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Vitamins

Biochemistry

Classification of vitamins, sources, functions, daily requirements, deficiency disorders and toxicity of fat-soluble and water-soluble vitamins.

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CHAPTER 06

Vitamins

Fat- and water-soluble vitamins, dietary requirements, deficiencies and toxicity

1. Definition and classification

Vitamins are organic micronutrients needed in small amounts for normal metabolism, growth and physiological function. They do not directly supply energy. Most must be obtained from food; some can be synthesized in limited amounts. Fat-soluble vitamins are A, D, E and K; water-soluble vitamins include the B-complex and C. Vitamin B12 is stored substantially in the liver, despite being water-soluble.

2. Fat-soluble vitamins

Vitamin	Sources and functions	Deficiency	Excess / toxicity
A (retinoids)	Liver, dairy, eggs; carotenoids in carrots and leafy vegetables. Vision, epithelial integrity and immunity.	Night blindness, xerophthalmia, keratomalacia	Preformed vitamin A excess: liver injury, headache, fetal harm
D (calciferol)	Sunlight-driven skin synthesis, fortified foods, oily fish. Calcitriol promotes calcium and phosphate absorption.	Rickets in children, osteomalacia in adults	Hypercalcaemia, renal injury
E (tocopherols)	Vegetable oils, seeds, nuts. Antioxidant protecting membranes.	Neuropathy, ataxia, haemolysis in susceptible individuals	High supplemental doses may increase bleeding risk
K (phylloquinone, menaquinones)	Leafy vegetables, some fermented foods. Activates clotting factors II, VII, IX, X and proteins C and S.	Bleeding, prolonged PT/INR	Low natural-form toxicity; interacts with warfarin

3. Water-soluble vitamins

Vitamin	Sources and key biochemical role	Deficiency and excess
B1 Thiamine	Whole grains, legumes, pork; TPP in oxidative decarboxylation	Beriberi; Wernicke encephalopathy. Oral toxicity uncommon.
B2 Riboflavin	Milk, eggs, grains; FAD and FMN for redox reactions	Cheilosis, angular stomatitis, glossitis. Toxicity uncommon.

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B3 Niacin	Meat, fish, nuts; NAD/NADP redox reactions	Pellagra: dermatitis, diarrhoea, dementia (and death untreated). High doses: flushing, liver injury.
B5 Pantothenate	Widely distributed foods; coenzyme A	Rare deficiency, fatigue and paraesthesia. High doses may cause diarrhoea.
B6 Pyridoxine	Fish, meat, legumes, potatoes; PLP in amino acid metabolism and haem synthesis	Neuropathy, dermatitis, microcytic anaemia; excess supplements can cause sensory neuropathy.
B7 Biotin	Eggs, nuts, legumes; carboxylase cofactor	Dermatitis, alopecia. High supplements can interfere with laboratory assays.

Vitamin	Sources and key biochemical role	Deficiency and excess
B9 Folate	Leafy vegetables, legumes; one-carbon transfer and DNA synthesis	Megaloblastic anaemia, fetal neural tube defects; excess folic acid may mask B12-related anaemia.
B12 Cobalamin	Animal foods, fortified foods; methionine synthase and methylmalonyl-CoA mutase	Megaloblastic anaemia, neuropathy, raised methylmalonic acid. Toxicity uncommon.
C Ascorbic acid	Amla, guava, citrus, vegetables; collagen synthesis, antioxidant, iron absorption	Scurvy, bleeding gums, poor wound healing. High doses may cause GI upset and increase stone risk in susceptible people.

4. Daily intake recommendations for healthy adults

Indian ICMR-NIN 2020 values reproduced in the FSSAI summary [1]. Figures are for non-pregnant reference adults with sedentary activity unless stated; dietary requirements differ in pregnancy, lactation, age and illness. AI = adequate intake; IU = international units. Values are not therapeutic replacement doses.

Vitamin	Men / women per day
A	1000 / 840 microg
D	600 IU (15 microg) each
E	7.5-10 mg tocopherol (adult reference)
K	55 microg (adult reference)
B1	1.4 / 1.4 mg
B2	2.0 / 1.9 mg
B3	14 / 11 mg
B5	5 mg each (AI)
B6	1.9 mg each
B7	40 microg each (AI)

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B9	300 / 220 microg
B12	2.2 microg each
C	80 / 65 mg

5. Vitamin absorption and storage

Fat-soluble vitamins are absorbed with dietary lipids and depend on adequate bile and pancreatic function. They may accumulate in the body, particularly vitamins A and D; high-dose supplements can cause toxicity. Most water-soluble vitamins have limited storage, although B12 is an important exception. Dietary deficiency, impaired absorption, increased demand and drug interactions can produce deficiency even when some intake is present.

6. Coenzyme forms and biochemical pathways

Vitamin	Active form or derivative	Key reaction
B1	Thiamine pyrophosphate (TPP)	Pyruvate and alpha-ketoglutarate dehydrogenases; transketolase
B2	FAD, FMN	Oxidation-reduction reactions

Vitamin	Active form or derivative	Key reaction
B3	NAD+, NADP+	Electron transfer and reductive synthesis
B5	Coenzyme A	Acyl-group transfer
B6	Pyridoxal phosphate (PLP)	Transamination and decarboxylation
B7	Biotin	ATP-dependent carboxylation
B9	Tetrahydrofolate derivatives	One-carbon transfer and nucleotide synthesis
B12	Methylcobalamin, adenosylcobalamin	Methionine synthesis and methylmalonyl-CoA rearrangement

7. Distinguishing major deficiency syndromes

Vitamin A and D

Vitamin A-derived retinal participates in the visual cycle; deficiency initially affects dark adaptation and may progress to ocular injury. Vitamin D promotes mineral homeostasis. Rickets affects growing bones, while osteomalacia describes defective mineralization of adult bone.

Folate and vitamin B12

Both deficiencies can impair DNA synthesis and cause megaloblastic anaemia. B12 deficiency can cause neurological injury even without obvious anaemia. Folate alone may improve blood findings without treating B12-related neurological damage; suspected deficiency requires appropriate testing and treatment.

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8. Biochemical and nursing importance

Vitamin D activation occurs via liver 25-hydroxylation and kidney 1alpha-hydroxylation. Vitamin B12 absorption requires intrinsic factor and a functioning terminal ileum. Fat malabsorption increases risk of A, D, E and K deficiency. In at-risk patients with suspected thiamine deficiency, prompt thiamine treatment is time-critical; emergency glucose for hypoglycaemia must not be delayed. Teach food-based nutrition, identify supplement use, monitor prescribed replacement and report suspected deficiency or toxicity. High-dose supplements should not be used without appropriate assessment [2].

9. Important Examination Questions

Long Answer Questions

1. 1. Classify vitamins and describe sources, functions and deficiency disorders.
2. 2. Describe the fat-soluble vitamins with sources, functions and deficiency manifestations.
3. 3. Explain the biochemical roles of water-soluble vitamins and their coenzyme forms.

Short Answer Questions

1. 1. Write a short note on B12 absorption.
2. 2. Write a short note on vitamin D activation.
3. 3. Write a short note on pellagra.
4. 4. Write a short note on scurvy.

Very Short Answer Questions

1. 1. Which deficiency causes pellagra? - Niacin (B3).
2. 2. Which vitamin deficiency causes scurvy? - Vitamin C.
3. 3. What vitamin is needed for intrinsic-factor-dependent absorption? - Vitamin B12.
4. 4. Where is 25-hydroxylation of vitamin D performed? - Primarily the liver.

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Additional objective question

Which vitamin is required for gamma-carboxylation of clotting factors? - Vitamin K.

10. Quick Revision

- Fat-soluble vitamins: A, D, E, K; water-soluble: B group and C.
- Vitamin K is required for normal activation of several coagulation proteins.
- Vitamin C supports collagen hydroxylation and non-haem iron absorption.
- Many B vitamins serve as precursors of coenzymes.

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Conclusion

Understanding vitamin function, absorption, deficiency and toxicity supports nutritional evaluation and safe patient education.

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Practice questions

Choose one best answer. The answer key and explanations follow the questions.

1. Deficiency of which vitamin causes pellagra?

A. Riboflavin
B. Niacin
C. Thiamine
D. Biotin

2. Which vitamin requires intrinsic factor and a functioning terminal ileum for absorption?

A. Vitamin C
B. Vitamin B6
C. Vitamin B12
D. Vitamin B2

3. Which vitamin supports activation of clotting factors II, VII, IX and X?

A. Vitamin A
B. Vitamin D
C. Vitamin E
D. Vitamin K

4. A patient with prolonged poor intake has bleeding gums and impaired wound healing. Deficiency of which vitamin best explains defective collagen support?

A. Vitamin B12
B. Vitamin D
C. Vitamin C
D. Vitamin K as the collagen cofactor

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Answer key and explanations

1. B - Niacin

Niacin deficiency produces pellagra, classically dermatitis, diarrhoea and dementia.

2. C - Vitamin B12

B12 absorption depends on intrinsic factor and the terminal ileum.

3. D - Vitamin K

Vitamin K is needed to activate these clotting factors.

4. C - Vitamin C

Vitamin C is required for normal collagen synthesis. Its deficiency can cause fragile tissues, bleeding gums and poor wound healing.

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