PIPADOR REVISION

MRCOG Part 1 Revision Notes

Iron Metabolism

PHYSIOLOGY • LECTURE 161

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

161 Iron Metabolism

Distribution, absorption, regulation by hepcidin, pregnancy and fetal iron, iron studies, deficiency and overload

≈ 3–4 g	1–2 mg/day	≈ 1000 mg	< 30 µg/L
Total body iron (women ≈ 2.5–3 g)	Absorbed and lost (+ menses)	Extra iron needed in pregnancy	Ferritin = iron deficiency (pregnancy)

■ KEY FACTS: Learning objectives

- Describe body iron distribution, absorption, transport, storage and recycling.
- Describe the regulation of iron by hepcidin and the IRE/IRP system.
- Describe iron requirements in pregnancy and fetal iron acquisition.
- Interpret iron studies; describe the stages, treatment and prevention of iron deficiency and causes of iron overload.

161.1 Distribution and turnover

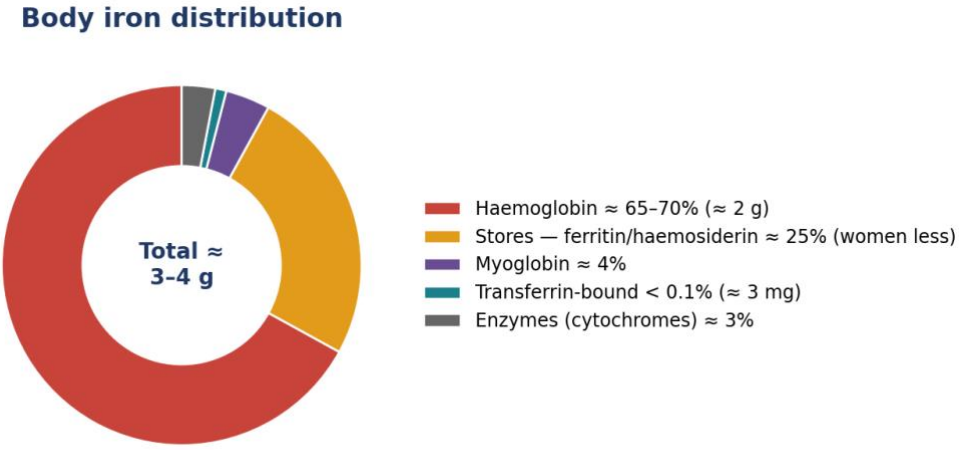


Figure 161.1 — Body iron distribution

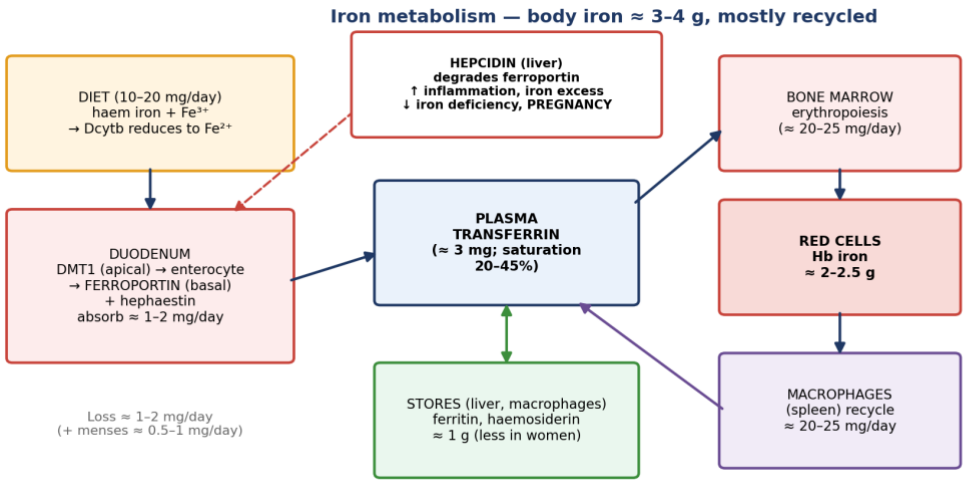


Figure 161.2 — Iron absorption, transport, use, storage and recycling

- **Closed system**: no regulated excretion — losses (≈ 1 mg/day in men; ≈ 1.5–2 mg/day in menstruating women) are passive (shed enterocytes, skin, blood). Balance is controlled **only at absorption**.
- **Recycling** of senescent red cells by splenic/hepatic macrophages supplies ≈ 20–25 mg/day — far more than absorption.

161.2 Absorption

Form	Mechanism	Enhancers / inhibitors
Haem iron (meat, fish)	Taken up intact (haem carrier) → haem oxygenase releases Fe^{2+}; ≈ 15–35% absorbed; little affected by diet	—
Non-haem iron (plants, fortified foods, supplements)	Fe^{3+} reduced to Fe^{2+} by duodenal cytochrome b (Dcytb) → DMT1 (apical) → stored as ferritin or exported by ferroportin + hephaestin (Fe^{2+} → Fe^{3+}) → transferrin	↑ Vitamin C, acid, meat ('meat factor'); ↓ phytates (cereals), tannins (tea, coffee), calcium, PPIs/antacids, coeliac disease, H. pylori, bariatric surgery

161.3 Regulation



Figure 161.3 — Hepcidin: the master regulator of iron

- **Systemic**: hepcidin binds **ferroportin** and causes its internalisation → iron is retained in enterocytes (lost when they shed) and macrophages. **Erythroferrone** from erythroblasts suppresses hepcidin when erythropoiesis is increased.
- **Cellular (IRE/IRP)**: in iron deficiency, iron-regulatory proteins bind iron-responsive elements → **↑ transferrin receptor 1** (mRNA stabilised) and **↓ ferritin** translation (and vice versa).
- **Clinical**: inflammation → ↑ hepcidin → **anaemia of chronic disease** and poor response to oral iron; a single oral dose raises hepcidin for ≈ 24 h → **alternate-day dosing** improves fractional absorption.

161.4 Iron in pregnancy and the fetus

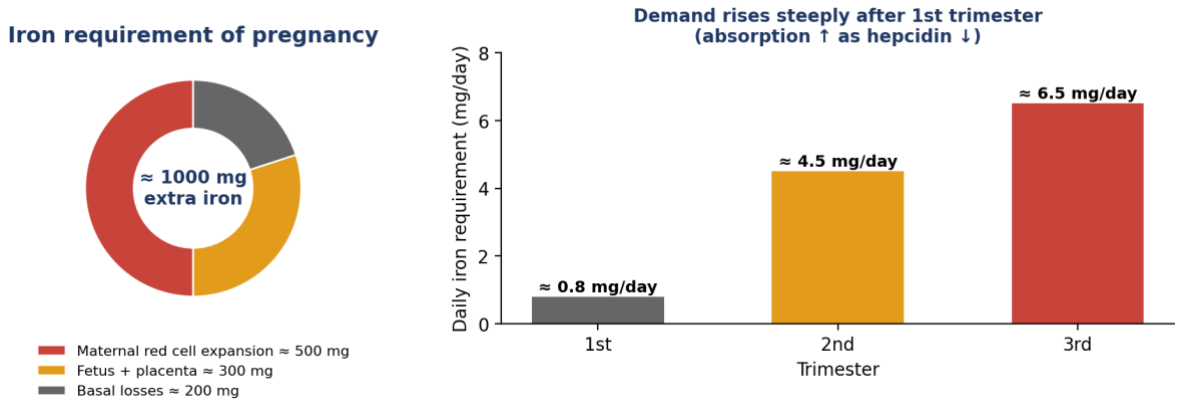


Figure 161.4 — Iron requirements of pregnancy

- Requirement ≈ **1000 mg**, mostly in the **2nd and 3rd trimesters** (≈ 6–7 mg/day at term) — met partly by amenorrhoea and ↑ **absorption** as hepcidin falls; many women start pregnancy with low stores.
- **Placental transfer is active and unidirectional**: maternal transferrin–iron binds **TfR1** on syncytiotrophoblast → endocytosis → **ferroportin** on the fetal side. The fetus is prioritised (fetal Hb maintained even with maternal deficiency) but severe maternal deficiency ↓ fetal stores.
- Fetus accrues ≈ **250–300 mg**, mainly in the 3rd trimester (≈ 75 mg/kg at term). **Delayed cord clamping** adds ≈ 30–50 mg; preterm and FGR infants and infants of diabetic mothers have low stores.
- **Consequences of maternal iron deficiency**: fatigue, ↓ work capacity, ↑ transfusion risk with PPH, postnatal depression; infant iron deficiency and **impaired neurodevelopment**.

161.5 Iron studies and deficiency

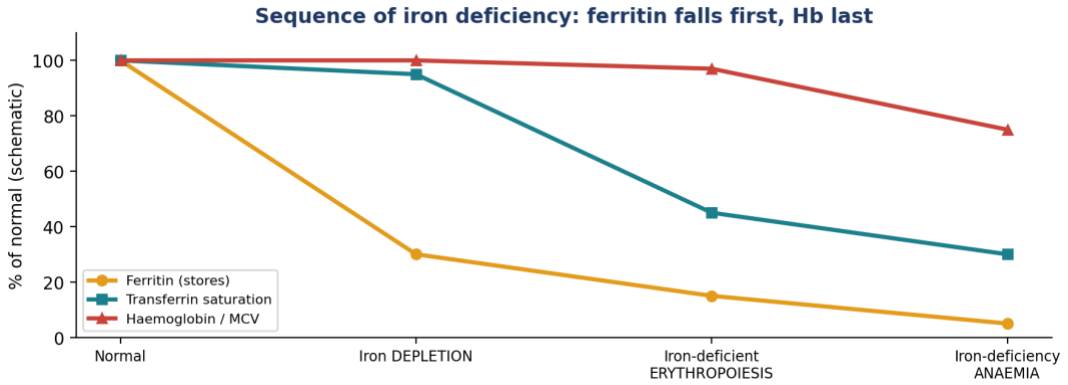


Figure 161.5 — Progression of iron deficiency

Test	Iron deficiency	Anaemia of inflammation	Iron overload
Ferritin	↓ (< 30 µg/L in pregnancy — BSH)	↔ / ↑ (acute-phase)	↑↑
Transferrin saturation	↓ (< 20%)	↓	↑ (> 45–50%)
Transferrin / TIBC	↑	↓	↓
Soluble TfR	↑	↔	↓
Zinc protoporphyrin	↑	↑	—
Reticulocyte Hb (CHr)	↓ (early marker)	↓	—

Treatment	Key points
Oral iron	Ferrous sulphate 200 mg (≈ 65 mg elemental), fumarate 210 mg (≈ 68 mg), gluconate 300 mg (≈ 35 mg); 40–80 mg elemental once daily or alternate days; take with vitamin C, avoid tea/calcium; GI side effects; check Hb at 2–4 weeks; continue ≈ 3 months after correction
IV iron	Ferric carboxymaltose, ferric derisomaltose, iron sucrose — from the 2nd trimester; rapid repletion; rare hypersensitivity (observe 30 min); hypophosphataemia with carboxymaltose
Transfusion	Reserved for severe/symptomatic anaemia or haemorrhage (Lecture 129)
Prevention	Diet, treat menorrhagia, inter-pregnancy interval; targeted supplementation (NICE: no routine iron in UK pregnancy)

161.6 Iron overload

Hereditary haemochromatosis (HFE **C282Y** homozygous; ↓ hepcidin) → ↑ absorption → liver cirrhosis, diabetes, cardiomyopathy, hypogonadism, arthropathy; venesection. **Transfusional overload** (thalassaemia major, sickle cell disease) — chelation (desferrioxamine; deferasirox/deferiprone avoided in pregnancy). **Iron poisoning** in children (prenatal supplements) — desferrioxamine.

161.7 Must-know summary

■ EXAM TIP

- Iron is absorbed in the **duodenum**: Dcytb → **DMT1** → **ferroportin** (+ hephaestin) → transferrin; no active excretion.
- **Hepcidin ↓ ferroportin**: ↑ in inflammation/iron excess; ↓ in deficiency, erythropoiesis (erythroferrone) and pregnancy.
- Ferritin falls first, then TSAT, then Hb; **ferritin < 30 µg/L** confirms deficiency in pregnancy.
- Pregnancy needs ≈ **1 g**; placental transfer is active (TfR1/ferroportin) and favours the fetus.
- Oral iron 40–80 mg elemental daily/alternate days; IV iron from 2nd trimester.

■ MNEMONIC / MEMORY AID

Absorption — 'Vitamin C helps, Tea and Tums (calcium/antacids) Trap'. **'Hepcidin Hides iron'** (locks ferroportin).