PIPADOR REVISION

MRCOG Part 1 Revision Notes

Cell Signalling

BIOCHEMISTRY • LECTURE 205

VISUAL EXAMINATION REVISION NOTES

2026 Edition

205

MRCOG PART 1 REVISION NOTES | BIOCHEMISTRY

Cell Signalling

Endocrine, paracrine, autocrine, juxtacrine and neurocrine signalling — principles and reproductive examples

Endocrine Via blood — distant targets

Paracrine Neighbouring cells

Autocrine Same cell

Receptor Determines specificity and response

■ **KEY FACTS: Learning objectives**

- Define endocrine, paracrine, autocrine, juxtacrine and neurocrine signalling with reproductive examples.
- Describe general principles: ligand–receptor binding, specificity, amplification, termination.
- Relate signalling modes to physiology of the ovary, uterus, placenta and breast.

205.1 Modes of signalling

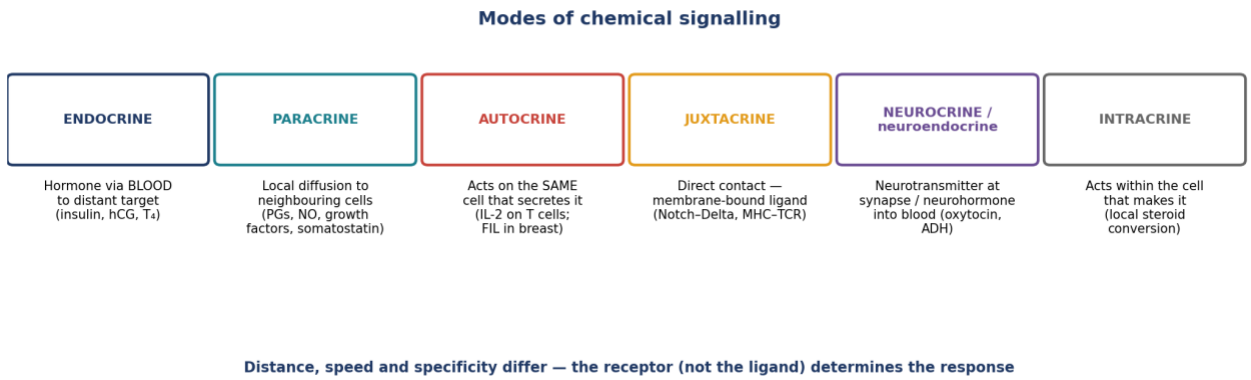


Figure 205.1 — Modes of chemical signalling

Mode	Reproductive examples
Endocrine	FSH/LH (pituitary → ovary), hCG (placenta → corpus luteum), oestradiol (ovary → pituitary/endometrium), hPL, oxytocin (as hormone), insulin
Paracrine	Two-cell system (theca androstenedione → granulosa), oocyte GDF-9/BMP-15 → granulosa, decidual prostaglandins → myometrium, NO and PGI_2 (endothelium → smooth muscle), testosterone (Leydig → Sertoli), somatostatin in islets, ovarian inhibin/activin
Autocrine	Feedback inhibitor of lactation (FIL) in the breast, IL-2 on T cells, growth factors in tumours (autocrine loops), trophoblast hCG
Juxtacrine	Contact-dependent: Notch–Delta, HLA-C on trophoblast ↔ KIR on uNK cells, MHC–TCR, gap junctions
Neurocrine / neuroendocrine	Neurotransmitters at synapses; oxytocin and ADH released from posterior pituitary nerve terminals; Ferguson reflex
Intracrine	Local steroid conversion within a cell (e.g. 17β-HSD, aromatase in breast/endometrial tumours)

205.2 General principles

Principle	Explanation
Specificity	Determined by receptor expression and affinity — the same ligand can produce different effects (adrenaline: β_2 relaxes myometrium, α_1 constricts vessels)
Affinity & saturability	Kd — ligand concentration at 50% occupancy; spare receptors allow maximal effect at partial occupancy
Agonists / antagonists	Full/partial agonists, competitive antagonists (atosiban), inverse agonists; SERMs show tissue-selective agonism (Lecture 170)
Amplification	One hormone → many second-messenger molecules → kinase cascades (cAMP/PKA, MAPK)
Termination	Ligand degradation (PDE, peptidases, MAO/COMT), receptor desensitisation (phosphorylation by GRKs, β-arrestin), internalisation and downregulation (GnRH agonists)
Integration	Cross-talk between pathways; permissive actions (cortisol permits catecholamine vasoconstriction; oestrogen induces PR)

205.3 Signal molecules by solubility

Signal type	Receptor location	Speed / duration	Examples
Hydrophilic (peptides, catecholamines)	Cell surface	Fast (seconds–minutes); short	Insulin, LH, oxytocin, adrenaline
Lipophilic (steroids, thyroid hormone, vitamin D, retinoids)	Intracellular/nuclear	Slow (hours–days); sustained	Oestradiol, progesterone, cortisol, T_3
Gases	Intracellular enzymes	Very fast, very local	Nitric oxide → soluble guanylyl cyclase → cGMP (vasodilatation; GTN; sildenafil)
Eicosanoids	Cell surface GPCRs	Local, rapid inactivation (lungs)	PGE_2, $PGF_2\alpha$, TXA_2, PGI_2, leukotrienes

205.4 Must-know summary

■ EXAM TIP

- Endocrine = blood; paracrine = neighbours; autocrine = self; juxtacrine = contact; neurocrine = synapse/neurohormone.
- Two-cell system, decidual PGs, NO, Leydig → Sertoli testosterone are **paracrine**; FIL is **autocrine**; hCG → CL is **endocrine**.
- Hydrophilic signals → surface receptors (fast); lipophilic → intracellular (slow, sustained); NO → cGMP.
- Termination by degradation, desensitisation and downregulation.

■ MNEMONIC / MEMORY AID

'Endo = Everywhere (blood), Para = Partners, Auto = Ourselves, Juxta = Just touching'.