PharmaRecall™

PHARMACODYNAMICS

Exam Pearls & Rapid Review

HIGH-YIELD SUMMARIES • COMPARISON TABLES • CLINICAL APPLICATIONS

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A concise companion to the Pharmacodynamics Recall Cards and MCQ Practice Pack. Use it for last-minute revision, concept consolidation and exam preparation.

How to Use This Review

Read each pearl as a testable principle. Then explain it aloud, draw the relevant curve or pathway from memory, and use the comparison tables to distinguish commonly confused concepts.

Part 1 — High-Yield Exam Pearls

1. Pharmacodynamics versus pharmacokinetics

Pharmacodynamics describes drug effects and mechanisms; pharmacokinetics describes absorption, distribution, metabolism and excretion. A change in response at the same drug concentration suggests a pharmacodynamic change, although clinical situations may involve both.

2. Affinity is not efficacy

A drug can bind strongly without activating the receptor. Affinity concerns binding; efficacy concerns the ability to produce a response after binding.

3. Potency is not maximal effect

Potency is commonly compared using EC50 in a graded system. Efficacy concerns the maximum response. A lower EC50 does not prove a drug is safer or more efficacious.

4. Full agonists and partial agonists

A full agonist can produce the maximal response in a particular system. A partial agonist has a lower maximum response in that same system, even when receptor occupancy is high.

5. Partial agonists can reduce full-agonist responses

When both compete for the same receptor, the partial agonist may displace a full agonist and reduce the overall response. The size of the effect depends on concentrations, affinities and the biological system.

6. Neutral antagonists versus inverse agonists

A neutral antagonist blocks agonist binding without changing constitutive receptor activity. An inverse agonist reduces constitutive activity where it exists.

7. Competitive antagonism

A reversible competitive antagonist usually shifts the agonist curve rightward and preserves Emax when enough agonist can overcome the blockade. The agonist's apparent potency decreases.

8. Noncompetitive antagonism

A noncompetitive antagonist commonly lowers Emax because increasing agonist concentration cannot fully restore the original maximal response. Real curves depend on the precise mechanism and system.

9. Graded versus quantal responses

Graded curves measure response magnitude in a tissue or system. Quantal curves measure the proportion of a population meeting a defined all-or-none endpoint.

10. EC50 and ED50 need context

EC50 is the concentration producing half-maximal response in a specified graded system. In quantal population analysis, ED50 is the dose effective in 50% of the population for a defined endpoint; terminology can vary across texts.

11. Receptor reserve

A maximal response may occur without occupation of all receptors because downstream signalling can amplify receptor activation. This helps explain why occupancy and response are not always proportional.

12. Therapeutic index is not a guarantee of safety

TD50/ED50 is a common experimental expression; LD50/ED50 may be used in suitable animal studies. A ratio does not capture every adverse effect, interaction or individual risk.

13. Therapeutic window

The therapeutic window is the range of exposure associated with useful benefit without unacceptable toxicity. It is related to, but not identical to, a therapeutic-index ratio.

14. Ceiling effect

Increasing dose may eventually produce little or no additional benefit for a particular endpoint. Other effects, including adverse effects, may still increase.

15. GPCRs use multiple signalling routes

Gs commonly stimulates adenylyl cyclase and raises cAMP; Gi commonly inhibits adenylyl cyclase; Gq activates phospholipase C, generating IP3 and DAG.

16. Second messengers amplify signals

cAMP, cGMP, IP3, DAG and intracellular calcium relay signals inside cells. They activate downstream proteins and can produce effects far beyond the initial ligand–receptor interaction.

17. Enzyme-linked and intracellular receptors

Receptor tyrosine kinases commonly signal through phosphorylation networks such as MAPK and PI3K–Akt. Intracellular steroid receptors can regulate gene transcription, often with slower-onset effects.

18. Desensitisation is not the same as downregulation

Desensitisation means reduced responsiveness; downregulation means reduced receptor number or availability. Internalisation can contribute to either transient loss from the surface or receptor degradation.

19. Tachyphylaxis and tolerance

Tachyphylaxis is a rapid decrease in response after repeated dosing over a short period. Tolerance is a broader reduction in response with continued exposure and may be pharmacodynamic or pharmacokinetic.

20. Drug interactions can be pharmacodynamic

Two drugs may add to, enhance or oppose each other's effects without changing either drug's concentration. Always consider the combined physiological effect as well as pharmacokinetic interactions.

Part 2 — Comparison Tables

Concept	Definition / meaning	Exam distinction
Affinity	Tendency of a drug to bind a target	Does not guarantee activation
Efficacy	Ability to produce a response	Related to maximum effect in a system
Potency	Amount or concentration needed for a specified effect	Lower EC50 usually indicates greater potency
Full agonist	Can produce the maximal response in a system	High efficacy in that system
Partial agonist	Produces a lower maximum response than a full agonist in the same system	Can oppose a full agonist when competing
Neutral antagonist	Blocks agonist action without changing constitutive activity	No intrinsic agonist or inverse-agonist effect
Inverse agonist	Reduces constitutive receptor activity	Requires a receptor system with baseline activity

Mechanism / term	Typical dose–response effect	Important qualification
Reversible competitive antagonism	Rightward shift; Emax preserved	Assumes surmountable antagonism and suitable conditions
Noncompetitive antagonism	Emax often reduced	Exact effect depends on mechanism and system
Receptor reserve	Maximal response before all receptors are occupied	May increase apparent sensitivity
Desensitisation	Response falls despite continued/repeated stimulation	Can occur without a decrease in receptor number
Downregulation	Receptor number or availability decreases	Can coexist with desensitisation
Tachyphylaxis	Rapid decline in response after repeated dosing	Mechanism may include mediator depletion or signalling adaptation

Part 3 — Signal Transduction at a Glance

Pathway	Key sequence	Typical downstream consequence
Gs	GPCR → Gs → adenylyl cyclase → ↑ cAMP	cAMP-dependent signalling, often including PKA activation
Gi	GPCR → Gi → inhibition of adenylyl cyclase → ↓ cAMP	Reduced cAMP signalling; other G-protein effects may occur
Gq	GPCR → Gq → PLC-β → IP3 + DAG	IP3 promotes Ca²■ release; DAG contributes to PKC activation
Receptor tyrosine kinase	Ligand binding → receptor activation/phosphorylation → signalling proteins	Pathways may include MAPK and PI3K–Akt
Intracellular steroid receptor	Lipophilic ligand enters cell → receptor binding → transcriptional regulation	Often slower onset and longer-lasting gene-expression effects

Part 4 — Clinical and Examination Scenarios

Scenario 1: Curve shifts right, Emax remains unchanged.

A reversible competitive antagonist is one possible explanation. The agonist needs a higher concentration to achieve a given response, but sufficient agonist can restore the maximum in the simple model.

Scenario 2: The maximum response is lower despite increasing agonist concentration.

Consider noncompetitive antagonism, irreversible receptor blockade or another limitation in the response system. Do not identify the mechanism from Emax alone without context.

Scenario 3: A drug has a lower EC50 but the same Emax as another drug.

The first drug is more potent in that tested system, but not necessarily more efficacious or safer.

Scenario 4: A partial agonist is given to a patient already taking a full agonist at the same receptor.

The partial agonist may reduce the overall response by competing for receptor occupancy. The clinical effect depends on the drugs, doses, receptor system and patient context.

Scenario 5: The response to repeated doses falls rapidly.

Consider tachyphylaxis, desensitisation or depletion of a required mediator. Also check administration conditions and whether exposure has changed.

Scenario 6: Two drugs produce greater sedation when used together, without changing plasma concentrations.

This is consistent with a pharmacodynamic interaction. The combined effect may increase harm even if pharmacokinetics are unchanged.

Part 5 — One-Page Rapid Review Checklist

- ■ Can I distinguish affinity, efficacy and potency?
- ■ Can I explain full agonism, partial agonism, neutral antagonism and inverse agonism?
- ■ Can I predict the typical effect of reversible competitive versus noncompetitive antagonism?
- ■ Can I distinguish graded from quantal dose–response curves?
- ■ Can I define EC50, ED50, Emax, therapeutic index and therapeutic window?
- ■ Can I explain receptor reserve and why occupancy does not always equal response?
- ■ Can I outline Gs, Gi and Gq signalling and name common second messengers?
- ■ Can I distinguish desensitisation, downregulation, tolerance and tachyphylaxis?
- ■ Can I recognise pharmacodynamic interactions and factors that modify individual response?

Educational note: This material is designed for revision and examination preparation. Concepts are simplified for rapid recall; refer to standard pharmacology texts, course materials and relevant clinical guidance for fuller context.

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