PIPADOR REVISION

MRCOG Part 1 Revision Notes

Fetal Endocrinology

ENDOCRINOLOGY • LECTURE 187

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | ENDOCRINOLOGY

187 Fetal Endocrinology

Fetal endocrine glands and transfer, fetal adrenal and HPA axis, thyroid, pancreas, growth, sexual differentiation and transition at birth

Fetal zone	Insulin	Maternal T_4	hCG → T
≈ 80% of adrenal at term (DHEAS)	Key fetal growth hormone (3rd tri)	Vital before ≈ 18–20 wk	Male differentiation 8–12 wk

■ KEY FACTS: Learning objectives

- Describe the development of fetal endocrine glands and the control of fetal hormones.
- Describe the fetal HPA axis, the fetal adrenal and the fetoplacental unit, and cortisol-driven maturation.
- Describe fetal thyroid, pancreatic, growth, gonadal and calcium endocrinology and maternal–fetal transfer.
- Describe the endocrine transition at birth and fetal endocrine disorders.

187.1 Development of fetal endocrine glands

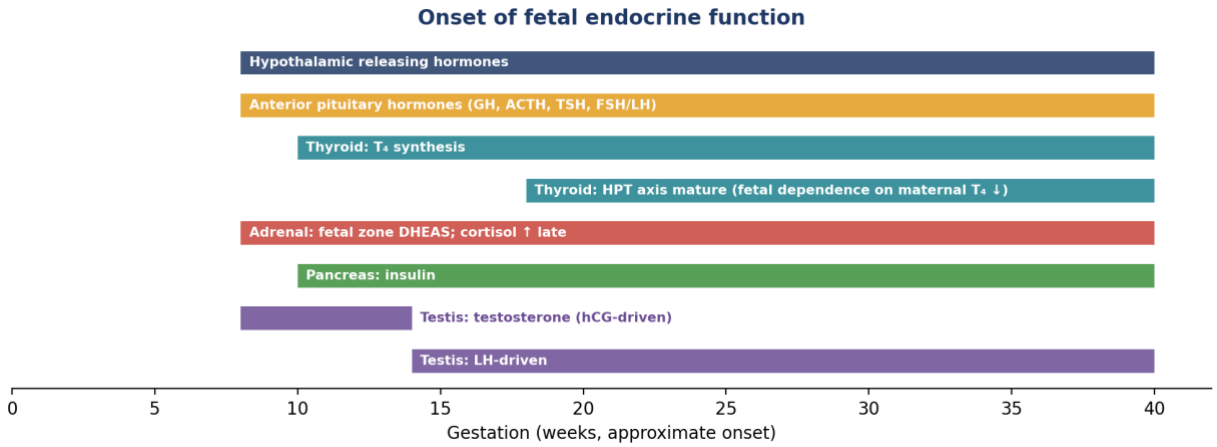


Figure 187.1 — Onset of fetal endocrine function

Maternal hormone	Placental transfer
Steroids	Cortisol/prednisolone largely inactivated by 11β-HSD2; betamethasone/dexamethasone cross
Thyroid	T_4 limited but important; TRH and iodide cross; TSH does not
Peptides	Insulin, GH, ACTH, PTH, TSH do not cross (fetus makes its own)
Antibodies (IgG)	TRAb (fetal thyrotoxicosis), anti-Ro (heart block), anti-AChR (neonatal MG) cross
Drugs	Carbimazole/PTU, metformin, androgens/anti-androgens cross

187.2 The fetal adrenal and HPA axis

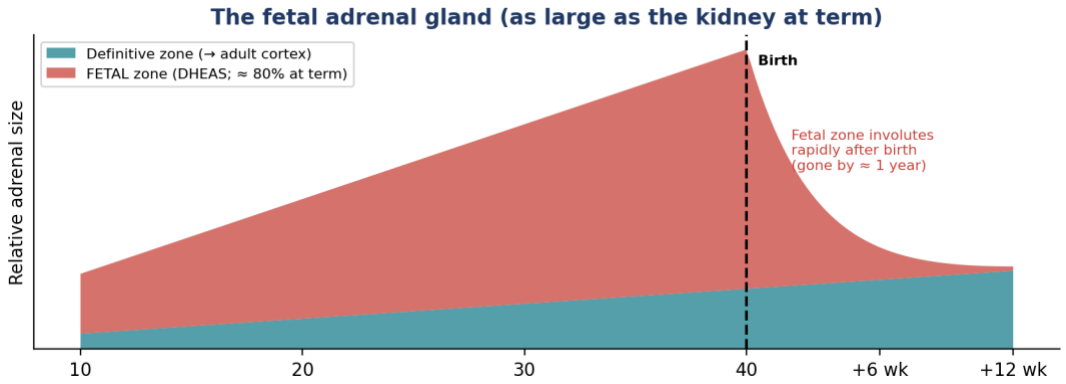


Figure 187.2 — Fetal and definitive zones of the adrenal

- The **fetal zone** (lacks 3β-HSD) produces large amounts of **DHEAS** (driven by fetal ACTH and **hCG**) → placental **oestrogens** via the fetoplacental unit (Lecture 182). It involutes after birth.
- The **definitive zone** produces aldosterone and, late in gestation (with the transitional zone), **cortisol** — fetal cortisol rises steeply in the last weeks (↑ ACTH, ↑ placental CRH).
- **Cortisol-driven maturation**: **lung** (surfactant, Na^+ channels), **liver** (glycogen, gluconeogenic enzymes), gut, thyroid (**T_4 → T_3 deiodination**), adrenal medulla (PNMT), and parturition signals (Lecture 185).

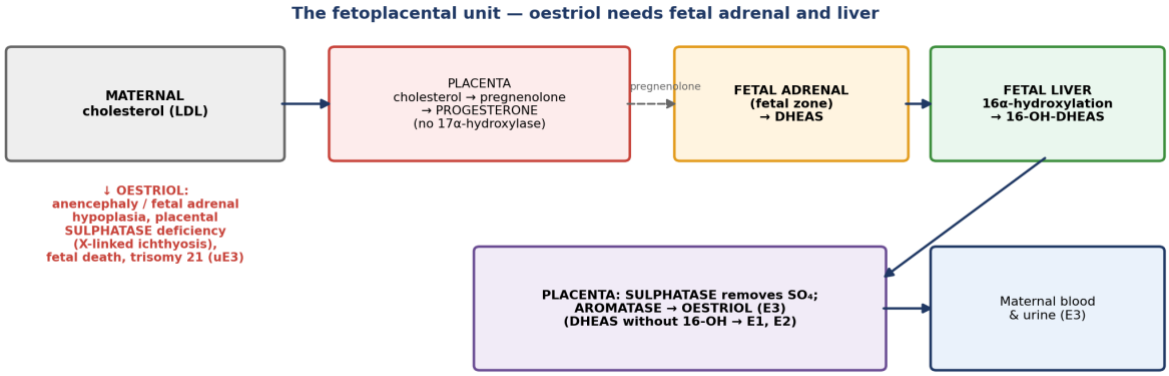
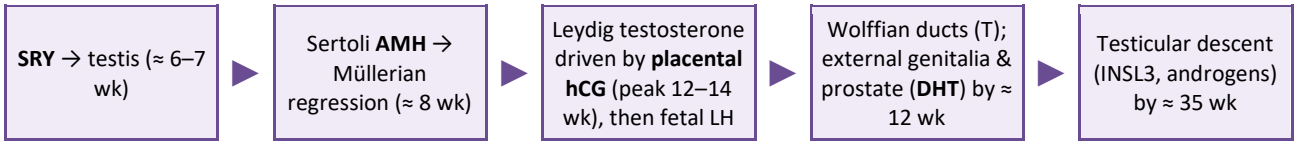


Figure 187.3 — The fetoplacental unit

187.3 Thyroid, pancreas and growth

System	Fetal endocrinology
Thyroid	T_4 synthesis from 10–12 wk; HPT axis matures 18–20 wk; fetal T_3 kept low (placental and fetal D3) until late gestation; neonatal TSH surge at birth. Maternal hypothyroxinaemia → neurodevelopmental impairment; congenital hypothyroidism (dysgenesis, dyshormonogenesis) — bloodspot screening day 5
Pancreas / insulin	Insulin from ≈ 10–12 wk; the major fetal growth hormone in the 3rd trimester (fat, glycogen); fetal hyperinsulinaemia (maternal diabetes) → macrosomia, delayed lung maturation, neonatal hypoglycaemia; insulin deficiency → severe FGR
IGFs / GH	IGF-2 (paternally imprinted) dominates early growth and placental growth; IGF-1 later, nutrient-regulated; GH has little role in fetal growth (anencephalic fetuses grow near-normally)
Calcium	Active placental Ca^{2+} transfer (PTHrP) → fetal hypercalcaemia suppresses fetal PTH; neonatal nadir 24–48 h (Lecture 160)
Posterior pituitary	AVP surges in fetal hypoxia (redistribution, ↓ urine); oxytocin present
Catecholamines	Fetal adrenal medulla and paraganglia (organ of Zuckerkandl); surge in hypoxia and at birth

187.4 Gonads and sexual differentiation



Female differentiation proceeds without gonadal hormones (no AMH → Müllerian ducts persist; no androgen → external female genitalia). Fetal ovaries contain oocytes from ≈ 20 weeks peak (Lecture 143). **Virilised 46,XX**: **21-hydroxylase CAH**, maternal androgen-secreting tumours/luteoma, placental aromatase deficiency, maternal drugs.

187.5 The endocrine transition at birth

Hormone	Change at birth	Purpose
Catecholamines	Surge (vaginal birth > elective CS)	Lung fluid absorption, surfactant release, glucose mobilisation, brown-fat thermogenesis, cardiovascular adaptation
Cortisol	↑	Glucose, blood pressure, lung
TSH → T_4/T_3	TSH surge within minutes (cold exposure)	Non-shivering thermogenesis
Glucagon / insulin	Glucagon ↑, insulin ↓	Glycogenolysis, gluconeogenesis after loss of placental glucose (glucose nadir 1–2 h)
Placental hormones	Fall rapidly (oestrogens → neonatal breast enlargement, 'witch's milk', withdrawal bleeding in girls)	—

187.6 Must-know summary

■ EXAM TIP

- Peptide hormones (insulin, GH, TSH, ACTH) **do not cross**; steroids are modified (11β-HSD2); IgG antibodies cross.
- **Fetal zone → DHEAS → placental oestriol**; involutes after birth.
- Late fetal **cortisol** matures lung, liver, gut and thyroid (T_4 → T_3).
- **Insulin** (3rd trimester) and **IGF-2** (early) drive growth; GH minor.
- Male differentiation: SRY → AMH + **hCG-driven** testosterone → DHT.

■ MNEMONIC / MEMORY AID

Fetal growth hormones — 'I Grow In Utero': Insulin, IGF-2, IGF-1 (GH not needed).