PIPADOR REVISION

MRCOG Part 1 Revision Notes

Inhibin

ENDOCRINOLOGY • LECTURE 173

VISUAL EXAMINATION REVISION NOTES

2026 Edition

173

MRCOG PART 1 REVISION NOTES | ENDOCRINOLOGY

Inhibin

Structure and mechanism, inhibin A and B in the cycle, ageing and pregnancy, and clinical applications

α + β	Inhibin B	Inhibin A	↓ FSH
Heterodimer (TGF-β family)	Follicular phase; Sertoli cells	Corpus luteum; placenta	Selective — LH unaffected

■ **KEY FACTS: Learning objectives**

- Describe the structure, sources and receptor mechanism of inhibins.
- Describe the patterns of inhibin A and B in the menstrual cycle, ageing and pregnancy.
- Describe the clinical uses of inhibin: aneuploidy screening, ovarian reserve, granulosa cell tumours, pre-eclampsia and male infertility.

173.1 Structure and mechanism

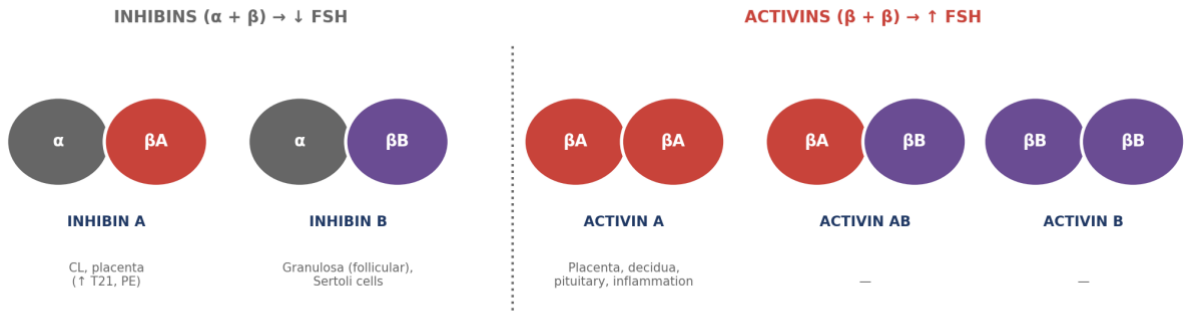


Figure 173.1 — Inhibins and activins share β-subunits

- Inhibins are **disulphide-linked heterodimers** of a common **α-subunit** with a **βA (inhibin A)** or **βB (inhibin B)** subunit — members of the **TGF-β superfamily** (Lecture 175).
- Mechanism: inhibin binds the co-receptor **betaglycan** and sequesters activin type II receptors → **antagonises activin** signalling in pituitary gonadotrophs → **selective suppression of FSH synthesis** (no effect on LH).
- Paracrine roles in the ovary/testis (↑ LH-stimulated theca androgen production — supports oestradiol synthesis).

173.2 Inhibin in the menstrual cycle

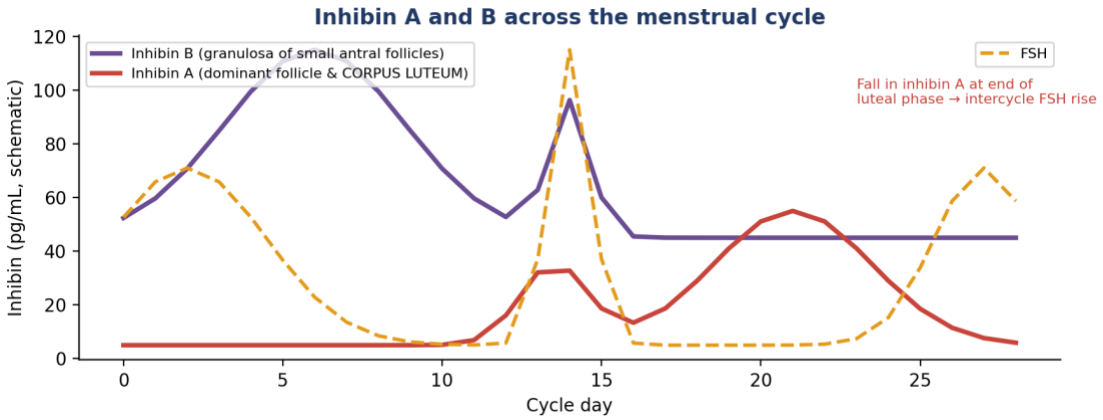


Figure 173.2 — Inhibin A and B across the cycle

	Inhibin B	Inhibin A
Source	Granulosa cells of small antral follicles (recruited cohort)	Dominant follicle and corpus luteum
Peak	Early–mid follicular phase (+ brief mid-cycle peak)	Mid-luteal phase
Role	Suppresses FSH → only the dominant follicle survives	Luteal FSH suppression; fall at luteolysis → intercycle FSH rise
Men	Sertoli cells — marker of spermatogenesis	Negligible

173.3 Ovarian ageing

With falling follicle numbers, **inhibin B declines early** (before oestradiol), releasing FSH from suppression — the **monotropic rise in early-follicular FSH** of the late reproductive years. AMH has largely replaced inhibin B as a marker of ovarian reserve (less cycle-dependent). After menopause inhibins are undetectable.

173.4 Inhibin in pregnancy

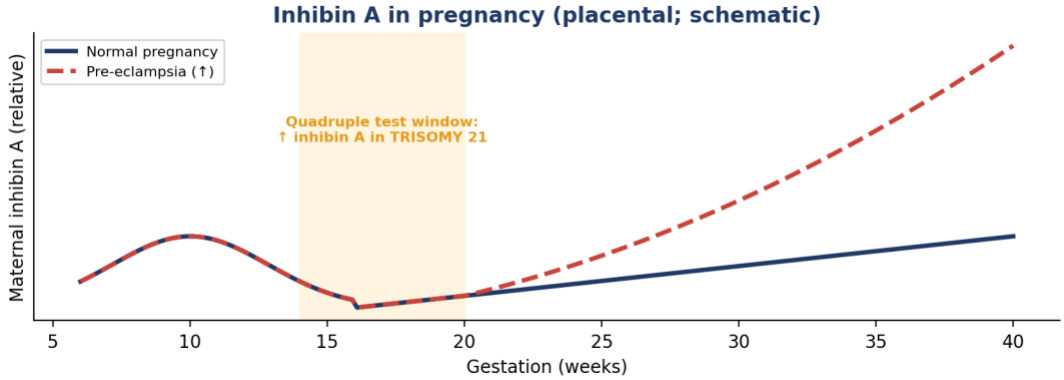


Figure 173.3 — Maternal inhibin A in normal pregnancy and pre-eclampsia

- Inhibin A in pregnancy is mainly **placental** (syncytiotrophoblast): peaks ≈ 8–10 weeks, falls to a nadir in the 2nd trimester, then rises in the 3rd trimester.
- **Quadruple test** (14+2–20+0 weeks): **inhibin A ↑ (≈ 2 MoM) in trisomy 21**, along with ↑ hCG, ↓ AFP and ↓ uE3 (Lecture 159).
- **Pre-eclampsia**: inhibin A and activin A are ↑ (placental stress) — research marker, superseded clinically by PlGF-based tests.

173.5 Inhibin as a tumour marker

Tumour	Marker profile
Granulosa cell tumour (adult — oestrogen-secreting; postmenopausal bleeding, endometrial hyperplasia)	↑ Inhibin B (and A), ↑ AMH; used for diagnosis and monitoring recurrence (late relapses)
Sertoli–Leydig cell tumour	May ↑ inhibin; androgen excess
Mucinous ovarian tumours	Inhibin may be raised
Histology	Inhibin-α immunostaining — sex cord–stromal tumours, adrenocortical tumours

Male infertility: low inhibin B with high FSH indicates impaired spermatogenesis (non-obstructive azoospermia), whereas normal inhibin B with azoospermia suggests obstruction.

173.6 Must-know summary

■ EXAM TIP

- Inhibin = **α + β** dimer (TGF-β family); selectively **↓ FSH** by antagonising activin (betaglycan).
- **Inhibin B**: small antral follicles (follicular phase) and Sertoli cells. **Inhibin A**: dominant follicle, **corpus luteum**, **placenta**.
- Early fall in inhibin B → ↑ FSH in ovarian ageing.
- Inhibin A ↑ in **trisomy 21** (quadruple test) and pre-eclampsia.
- **Granulosa cell tumour** marker: inhibin B (+ AMH).

■ MNEMONIC / MEMORY AID

'B for Beginning' (follicular, inhibin B); **'A for After ovulation'** (luteal, inhibin A). **Inhibin INHIBITS FSH; Activin ACTIVATES FSH**.