Biochemistry

Unit 4 · Lesson 4 · Fat-soluble vitamins and minerals

The notes for Lesson 4 of Unit 4: chapters 4.7 to 4.8 of the unit book, with their figures, worked examples, exam traps and quizzes, in one file. The full unit book, with every chapter, the exam question bank and the glossary, is linked at the end.

Lesson 4 of 4 | 2 chapters | Chapters 4.7 to 4.8

About these notes

These are the notes for **Lesson 4 · Fat-soluble vitamins and minerals** of the University free n easy video lessons on Biochemistry Unit 4, Nucleic Acids, Vitamins and Minerals. They are chapters 4.7 to 4.8 of the unit book, unchanged, so a page here is the same page in the full book.

4.7 Fat-soluble vitamins: A, D, E and K

Vitamins A, D, E and K are oily molecules built from isoprene units, so they dissolve in fat, not water. They travel with dietary fat, are stored in the liver and fat tissue, and most of them act less like coenzymes and more like hormones or membrane protectors: A for vision and gene control, D for calcium, E against oxidation, K for blood clotting. Because they are stored, too much can be as harmful as too little.

You need: isoprenoids, cholesterol and steroids from Unit 3 (Lipids); calcium from Chapter 4.8 links to vitamin D.

Common features

- Absorbed in the small intestine inside bile-salt micelles together with fat, then carried in chylomicrons. Any condition that blocks fat absorption (obstructed bile flow, chronic pancreatitis, cystic fibrosis) can cause all four deficiencies.
- Stored: A and D mainly in the liver, E in fat tissue. Deficiency develops slowly; excess builds up, so A and D can be toxic.

Vitamin A (retinoids)

Vitamin A exists as three related molecules: **retinol** (the alcohol; stored in the liver as retinyl esters), **retinal** (the aldehyde; used in vision) and **retinoic acid** (the acid; controls genes through nuclear receptors, needed for growth and for healthy skin and mucous membranes). Retinol and retinal interconvert, but retinoic acid cannot go back, so it cannot support vision. Plants supply **β-carotene**, a provitamin that the intestine cleaves into retinal.

Vision (Wald's visual cycle). Rod cells contain **rhodopsin**, the protein opsin bound to 11-*cis*-retinal. Light switches 11-*cis*-retinal to all-*trans*-retinal; opsin changes shape, activates the G protein transducin, and a phosphodiesterase lowers cGMP, closing sodium channels and sending a nerve signal. All-*trans*-retinal then leaves opsin and is converted back to the 11-*cis* form before rhodopsin can be rebuilt. In dim light the rods depend on this cycle, so the first sign of deficiency is **night blindness**, followed by xerophthalmia (dry eye), Bitot's spots and keratomalacia, which can blind a child. India runs vitamin A supplementation for young children for this reason. Excess vitamin A is toxic and harms the developing foetus.

Vitamin D (calciferols)

Vitamin D is really a prohormone. **Cholecalciferol (D3)** is made in the skin when ultraviolet-B light opens a ring of 7-dehydrocholesterol; **ergocalciferol (D2)** comes from ergosterol in fungi and yeast. Neither is active until it has been hydroxylated twice.

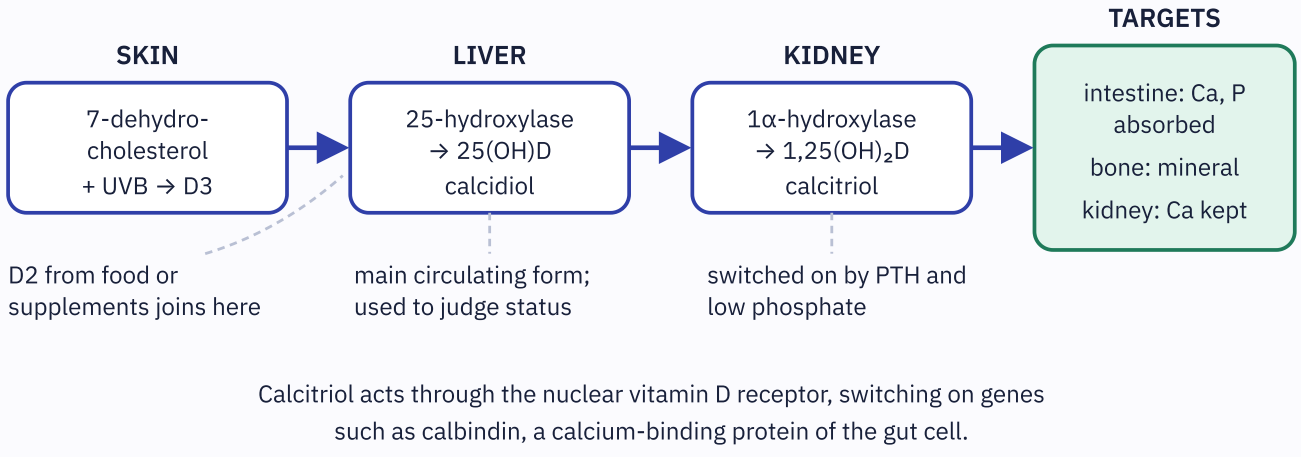


Figure 4.7.1 Vitamin D becomes a hormone in three organs. Notice which form is which: calcidiol (25-hydroxy) is the storage and status marker made by the liver; calcitriol (1,25-dihydroxy) is the active hormone made by the kidney under control of parathyroid hormone (PTH).

Calcitriol raises blood calcium and phosphate, mainly by increasing their absorption from the gut. Deficiency gives **rickets** in children (soft, bent bones, bowed legs, a beaded "rickety rosary"

on the ribs) and **osteomalacia** in adults. Surveys find vitamin D deficiency common in India despite plentiful sunshine, linked to indoor life, covered skin, pigmentation and air pollution. Excess causes high blood calcium and calcium deposits in soft tissues.

Vitamin E (tocopherols)

Vitamin E is a family of tocopherols and tocotrienols; **α-tocopherol** is the most active. It sits in membranes and lipoproteins and is the main fat-soluble **chain-breaking antioxidant**: it gives a hydrogen atom to a lipid peroxyl radical, stopping the chain reaction that would destroy polyunsaturated fatty acids. The tocopheroxyl radical formed is regenerated by vitamin C. Deficiency is rare and mostly seen in premature babies (haemolytic anaemia, because red-cell membranes break) and in long-term fat malabsorption (nerve and muscle damage). Vegetable oils, nuts and wheat germ are rich sources.

Vitamin K (quinones)

K1 (phylloquinone) comes from green leafy vegetables, K2 (menaquinones) from bacteria including gut bacteria, and K3 (menadione) is synthetic. Reduced vitamin K is the cofactor of **γ-glutamyl carboxylase**, which adds a carboxyl group to specific glutamate residues, making γ-carboxyglutamate (Gla). Gla residues bind Ca^{2+}, which lets clotting **factors II (prothrombin), VII, IX and X** and proteins C and S attach to phospholipid surfaces where clotting happens. In the reaction vitamin K becomes an epoxide, and the enzyme vitamin K epoxide reductase recycles it. The anticoagulant **warfarin** blocks this reductase. Newborns have little vitamin K (poor transfer across the placenta and a sterile gut), so a vitamin K injection is given at birth to prevent bleeding.

WORKED EXAMPLE

Question (5 marks). (a) A vitamin D3 sachet contains 60,000 IU. Express this in micrograms, given 1 µg = 40 IU. (b) A food label lists 3,000 IU of retinol, and the same food also supplies 6 mg of dietary β-carotene. Using 1 IU retinol = 0.3 µg retinol activity equivalents (RAE) and 12 µg dietary β-carotene = 1 µg RAE, find the total vitamin A activity in µg RAE.

1. (a) 60,000 IU ÷ 40 IU per µg = **1,500 µg** = 1.5 mg of cholecalciferol.
2. (b) Retinol: 3,000 × 0.3 = 900 µg RAE.
3. β-carotene: 6 mg = 6,000 µg; 6,000 ÷ 12 = 500 µg RAE.
4. Total = 900 + 500 = **1,400 µg RAE**. Note that β-carotene counts far less than retinol per microgram, because it is absorbed and converted inefficiently. (These conversion factors are the US ones; check the convention your textbook uses.)

EXAM TRAPS

- Calcidiol is 25(OH)D from the liver; calcitriol is 1,25(OH)$_2$D from the kidney. Calcitriol is the active form.
- Retinoic acid supports growth and gene control but not vision; only retinal works in rhodopsin.
- Vitamin K "K" comes from the German *Koagulation*, not potassium. Warfarin is a vitamin K antagonist.
- Vitamin E is an antioxidant, not a coenzyme; vitamin C regenerates it.
- IU conversions differ between vitamins: 1 µg vitamin D = 40 IU, but 1 IU retinol = 0.3 µg.

CHECK YOURSELF

– REMEMBER **Name the active form of vitamin D and the organ that makes it.** 1 mark

Calcitriol, 1,25-dihydroxycholecalciferol, made in the kidney.

– REMEMBER **List the vitamin K-dependent clotting factors.** 2 marks

Factors II (prothrombin), VII, IX and X (also proteins C and S).

– UNDERSTAND **Why is night blindness the earliest sign of vitamin A deficiency?** 3 marks

Rods, which work in dim light, need a steady supply of 11-cis-retinal to rebuild rhodopsin after each bleach. When vitamin A is short, rhodopsin regenerates slowly, so dim-light vision fails before the other tissues show damage.

– UNDERSTAND **How does vitamin E protect cell membranes?** 2 marks

α-Tocopherol donates a hydrogen atom to lipid peroxyl radicals, ending the chain reaction of lipid peroxidation that would damage polyunsaturated fatty acids.

– APPLY **Convert 25 µg of vitamin D to IU.** 1 mark

25 × 40 = **1,000 IU**.

– APPLY **A patient with blocked bile flow bruises easily. Which vitamin is involved and why?** 2 marks

Vitamin K. Without bile salts fat-soluble vitamins are poorly absorbed, so prothrombin and factors VII, IX and X are not carboxylated and clotting fails.

– ANALYZE **A child with chronic kidney disease develops rickets even with enough sunlight and diet. Explain.** 3 marks

Skin and liver make D3 and 25(OH)D normally, but the damaged kidney cannot carry out 1α-hydroxylation, so calcitriol is low; calcium absorption falls and bone is poorly mineralised. Treatment uses calcitriol or a 1α-hydroxylated analogue, not plain vitamin D.

BEYOND THE SYLLABUS

Golden Rice is a genetically engineered rice that makes β-carotene in its grain, designed for regions with vitamin A deficiency. Retinoic acid derivatives treat severe acne and a form of leukaemia (acute promyelocytic leukaemia), and newer oral anticoagulants act on clotting factors directly instead of on vitamin K recycling. Search “Golden Rice” and “vitamin D receptor gene regulation”. This is not for the exam.

4.8 Macro and micro minerals

Minerals are the inorganic elements the body needs from food. They build bone, carry charge across membranes, hold water in the right compartments, and sit in the active sites of enzymes. They are divided by how much we need: **macrominerals** (major elements) are needed at more than about 100 mg a day, and **microminerals** (trace elements) at less than that. A trace element is needed in small amounts, but it is no less essential; iron and iodine deficiency are among the most common nutritional problems in India.

You need: vitamin D and calcium absorption from Chapter 4.7; B12 (which contains cobalt) from Chapter 4.6.

Macrominerals

MINERAL	MAIN ROLES	TOO LITTLE OR TOO MUCH
Calcium (Ca)	About 99% is in bones and teeth as hydroxyapatite; the rest controls muscle contraction, nerve signalling, blood clotting and acts as a second messenger. Blood level about 9–11 mg/dL, held by PTH, calcitriol and calcitonin.	Low: rickets, osteomalacia, osteoporosis; tetany when ionised Ca falls. High: kidney stones, soft-tissue calcification.

MINERAL	MAIN ROLES	TOO LITTLE OR TOO MUCH
Phosphorus (P)	Bone mineral with Ca; part of ATP, nucleic acids, phospholipids; phosphate buffer	Low: rickets and muscle weakness
Magnesium (Mg)	Binds ATP: kinases use Mg–ATP, not free ATP; muscle and nerve function; bone	Low: tremor and tetany-like spasms
Sodium (Na)	Main cation outside cells; sets extracellular volume and osmotic pressure; nerve impulses	Low: hyponatraemia, confusion. High intake: raises blood pressure
Potassium (K)	Main cation inside cells; resting membrane potential; heart rhythm	Low or high: dangerous heart arrhythmias
Chloride (Cl)	Main anion outside cells; HCl of gastric juice; chloride shift in red cells	Lost with vomiting, giving alkalosis
Sulfur (S)	Taken in as methionine and cysteine; disulfide bonds; part of CoA, biotin and thiamine	No separate deficiency if protein is adequate

Sodium and potassium are kept on opposite sides of the membrane by the **Na^+/K^+-ATPase**, which pumps 3 Na^+ out and 2 K^+ in for each ATP it splits. This gradient drives nerve impulses and the uptake of glucose and amino acids in the gut and kidney.

Microminerals (trace elements)

ELEMENT	WHERE IT WORKS	DISORDER
Iron (Fe)	Haemoglobin, myoglobin, cytochromes, iron–sulfur proteins	Iron-deficiency anaemia; iron overload (haemochromatosis)
Zinc (Zn)	Carbonic anhydrase, alcohol dehydrogenase, carboxypeptidase; zinc-finger DNA-binding proteins; insulin storage	Poor growth, delayed wound healing, loss of taste; acrodermatitis enteropathica (inherited)
Copper (Cu)	Cytochrome c oxidase, Cu/Zn superoxide dismutase, lysyl oxidase (collagen cross-links), ceruloplasmin (oxidises Fe^{2+} for transport)	Wilson’s disease (copper builds up in liver and brain); Menkes disease (copper transport fails)
Iodine (I)	Thyroid hormones T3 and T4	Goitre; cretinism in children of deficient mothers
Selenium (Se)	As selenocysteine in glutathione peroxidase, thioredoxin reductase and the deiodinases that activate thyroid hormone	Keshan disease (a heart disease); toxic in excess

ELEMENT	WHERE IT WORKS	DISORDER
Manganese (Mn)	Arginase, mitochondrial superoxide dismutase, pyruvate carboxylase	Deficiency rare
Cobalt (Co)	Only as part of vitamin B12	Seen as B12 deficiency
Molybdenum (Mo)	Xanthine oxidase, sulfite oxidase	Deficiency rare
Fluorine (F)	Forms fluorapatite, which makes enamel resist acid	Low: dental caries. High: dental and skeletal fluorosis, found in areas of India with high-fluoride groundwater

India adds iodine to salt (universal salt iodisation) to prevent goitre, and gives iron and folic acid tablets under the Anaemia Mukt Bharat programme.

Iron: a mineral the body cannot excrete

Iron is the trace element examiners ask about most, because its handling is unusual: the body has **no regulated way to excrete iron**, so the total is controlled at absorption.

1. **In food:** haem iron (meat, fish) is absorbed well. Non-haem iron in plant foods is mostly Fe^{3+} and must be reduced to Fe^{2+}; vitamin C helps, while phytates (cereals), tannins (tea) and calcium hinder it.
2. **Into the gut cell:** Fe^{2+} enters cells of the duodenum through a transporter called DMT1. It is either stored there as **ferritin** or passed out of the cell through **ferroportin**.
3. **In blood:** leaving iron is oxidised to Fe^{3+} by copper-containing ferroxidases (hephaestin, ceruloplasmin) and carried by **transferrin**, two Fe^{3+} per molecule.
4. **Use and storage:** cells take up transferrin through transferrin receptors; bone marrow uses most iron to make haemoglobin. Liver stores iron as ferritin and haemosiderin.
5. **Control:** when iron stores are high, the liver releases the hormone **hepcidin**, which destroys ferroportin and so blocks absorption.

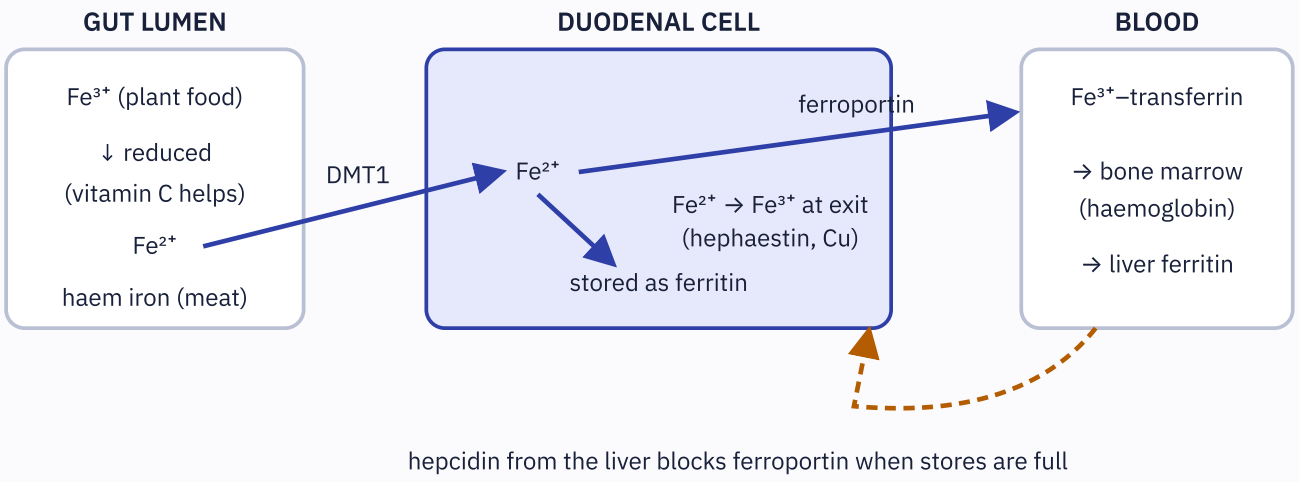


Figure 4.8.1 How dietary iron reaches the blood. Notice the two control points: iron can be locked in the gut cell as ferritin (and lost when the cell is shed), and hepcidin shuts the exit door, ferroportin, when the body has enough.

WORKED EXAMPLE

Question (6 marks). (a) Haemoglobin has a molar mass of about 64,500 g/mol and contains 4 iron atoms (Fe = 55.85 g/mol). Find the iron content in mg per gram of haemoglobin. (b) A woman has 12 g/dL haemoglobin and 4.5 L of blood. How much iron is in her circulating haemoglobin? (c) How many ATP molecules does the Na^+/K^+-ATPase use to move 3,000 Na^+ out of a cell, and how many K^+ come in?

1. (a) Iron per mole Hb = 4 × 55.85 = 223.4 g. Fraction = 223.4 ÷ 64,500 = 0.00346, so **3.46 mg iron per g Hb**.
2. (b) Blood = 4.5 L = 45 dL. Hb mass = 12 g/dL × 45 dL = 540 g.
3. Iron = 540 × 3.46 mg = 1,868 mg ≈ **1.9 g**. This is the largest single pool of body iron, which is why blood loss (heavy periods, hookworm, frequent pregnancies) leads to anaemia.
4. (c) The pump moves 3 Na^+ per ATP: 3,000 ÷ 3 = **1,000 ATP**, bringing in 1,000 × 2 = **2,000 K^+**.

EXAM TRAPS

- The macro/micro division is by daily need (about 100 mg/day), not by importance.
- Iron is absorbed as Fe^{2+} but carried by transferrin as Fe^{3+}; vitamin C helps by reducing Fe^{3+}.
- The body controls iron at absorption, not excretion. Hepcidin lowers absorption.
- Wilson's disease is copper excess in tissues; Menkes disease is copper deficiency. Ceruloplasmin is a copper protein that also oxidises iron.
- Selenium works as selenocysteine in glutathione peroxidase; do not write "selenium is a vitamin".

CHECK YOURSELF

– REMEMBER **List four macrominerals and four trace elements.** 2 marks

Macro: Ca, P, Mg, Na, K, Cl, S (any four). Trace: Fe, Zn, Cu, I, Se, Mn, Co, Mo, F (any four).

– REMEMBER **Name one enzyme each that needs zinc, copper and selenium.** 3 marks

Zinc: carbonic anhydrase. Copper: cytochrome c oxidase. Selenium: glutathione peroxidase.

– UNDERSTAND **Why does drinking tea with a vegetarian meal lower iron absorption, and lemon juice raise it?** 2 marks

Tannins in tea bind non-haem iron into forms that cannot be absorbed. Vitamin C in lemon reduces Fe^{3+} to Fe^{2+}, the form DMT1 carries, and keeps it soluble.

– UNDERSTAND **How does iodine deficiency cause goitre?** 3 marks

Without iodine the thyroid cannot make enough T3 and T4. Low hormone levels raise TSH from the pituitary, which keeps stimulating the thyroid to grow, and the gland enlarges.

– APPLY **A man has 15 g/dL haemoglobin and 5 L of blood. Estimate the iron in his haemoglobin.** 2 marks

Hb = 15 × 50 = 750 g; iron = 750 × 3.46 mg = 2,595 mg ≈ **2.6 g**.

– ANALYZE **A patient with copper deficiency also has anaemia even though iron intake is normal. Explain the link.** 4 marks

Iron leaves the gut cell and the liver as Fe^{2+} and must be oxidised to Fe^{3+} before transferrin can carry it. This is done by the copper-containing ferroxidases hephaestin and ceruloplasmin. With too little copper, iron is trapped in cells, too little reaches the bone marrow, and haemoglobin synthesis falls.

BEYOND THE SYLLABUS

Biofortified crops, such as high-iron pearl millet and high-zinc wheat varieties released in India, aim to raise mineral intake through ordinary food. An often-quoted estimate is that about a third of enzymes need a metal, and metalloprotein design is an active field of protein engineering. Hepcidin-based drugs are being tested for iron-overload diseases. Search “biofortification HarvestPlus” and “metalloenzymes”. This is not for the exam.

Key terms of this lesson

TERM	MEANING OR FORMULA	CHAPTER
Vitamin units	1 µg vitamin D = 40 IU; 1 IU retinol = 0.3 µg RAE; 12 µg dietary β-carotene = 1 µg RAE	4.7
Iron in haemoglobin	≈ 3.46 mg Fe per g Hb (4 × 55.85 ÷ 64,500)	4.8
Na^+/K^+-ATPase	3 Na^+ out, 2 K^+ in per ATP	4.8

Glossary (terms used in this lesson)

Calcitriol 1,25-dihydroxycholecalciferol, the active hormone form of vitamin D, made in the kidney.

Coenzyme A small organic molecule, often made from a vitamin, that an enzyme needs to catalyse its reaction.

Ferritin The protein that stores iron inside cells.

Hepcidin	Liver hormone that lowers iron absorption by destroying ferroportin.
Retinal	The aldehyde form of vitamin A; 11-cis-retinal binds opsin to form rhodopsin.
Trace element	A mineral needed at less than about 100 mg per day, such as iron, zinc or iodine.
Transferrin	The plasma protein that carries iron as Fe^{3+}.

The full unit book

Every chapter of Unit 4, the unit summary, the exam question bank with model answers and the full glossary: Biochemistry · Unit 4 · Nucleic Acids, Vitamins and Minerals (one file, works offline).

Sources and further reading

Standard textbooks

- David L. Nelson and Michael M. Cox, *Lehninger Principles of Biochemistry*: the chapters on nucleotides and nucleic acids, and on vitamins as coenzymes (from memory; check the chapter numbers in your edition).
- U. Satyanarayana and U. Chakrapani, *Biochemistry*: the chapters on nucleic acids, vitamins and minerals; widely prescribed in Indian universities (from memory; check).
- Jeremy M. Berg, John L. Tymoczko and Lubert Stryer (later editions with Gregory Gatto), *Biochemistry* (from memory; check).
- Donald Voet and Judith G. Voet, *Biochemistry*: detailed treatment of DNA conformations A, B and Z (from memory; check).

Open web resources

- Wikipedia, “Nucleic acid double helix”, comparison table of A-, B- and Z-DNA geometry: en.wikipedia.org/wiki/Nucleic_acid_double_helix
- NIH Office of Dietary Supplements, “Vitamin A and Carotenoids: Fact Sheet for Health Professionals”, for the IU and RAE conversion factors: ods.od.nih.gov/factsheets/VitaminA-HealthProfessional

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