Pantothenic acid in food is determined with microbiological or ELISA (cf. 2.6.3) techniques. A gas chromatographic method using a ^{13}C-isotopomer of pantothenic acid as the internal standard is very accurate and much more sensitive. Table 6.7 lists data on pantothenic acid occurrence in food.

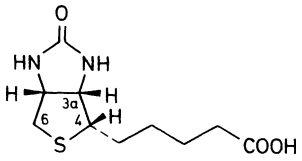
6.3.5.3 Stability, Degradation

Pantothenic acid is quite stable. Losses of 10% are experienced in processing of milk. Losses of 10–30%, mostly due to leaching, occur during cooking of vegetables.

6.3.6 Biotin

6.3.6.1 Biological Role

Biotin is the prosthetic group of carboxylating enzymes, such as acetyl-CoA-carboxylase, pyruvate carboxylase and propionyl-CoA-carboxylase, and therefore plays an important role in fatty acid biosynthesis and in gluconeogenesis. The carboxyl group of biotin forms an amide bond with the ε-amino group of a lysine residue of the particular enzyme protein. Only the (3aS, 4S, 6aR) compound, D-(+)-biotin, is biologically active:



(6.15)

Biotin deficiency rarely occurs. Consumption of large amounts of raw egg white might inactivate biotin by its specific binding to avidin (cf. 11.2.3.1.9).

6.3.6.2 Requirement, Occurrence

The daily requirement is shown in Table 6.3. An indicator of sufficient biotin supply is the excretion level in the urine, which is normally 30–50 µg/day. A deficiency is indicated by a drop to 5 µg/day.
Biotin is not free in food, but is bound to proteins. Table 6.7 provides data on its occurrence in food.

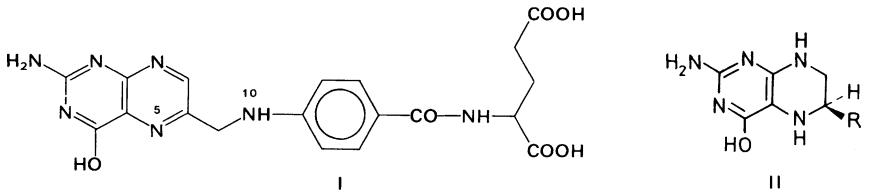
6.3.6.3 Stability, Degradation

Biotin is quite stable. Losses during processing and storage of food are 10–15%.

6.3.7 Folic Acid

6.3.7.1 Biological Role

The tetrahydrofolate derivative (Formula (6.16), II) of folic acid (I, pteroylmonoglutamic acid, PGA) is the cofactor of enzymes which trans-



(6.16)

fer single carbon units in various oxidative states, e. g., formyl or hydroxymethyl residues. In transfer reactions the single carbon unit is attached to the N^5- or N^{10}-atom of tetrahydrofolic acid.

Folic acid deficiency caused by insufficient supply in the diet or by malfunction of absorptive processes is detected by a decrease in folic acid concentration in red blood cells and plasma, and by a change in blood cell patterns. There are clear indications that a congenital defect (neural tube defect) and a number of diseases are based on a deficiency of folate.

6.3.7.2 Requirement, Occurrence

The requirement shown in Table 6.3 is not often reached. In some countries, cereal products are supplemented with folic acid in order to avoid deficiency, e. g., with 1.4 mg/kg in the USA. Correspondingly, positive effects on consumer health have been observed.

In cooperation with vitamin B_{12}, folic acid methylates homocysteine to methionine. Therefore, homocysteine is a suitable marker for the supply of folate. In the case of a deficiency, the serum concentration of this marker is clearly raised compared with the normal value of 8–10 µmol/ml, resulting in negative effects on health because higher concentrations of homocysteine are toxic.

In food folic acid is mainly bound to oligo-γ-L-glutamates made up of 2–6 glutamic acid residues. Unlike free folic acid, the absorption of this conjugated form is limited and occurs only after the glutamic acid residues are cleaved by folic acid conjugase, a γ-glutamyl-hydrolase, to give the monoglutamate compound. Since certain constituents can reduce the absorption of folates, the average bioavailability is estimated at 50%. The folic acid content of foods varies. Data on folic acid occurrence in food are compiled in Table 6.7.

6.3.7.3 Stability, Degradation

Folic acid is quite stable. There is no destruction during blanching of vegetables, while cooking of meat gives only small losses. Losses in milk are apparently due to an oxidative process and parallel those of ascorbic acid. Ascorbate added to food preserves folic acid.

6.3.8 Cyanocobalamin (Vitamin B_{12})

6.3.8.1 Biological Role

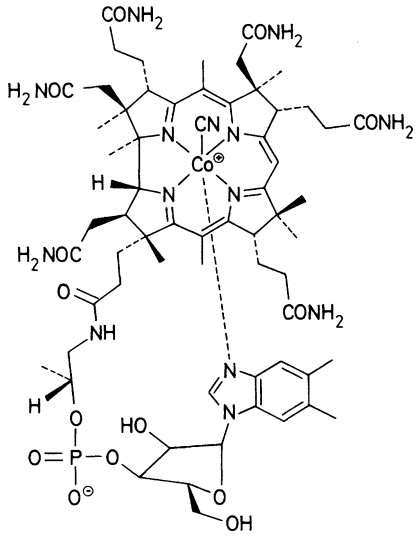
Cyanocobalamin (Formula 6.17) was isolated in 1948 from *Lactobacillus lactis*. Due to its stability and availability, it is the form in which the vitamin is used most often. In fact, cyanocobalamin is formed as an artifact in the processing of biological materials. Cobalamins occur naturally as adenosylcobalamin and methylcobalamin, which instead of the cyano group contain a 5′-deoxyadenosyl residue and a methyl group respectively.

Adenosylcobalamin (coenzyme B_{12}) participates in rearrangement reactions in which a hydrogen atom and an alkyl residue, an acyl group or an electronegative group formally exchange places on two neighboring carbon atoms. Reactions of this type play a role in the metabolism of a series of bacteria. In mammals and bacteria a rearrangement reaction that depends on vitamin B_{12} is the conversion of methylmalonyl CoA to succinyl CoA (cf. 10.2.8.3). Vitamin B_{12} deficiency results in the excretion of methylmalonic acid in the urine.

Another reaction that depends on adenosylcobalamin is the reduction of ribonucleoside triphosphates to the corresponding 2′-deoxy compounds, the building blocks of deoxyribonucleic acids.

Methylcobalamin is formed, e. g., in the methylation of homocysteine to methionine with N^5-

methyltetrahydrofolic acid as the intermediate stage. The enzyme involved is a cobalamin-dependent methyl transferase.



(6.17)

The absorption of cyanocobalamin is achieved with the aid of a glycoprotein, the "intrinsic factor" formed by the stomach mucosa. Deficiency of vitamin B_{12} is usually caused by impaired absorption due to inadequate formation of "intrinsic factor" and results in pernicious anemia.

6.3.8.2 Requirement, Occurrence

The daily requirement of vitamin B_{12} is shown in Table 6.3. The plasma concentration is normally 450 pg/ml.

The ability of vitamin B_{12} to promote growth alone or together with antibiotics, for example in young chickens, suckling pigs and young hogs, is of particular importance. This effect appears to be due to the influence of the vitamin on protein metabolism, and it is used in animal feeding. The increase in feed utilization is exceptional with underdeveloped young animals. Vitamin B_{12} is of importance also in poultry operations (enhanced egg laying and chick hatching). The use of vitamin B_{12} in animal feed vitamin fortification is obviously well justified.

Liver, kidney, spleen, thymus glands and muscle tissue are abundant sources of vitamin B_{12} (Table 6.7). Consumption of internal organs (variety meats) of animals is one method of alleviating vitamin B_{12} deficiency symptoms in humans.

6.3.8.3 Stability, Degradation

The stability of vitamin B_{12} is very dependent on a number of conditions. It is fairly stable at pH 4–6, even at high temperatures. In alkaline media or in the presence of reducing agents, such as ascorbic acid or SO_2, the vitamin is destroyed to a greater extent.

6.3.9 L-Ascorbic Acid (Vitamin C)

6.3.9.1 Biological Role

Ascorbic acid (L-3-keto-threo-hexuronic acid-γ-lactone, Formula 6.18, I) is involved in hydroxylation reactions, e. g., biosynthesis of catecholamines, hydroxyproline and corticosteroids (11-β-hydroxylation of deoxycorticosterone and 17-β-hydroxylation of corticosterone). Vitamin C is fully absorbed and distributed throughout the body, with the highest concentration in adrenal and pituitary glands.

About 3% of the body's vitamin C pool, which is 20–50 mg/kg body weight, is excreted in the urine as ascorbic acid, dehydroascorbic acid (a combined total of 25%) and their metabolites, 2,3-diketo-L-gulonic acid (20%) and oxalic acid (55%). An increase in excreted oxalic acid occurs only with a very high intake of ascorbic acid. Scurvy is caused by a dietary deficiency of ascorbic acid.

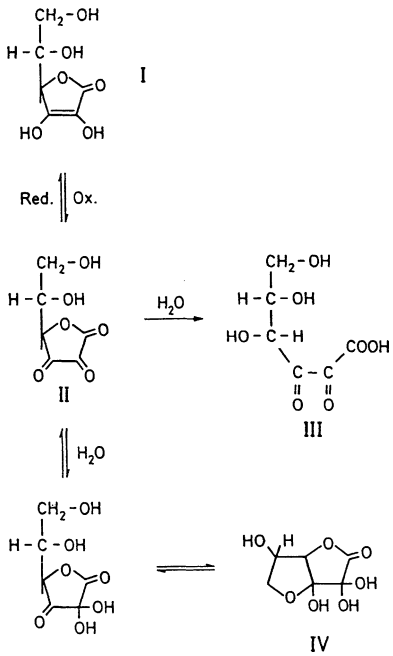
6.3.9.2 Requirement, Occurrence

The daily requirement is shown in Table 6.3. An indicator of insufficient vitamin supply in the diet is a low level in blood plasma (0.65 mg/100 ml). Vitamin C is present in all animal and plant cells, mostly in free form, and it is probably bound to protein as well. Vitamin C is particularly abundant in rose hips, black and red currants, strawberries, parsley, oranges, lemons (in peels more than in pulp), grapefruit, a variety of cabbages and potatoes. Vitamin C loss during storage of

vegetables from winter through late spring can be as high as 70%.
Table 6.7 provides data on vitamin C occurrence in a variety of foods.
Ascorbic acid is chemically synthesized. However, the synthesis by means of genetically modified microorganisms (GMO vitamin C) is more cost effective. Therefore, the largest proportion is synthesized by these means.

6.3.9.3 Stability, Degradation

Ascorbic acid (I) has an acidic hydroxyl group ($pK_1 = 4.04$, $pK_2 = 11.4$ at 25 °C). Its UV absorption depends on the pH value (Table 6.9). Ascorbic acid is readily and reversibly oxidized to dehydroascorbic acid (II), which is present in aqueous media as a hydrated hemiketal (IV). The biological activity of II is possibly weaker than that of I because the plasma and tissue concentrations of II are considerably lower after the administration of equal amounts of I and II. The activity is completely lost when the dehydroascorbic acid lactone ring is irreversibly opened, converting II to 2,3-diketogulonic acid (III), cf. Formula 6.18:

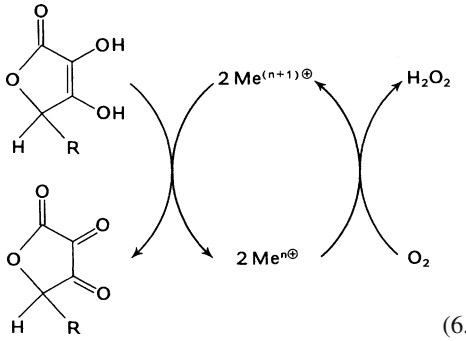


(6.18)

Table 6.9. Effect of pH on ultraviolet absorption maxima of ascorbic acid

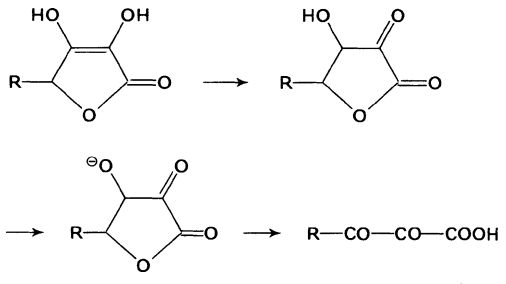
pH	λ max (nm)
2	244
6–10	266
>10	294

The oxidation of ascorbic acid to dehydroascorbic acid and its further degradation products depends on a number of parameters. Oxygen partial pressure, pH, temperature and the presence of heavy metal ions are of great importance. Metal-catalyzed destruction proceeds at a higher rate than noncatalyzed spontaneous autoxidation. Traces of heavy metal ions, particularly Cu^{2+} and Fe^{3+}, result in high losses.
The principle of metal catalysis is schematically presented in Reaction 6.19 (Me = metal ion).



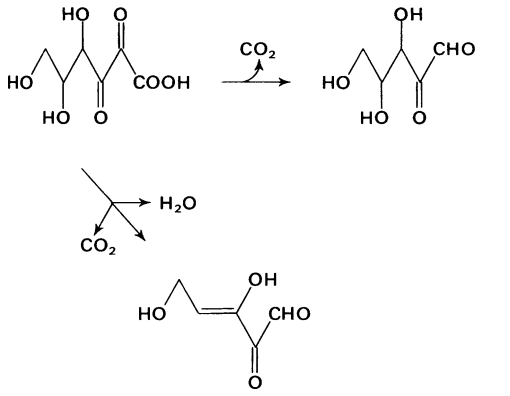
(6.19)

The rate of anaerobic vitamin C degradation, which is substantially lower than that of noncatalyzed oxidation, is maximal at pH 4 and minimal at pH 2. It probably proceeds through the ketoform of ascorbate, then via a ketoanion to diketogulonic acid:



(6.20)

Diketogulonic acid degradation products, xylosone and 4-deoxypentosone (Formula 6.21), are then converted into ethylglyoxal, various reductones (cf. 4.2.4.3.1), furfural and furancarboxylic acid.



(6.21)

In the presence of amino acids, ascorbic acid, dehydroascorbic acid and their degradation products might be changed further by entering into *Maillard*-type browning reactions (cf. 4.2.4.4). An example is the reaction of dehydroascorbic acid with amino compounds to give pigments, which can cause unwanted browning in citrus juices and dried fruits. The intermediates that have been identified are scorbamic acid (I in Formula 6.22), which is produced by *Strecker* degradation with an amino acid, and a red pigment (II). A wealth of data is available on ascorbic acid losses during preservation, storage and processing of food. Tables 6.1 and 6.2 and Figs. 6.3 and 6.4 present several examples. Ascorbic acid degradation is often used as a general indicator of changes occurring in food.

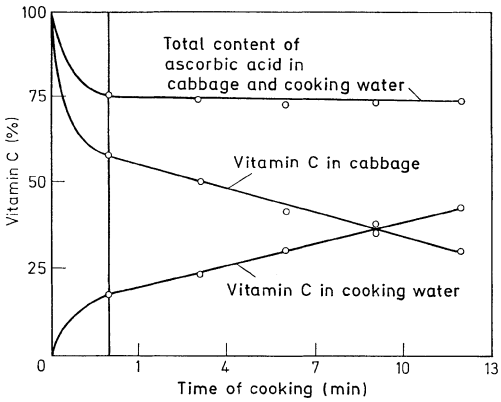
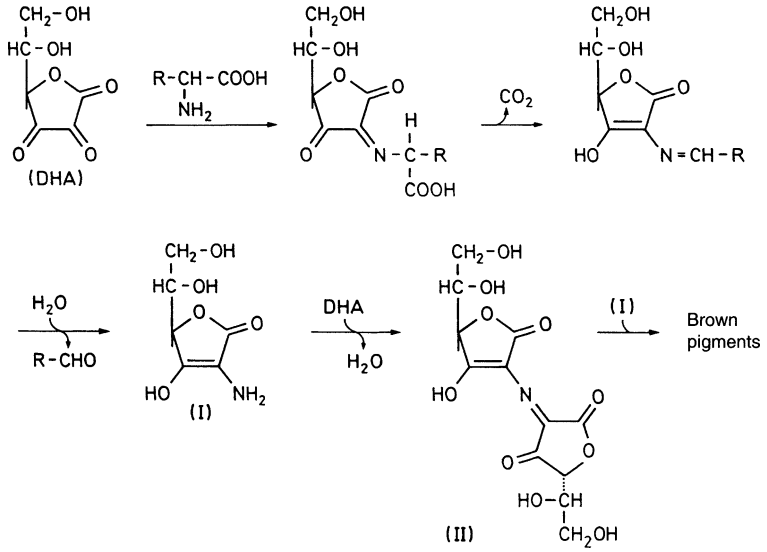


Fig. 6.3. Ascorbic acid losses as a result of cooking of cabbage (according to *Plank*, 1966)



(6.22)

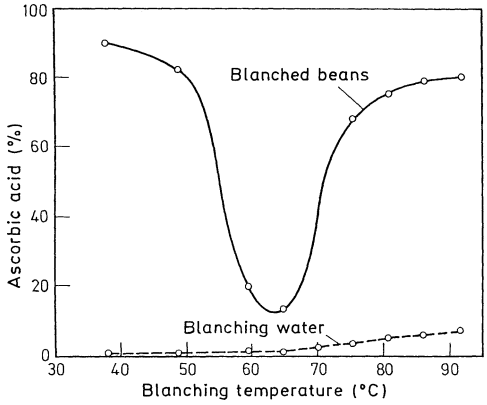


Fig. 6.4. Ascorbic acid losses in green beans versus blanching temperature (according to *Plank*, 1966)

6.4 References

Bässler, K.H.: On the problematic nature of vitamin E requirements: net vitamin E.Z. Ernährungswiss. *30*, 174 (1991)

Bässler, K.H., Lang, K.: Vitamine. Dr. Dietrich Steinkopff Verlag: Darmstadt. 1975

Booth, S.L., Mayer, J.: A hydrogenated form of vitamin K in food supply. Inform *11*, 258 (2000)

Counsell, J.N., Hornig, D.H. (Eds.): Vitamin C (Ascorbic Acid). Applied Science Publ.: London 1981

Deutsche Gesellschaft für Ernährung: Referenzwerte für die Nährstoffzufuhr. 1. Aufl. Umschau Braus Verlagsgesellschaft, Frankfurt a.M., 2000

Farrer, K.T.H.: The thermal destruction of vitamin B_1 in foods. Adv. Food Res. *6*, 257 (1955)

Gregory, J.F., Quinlivan, E.P., Davis, S.R.: Integrating the issues of folate bioavailability, intake and metabolism in the era of fortification. Trends Food Sci. Technol. *16*, 229 (2005)

Isler, O., Brubacher, G.: Vitamine I. Fettlösliche Vitamine. Georg Thieme Verlag: Stuttgart. 1982

Isler, O., Brubacher, G., Ghisla, S., Kräutler, B.: Vitamine II. Wasserlösliche Vitamine. Georg Thieme Verlag: Stuttgart, 1988

Körner, W.F., Völlm, J.: Vitamine. In: Klinische Pharmakologie und Pharmakotherapie. 3. Aufl. (Eds.: Kuemmerle, H.P., Garrett, E.R., Spitzy, K.H.), p. 361, Urban u. Schwarzenberg: München. 1976

Labuza, T.P., Riboh, D.: Theory and application of *Arrhenius* kinetics to the prediction of nutrient losses in foods. Food Technol. *36* (10), 66 (1982)

Liao, M.-L., Seib, P.A.: Chemistry of L-ascorbic acid related to foods. Food Chem. *30*, 289 (1988)

Lund, D.B.: Effect of commercial processing on nutrients. Food Technol. *33* (2), 28 (1979)

Machlin, L.J. (Ed.): Handbook of Vitamins. Marcel Dekker: New York. 1984

Ogiri, Y., Sun, F., Hayami, S., Fujimura, A., Yamamoto, K.; Yaita, M., Kojo, S.: The low vitamin C activity of orally administered L-dehydroascorbic acid. J. Agric. Food Chem. *50*, 227 (2002)

Plank, R.: Handbuch der Kältetechnik. Bd. XIV, p. 475, Springer-Verlag: Berlin. 1966

Rehner, G., Daniel, H.: Biochemie der Ernährung. Spektrum Akademischer Verlag, Heidelberg, 1999

Reiff, F., Paust, J., Meier, W., Fürst, A., Ernst, H.G., Kuhn, W., Härtner, H., Florent, J., Suter, C., Pollak, P.: Vitamine. Ullmanns Encyklopädie der technischen Chemie, 4th edn. Vol. 23, p. 621, Verlag Chemie: Weinheim. 1983

Rychlik, M.: Quantification of free and bound pantothenic acid in foods and blood plasma by stable isotope dilution assay. J. Agric. Food Chem. *48*, 1175 (2000)

Seib, P.A., Tolbert, B.M. (Eds.): Ascorbic acid: Chemistry, metabolism, and uses. Advances in Chemistry Series 200, American Chemical Society: Washington, D.C. 1982