PIPADOR REVISION

MRCOG Part 1 Revision Notes

Pancreatic Hormones (Insulin and Glucagon)

ENDOCRINOLOGY • LECTURE 180

VISUAL EXAMINATION REVISION NOTES

2026 Edition

180

MRCOG PART 1 REVISION NOTES | ENDOCRINOLOGY

Pancreatic Hormones (Insulin and Glucagon)

Islet hormones, insulin secretion and action, glucose metabolism in pregnancy, and diabetes in pregnancy

β-cells	α-cells	↓ 50–60%	5.6 / 7.8
Insulin (≈ 70% of islet cells)	Glucagon	Insulin sensitivity by 3rd trimester	GDM: fasting / 2-h 75 g OGTT (NICE)

■ KEY FACTS: Learning objectives

- Describe the structure, secretion, receptors and actions of insulin and glucagon (and somatostatin, incretins).
- Describe fed/fasting glucose regulation and the metabolic adaptation of pregnancy.
- Describe gestational and pre-existing diabetes physiology, diagnosis and fetal consequences.
- Describe placental transfer of glucose, insulin and hypoglycaemic agents.

180.1 The islets of Langerhans

Cell	Hormone	Stimulus	Main actions
β (≈ 70%)	Insulin (51 aa; A + B chains; from proinsulin with C-peptide)	↑ Glucose, amino acids, incretins (GLP-1, GIP), vagus; ↓ by α_2 (adrenaline), somatostatin	Anabolic: GLUT4 glucose uptake (muscle, fat), glycogenesis, lipogenesis, protein synthesis; ↓ gluconeogenesis, lipolysis, ketogenesis; K^+ into cells
α (≈ 20%)	Glucagon	↓ Glucose, amino acids, sympathetic; ↓ by insulin, glucose	Catabolic (liver): glycogenolysis, gluconeogenesis, ketogenesis
δ	Somatostatin	Glucose, amino acids	Paracrine inhibition of insulin and glucagon
PP / ε	Pancreatic polypeptide / ghrelin	Meals	Appetite regulation

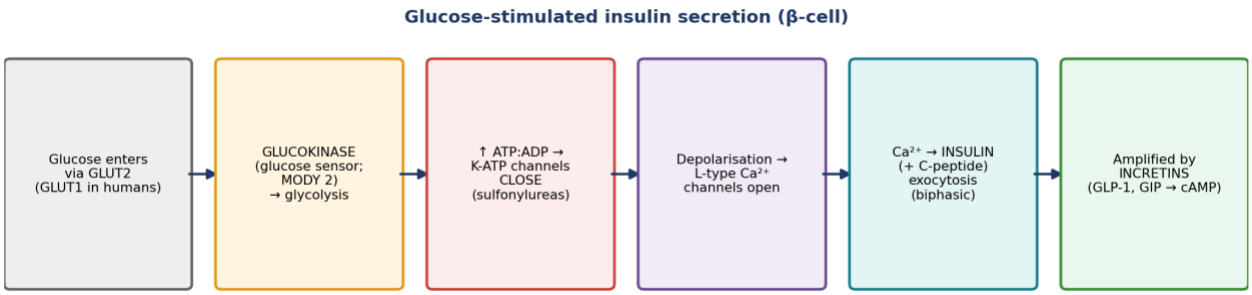


Figure 180.1 — Glucose-stimulated insulin secretion

Insulin receptor = receptor **tyrosine kinase** → IRS → **PI3K/AKT** (GLUT4 translocation, glycogen synthesis) and MAPK (growth). Insulin resistance (obesity, PCOS, pregnancy) is a post-receptor defect. **C-peptide** reflects endogenous secretion (low in type 1, high with insulinoma; low in factitious insulin use).

180.2 Glucose metabolism in pregnancy

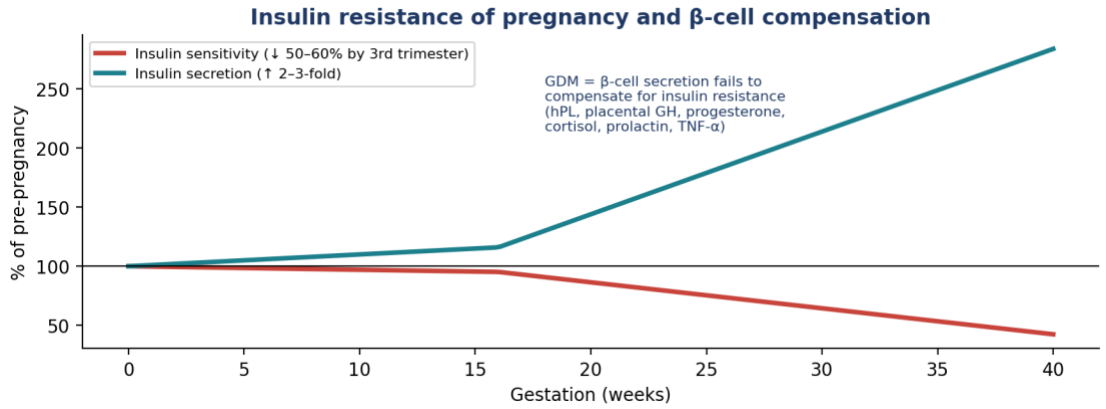


Figure 180.2 — Insulin sensitivity and secretion across pregnancy

Phase	Changes
Early (anabolic)	Normal or ↑ insulin sensitivity; β-cell hyperplasia (prolactin, hPL); fat storage; risk of hypoglycaemia in type 1 diabetes
Late (catabolic)	Insulin resistance (hPL, placental GH, progesterone, cortisol, prolactin, TNF-α, adipokines) → postprandial hyperglycaemia, lipolysis; secretion ↑ 2–3-fold to compensate
Fasting	'Accelerated starvation': fasting glucose lower (fetal uptake, ↑ plasma volume), faster ketosis — DKA can occur at near-normal glucose
After birth	Insulin resistance falls within hours of placental delivery → stop GDM treatment; halve/return insulin to pre-pregnancy doses in type 1/2

Maternal hyperglycaemia ▶ **Glucose crosses** (GLUT1, facilitated) — insulin does NOT ▶ **Fetal hyperinsulinaemia** (Pedersen) from ≈ 20 wk ▶ Macrosomia (shoulders, abdomen), organomegaly, polyhydramnios, ↑ O_2 demand (stillbirth) ▶ Neonatal **hypoglycaemia**, RDS (insulin delays surfactant), polycythaemia, jaundice, hypocalcaemia

180.3 Diabetes in pregnancy (NICE NG3)

Topic	Key points
GDM diagnosis	75 g OGTT at 24–28 weeks (earlier if previous GDM): fasting ≥ 5.6 mmol/L or 2-h ≥ 7.8 mmol/L. Risk factors: BMI > 30, previous macrosomia (≥ 4.5 kg), previous GDM, family history, high-risk ethnicity
Targets	Fasting < 5.3; 1-h post-meal < 7.8 or 2-h < 6.4 mmol/L
Treatment	Diet/exercise → metformin (crosses placenta) → insulin (does not cross); glibenclamide rarely
Pre-existing diabetes	Pre-conception HbA1c < 48 mmol/mol (avoid pregnancy if > 86); folic acid 5 mg; aspirin from 12 wk; retinal and renal assessment; congenital anomalies ↑ (cardiac, NTD, caudal regression) with poor control; fetal echo
Postnatal (GDM)	Fasting glucose at 6–13 weeks (or HbA1c after 13 weeks); annual HbA1c — ≈ 50% develop type 2 diabetes within 5–10 years
DKA in pregnancy	Fetal mortality high; occurs at lower glucose; triggers: hyperemesis, infection, steroids (fetal lung), β-agonists

180.4 Must-know summary

■ EXAM TIP

- β-cell: glucose → **glucokinase → ATP → K-ATP closure → Ca^{2+} → insulin**; incretins amplify.
- Insulin: tyrosine kinase receptor, GLUT4; glucagon: glycogenolysis, gluconeogenesis, ketogenesis.
- Pregnancy: insulin resistance (hPL, placental GH, progesterone, cortisol) ↑ in late gestation; **accelerated starvation** — DKA at lower glucose.
- **Glucose crosses; insulin does not; metformin does.** Pedersen: maternal hyperglycaemia → fetal hyperinsulinaemia → macrosomia.
- GDM OGTT: **≥ 5.6 fasting / ≥ 7.8 at 2 h**; stop treatment after birth; test at 6–13 weeks.

■ MNEMONIC / MEMORY AID

GDM numbers — '5-6-7-8': fasting ≥ 5.6, 2-h ≥ 7.8. **Neonatal effects of maternal diabetes — 'HHHH'**: Hypoglycaemia, Hypocalcaemia, Hyperbilirubinaemia, Hyaline membrane disease (RDS).