PharmaRecall™

PHARMACODYNAMICS

Rapid Revision Series • Volume 1

ACTIVE-RECALL CARDS & HIGH-YIELD REVIEW

Prepared by Dr Araromi Ebisola Jonathan Vincent

Pharmacology Tutor • Medical Content Writer

A concise, exam-oriented revision resource for medical, pharmacy, nursing, physiology and other health-science students. Attempt each question before checking the answer.

How to Use This Pack

Cover the answer column and attempt each question from memory. Mark cards you miss and revisit them during later revision sessions. These cards support, but do not replace, lectures or a full pharmacology text.

Learning Objectives

• Explain drug–receptor interactions and receptor theories.

• Differentiate full agonists, partial agonists, inverse agonists and antagonists.

• Interpret dose–response curves, potency, efficacy and therapeutic index.

• Describe receptor families, signal transduction and receptor regulation.

• Explain receptor reserve, desensitisation and tachyphylaxis.

A. Fundamental Concepts

QUESTION	ANSWER
1. Define pharmacodynamics.	The study of what a drug does to the body, including its biochemical and physiological effects and mechanisms of action.
2. Distinguish pharmacodynamics from pharmacokinetics.	Pharmacodynamics: what the drug does to the body. Pharmacokinetics: what the body does to the drug—absorption, distribution, metabolism and excretion.
3. What is a drug receptor?	A cellular macromolecule, commonly a protein, with which a drug interacts to initiate, modify or inhibit a biological response.
4. Differentiate affinity from efficacy.	Affinity is the tendency of a drug to bind to a receptor. Efficacy is the ability of the bound drug to produce a response.
5. What is intrinsic activity?	The capacity of a drug–receptor complex to activate the receptor and produce a response. Full agonists have high activity, partial agonists lower activity, and neutral antagonists none in the classical model.
6. State the central idea of receptor-occupancy theory.	Drug response is related to the proportion of receptors occupied. Receptor reserve and signal amplification mean occupancy and response are not always directly proportional.

B. Agonists and Antagonists

QUESTION	ANSWER
7. What is a full agonist? Give an example.	A drug that activates a receptor and can produce the maximal response in a given system. Morphine is an example of a μ-opioid receptor agonist.
8. What is a partial agonist? Give an example.	A drug that activates a receptor but produces a lower maximal response than a full agonist in the same system. Buprenorphine is a partial μ-opioid receptor agonist.
9. How can a partial agonist act as an antagonist?	It competes with a full agonist for receptor binding but produces less activation, reducing the overall response.
10. Define an inverse agonist.	A ligand that reduces constitutive baseline receptor activity, producing an effect opposite to that of an agonist.
11. What is a neutral competitive antagonist?	A drug that competes for the agonist binding site but has no intrinsic activity; it reduces agonist action.
12. Describe reversible competitive antagonism on a graded dose–response curve.	It usually shifts the agonist curve rightward, increasing the concentration needed for a given effect, while Emax is preserved if antagonism is surmountable.

QUESTION	ANSWER
13. What is noncompetitive antagonism?	Antagonism that cannot be fully overcome simply by increasing agonist concentration; Emax commonly decreases.
14. What is physiological antagonism? Give an example.	Two agents produce opposing effects through different mechanisms. Adrenaline can oppose histamine-induced bronchoconstriction through β■-adrenoceptor-mediated bronchodilation.

C. Dose–Response Relationships

QUESTION	ANSWER
15. What is a graded dose–response curve?	A graph showing the magnitude of response in a tissue or system as drug dose or concentration increases.
16. What is a quantal dose–response curve?	A graph showing the proportion of a population that exhibits a specified all-or-none response at different doses.
17. Define EC50.	The concentration producing 50% of the maximal response in a specified experimental system; commonly used to compare potency under comparable conditions.
18. Why must ED50 be interpreted in context?	In a quantal population curve, ED50 is effective in 50% of the population. In some graded-response contexts, it means the dose producing 50% of maximal effect.
19. Define Emax.	The maximum response a drug can produce in a particular biological system under the conditions studied; commonly used as a measure of efficacy.
20. How is potency commonly compared?	Under comparable conditions, a drug with a lower EC50 or ED50 is generally more potent because less drug is needed for the specified effect.
21. What happens when a reversible competitive antagonist is added to an agonist?	The curve usually shifts rightward; apparent potency decreases, while Emax is maintained if sufficient agonist can overcome antagonism.
22. What is the typical effect of a noncompetitive antagonist on Emax?	Emax commonly decreases because increasing agonist concentration cannot fully restore the original maximal response.
23. What is receptor reserve?	A situation in which maximal response can occur without occupancy of all available receptors, often due to efficient signal amplification.
24. Define therapeutic index.	A ratio comparing a toxic dose to an effective dose. Common expressions include TD50/ED50 or, in suitable animal studies, LD50/ED50. A wider ratio suggests a greater separation between toxicity and efficacy but does not guarantee safety.

D. Receptors and Signal Transduction

QUESTION	ANSWER
25. Name four major receptor families.	Ligand-gated ion channels; G-protein-coupled receptors (GPCRs); enzyme-linked or enzyme-associated receptors; and intracellular receptors that regulate gene transcription.
26. Summarise the basic GPCR signalling sequence.	Ligand binds GPCR → receptor activates a heterotrimeric G protein → GDP is exchanged for GTP on the α-subunit → G-protein components regulate an effector → second messengers and downstream effectors alter cell function. GTP hydrolysis helps terminate signalling.
27. Name common second messengers.	cAMP, cGMP, IP■, DAG and intracellular Ca^{2}■.

QUESTION	ANSWER
28. What is receptor desensitisation?	Reduced receptor responsiveness after continued or repeated agonist exposure, potentially involving phosphorylation, uncoupling or internalisation.
29. Differentiate receptor upregulation from downregulation.	Upregulation is an increase in receptor number or availability; downregulation is a decrease. Chronic blockade or persistent stimulation can sometimes produce these adaptations, respectively.
30. Define tachyphylaxis.	A rapid reduction in response to repeated doses over a short period, potentially due to mediator depletion, receptor desensitisation or other rapid adaptations.

High-Yield Comparison Table

Term	Meaning	Exam distinction
Affinity	Tendency to bind to receptor	Binding does not guarantee response
Efficacy	Ability to produce a response	Related to maximal effect in a system
Potency	Amount/concentration needed for an effect	Lower EC50 usually means greater potency
Full agonist	Can produce maximal response in system	High efficacy
Partial agonist	Lower maximal response than a full agonist in same system	Can oppose a full agonist when competing
Neutral antagonist	Binds without changing constitutive activity	Blocks agonist; no intrinsic activity
Inverse agonist	Reduces constitutive receptor activity	Opposite direction to agonist effect
Competitive antagonist	Competes at agonist binding site	Usually surmountable; rightward shift
Noncompetitive antagonist	Response not fully restored by more agonist	Often lowers Emax
Desensitisation	Reduced receptor responsiveness	May occur despite continued exposure
Downregulation	Reduced receptor number/availability	Change in receptor availability
Tachyphylaxis	Rapid decline after repeated dosing	Rapid tolerance-like phenomenon

Exam Pearls

• **1. Potency is not efficacy.** Potency concerns how much drug is needed; efficacy concerns the response the drug can produce.

• **2. A partial agonist can behave as an antagonist.** It may occupy receptors while producing less activation than a full agonist.

• **3. Competitive antagonism is usually surmountable.** In the simple reversible model, enough agonist can restore Emax.

• **4. Occupancy and response are not always proportional.** Receptor reserve and signal amplification can permit maximal response without full occupancy.

• **5. A wide therapeutic index does not mean zero risk.** Patient variability, interactions, organ dysfunction and clinical context still matter.

Rapid Review Checklist

■ I can distinguish pharmacodynamics from pharmacokinetics.

■ I can explain affinity, efficacy, potency, EC50, Emax and ED50.

■ I can distinguish full agonists, partial agonists, inverse agonists and neutral antagonists.

■ I can predict typical competitive and noncompetitive antagonist effects on dose–response curves.

■ I can name major receptor families and common second messengers.

■ I can explain receptor reserve, desensitisation, upregulation, downregulation and tachyphylaxis.

Educational note: This revision pack is intended for learning and examination preparation. Some descriptions are simplified for rapid recall; consult prescribed textbooks and course materials for fuller context.

PharmaRecall™ • Pharmacodynamics Rapid Revision Series • Volume 1