PharmaRecall™

PHARMACODYNAMICS

MCQ Practice Pack — Volume 2

Advanced and Case-Based Questions

30 single-best-answer questions designed to strengthen application of receptor theory, dose–response relationships, signal transduction, antagonism, tolerance, and clinical pharmacological reasoning.

Author: Dr Araromi Ebisola Jonathan Vincent
Medical Doctor • Pharmacology Educator

Study method: answer each question before checking the answer key and explanation. Focus on why the correct option fits and why the alternatives do not.

SECTION 1 — QUESTIONS

Choose the single best answer for each question. Answers and explanations are provided in Section 2.

1. A patient's dose–response curve for Drug A shifts to the right after Drug B is added, but the maximum response to Drug A can still be achieved by increasing its concentration. Which best describes Drug B's action?

A. Irreversible antagonism
B. Competitive antagonism
C. Non-competitive antagonism
D. Partial agonism

2. A drug produces 60% of the maximum response even when all available receptors are occupied. What is the most appropriate description?

A. Full agonist
B. Neutral antagonist
C. Partial agonist
D. Inverse agonist

3. Drug X has a lower EC50 than Drug Y in the same tissue, and both reach the same Emax. Which conclusion is justified?

A. Drug X has greater potency in that system
B. Drug X has greater efficacy
C. Drug Y is necessarily safer
D. Drug X has a higher therapeutic index

4. A receptor antagonist reduces the response to an agonist, and increasing agonist concentration cannot restore the original maximum. Which pattern is most consistent?

A. Competitive antagonism
B. Non-competitive antagonism
C. Physiological antagonism only
D. Enzyme induction

5. A patient taking a long-term agonist develops a reduced response. Receptor numbers on the cell surface are found to be decreased. This is best called:

A. Up-regulation
B. Supersensitivity
C. Down-regulation
D. Chemical antagonism

6. A drug binds to a receptor and decreases its constitutive activity below baseline. It is best classified as:

A. Neutral antagonist
B. Inverse agonist
C. Partial agonist
D. Full agonist

7. Two drugs have the same ED50, but Drug A has a lower TD50 than Drug B. Which statement is supported?

A. Drug A has a narrower therapeutic index
B. Drug A is more potent and therefore safer
C. Drug B has lower efficacy
D. Both drugs have identical safety margins

8. In an isolated tissue, a low concentration of agonist produces a near-maximal response even though only a fraction of receptors are occupied. This suggests:

A. Spare receptors
B. Absence of receptors
C. Zero intrinsic efficacy
D. Irreversible antagonism

9. A ligand activates a receptor-linked G protein, which stimulates adenylyl cyclase. Which second messenger increases?

A. IP3 only
B. cAMP
C. DAG only
D. cGMP exclusively

10. Activation of a Gq-coupled receptor most directly stimulates which effector enzyme?

A. Adenylyl cyclase
B. Phospholipase C
C. Guanylyl cyclase only
D. Tyrosine hydroxylase

11. A drug's response depends on a receptor tyrosine kinase that dimerises and autophosphorylates after ligand binding. Which mechanism is involved?

A. Direct opening of a ligand-gated chloride channel
B. Receptor tyrosine kinase signalling
C. Gs-mediated cAMP formation exclusively
D. Nuclear receptor binding only

12. A medicine binds an intracellular receptor and alters gene transcription. Compared with a ligand-gated ion channel, its onset is often slower because it:

A. Must always be metabolised first
B. Alters transcription and protein synthesis
C. Cannot enter cells
D. Has no receptor-mediated action

13. A patient receives a drug that blocks beta-adrenoceptors. Adrenaline's effect on heart rate is reduced. This is an example of:

A. Pharmacokinetic antagonism
B. Receptor-level pharmacological antagonism
C. Chemical inactivation in plasma
D. Enzyme replacement

14. A drug has a steep dose–response curve near its therapeutic range. Small dose increases produce large changes in effect. What is the practical implication?

A. Dose adjustments may need particular care
B. The drug must have low potency
C. The drug cannot cause toxicity
D. Its Emax is necessarily low

15. A patient's response to the same dose differs substantially from that of other patients because of a receptor gene variant. This is an example of:

A. Pharmacodynamic variability
B. First-pass metabolism only
C. Renal clearance only
D. Pharmaceutical incompatibility

16. A drug produces a response by binding to a receptor but has lower intrinsic activity than the endogenous agonist. In the presence of the endogenous agonist, it may reduce the overall response. Why?

A. It is a partial agonist competing for receptors
B. It is necessarily an irreversible antagonist
C. It increases receptor synthesis immediately
D. It prevents all ligand binding

17. After repeated administration, a drug's effect diminishes rapidly over a short period. Which term most specifically describes this pattern?

A. Tachyphylaxis
B. Idiosyncrasy
C. Potentiation
D. Summation

18. Two drugs acting on different receptors produce opposing physiological effects. For example, one increases heart rate and the other decreases it. This is:

A. Chemical antagonism
B. Physiological antagonism
C. Competitive antagonism at one receptor
D. Partial agonism

19. A chelating agent binds a toxic metal and forms a complex that is excreted. The interaction is best described as:

A. Receptor antagonism
B. Chemical antagonism
C. Inverse agonism
D. Receptor down-regulation

20. A drug's EC50 is 10 micromol/L in one tissue and 1 micromol/L in another. What is the safest interpretation?

A. It is always ten times more efficacious in the second tissue
B. Apparent potency differs between the tested systems
C. Its therapeutic index is ten in both tissues
D. It must bind a different receptor in each tissue

21. A patient develops bronchoconstriction after a drug activates a receptor that increases intracellular IP3 and calcium. Which G protein pathway is most likely involved?

A. Gi
B. Gs
C. Gq
D. G12 only

22. A receptor blocker is present, but it has no effect on baseline signalling and prevents an agonist from binding. Which description fits best?

A. Inverse agonist
B. Neutral antagonist
C. Full agonist
D. Partial agonist

23. A drug binds allosterically and changes the receptor's response to the endogenous ligand without occupying the ligand's main binding site. This is:

A. Allosteric modulation
B. Competitive antagonism by definition
C. Covalent enzyme inhibition
D. Receptor internalisation only

24. A patient taking a chronic antagonist develops increased receptor expression. After abrupt withdrawal, the agonist produces an exaggerated response. This is most consistent with:

A. Receptor up-regulation and supersensitivity
B. Receptor down-regulation
C. Chemical antagonism
D. Reduced receptor affinity in all tissues

25. A drug acts on an enzyme-linked receptor and triggers a kinase cascade. Which statement about signal amplification is correct?

A. One activated receptor can initiate multiple downstream events
B. Amplification occurs only with nuclear receptors
C. Every signalling step consumes one receptor permanently
D. Amplification makes dose irrelevant

26. An agonist has high affinity but produces little receptor activation. Which property is low?

A. Affinity
B. Intrinsic efficacy
C. Receptor occupancy at any concentration
D. Chemical stability necessarily

27. A drug's effect is mediated by opening an ion channel within milliseconds of ligand binding. Which receptor class is most likely?

A. Nuclear hormone receptor
B. Ligand-gated ion channel
C. Receptor tyrosine kinase
D. Cytokine receptor requiring gene transcription

28. A dose produces the desired therapeutic effect in 50% of a population. This dose is termed the:

A. TD50
B. ED50
C. LD50
D. Emax

29. A drug's maximum achievable effect is reduced after an irreversible antagonist is introduced. Increasing agonist concentration does not restore Emax, at least over the tested range. Why?

A. Some functional receptors are unavailable and receptor reserve is insufficient to compensate
B. The agonist has become more potent
C. The antagonist increases intrinsic efficacy
D. EC50 and Emax are synonymous

30. A clinician compares two drugs and finds Drug A has a larger Emax but a higher EC50 than Drug B. Which statement is correct?

A. Drug A is less potent but has greater maximal efficacy in that system
B. Drug A is more potent and less efficacious
C. Drug B must be safer
D. The drugs are pharmacologically identical

SECTION 2 — ANSWER KEY & EXPLANATIONS

Question	Answer
1	B
2	C
3	A
4	B
5	C
6	B
7	A
8	A
9	B
10	B
11	B
12	B
13	B
14	A
15	A
16	A
17	A
18	B
19	B
20	B
21	C
22	B
23	A
24	A
25	A
26	B
27	B
28	B
29	A
30	A

1. Answer: B — A reversible competitive antagonist competes at the same receptor site and typically produces a parallel rightward shift with no reduction in Emax when sufficient agonist is available.

2. Answer: C — A partial agonist has lower intrinsic efficacy than a full agonist and produces a submaximal response even at full receptor occupancy.

3. Answer: A — A lower EC50 indicates greater potency under the stated experimental conditions. Equal Emax means the observed maximal efficacy is the same; safety cannot be inferred from EC50 alone.

4. Answer: B — Non-competitive antagonism, including irreversible receptor blockade or interference with downstream signalling, can reduce the attainable Emax.

5. Answer: C — Prolonged agonist exposure can cause receptor desensitisation and/or down-regulation, contributing to tolerance.

6. Answer: B — An inverse agonist preferentially stabilises an inactive receptor state and reduces constitutive activity. A neutral antagonist blocks agonist binding without changing basal activity.

7. Answer: A — Therapeutic index is commonly expressed as TD50/ED50. With the same ED50, a lower TD50 gives Drug A a lower therapeutic index, indicating a narrower margin in this simplified comparison.

8. Answer: A — Spare receptors exist when maximal response can occur without occupation of all receptors, often because receptor-effector coupling amplifies the signal.

9. Answer: B — Gs activation stimulates adenylyl cyclase, increasing intracellular cAMP and activating protein kinase A.

10. Answer: B — Gq activates phospholipase C-beta, which cleaves PIP2 into IP3 and DAG. IP3 promotes calcium release; DAG contributes to protein kinase C activation.

11. Answer: B — Many growth factors and insulin signal through receptor tyrosine kinases, which dimerise or rearrange and autophosphorylate to recruit downstream signalling proteins.

12. Answer: B — Intracellular and nuclear receptor effects often involve altered gene expression and protein synthesis, generally producing slower onset and longer-lasting effects.

13. Answer: B — A beta-adrenoceptor blocker opposes adrenaline at the receptor level. This is pharmacodynamic antagonism rather than altered absorption or metabolism.

14. Answer: A — A steep curve means small dose changes can produce large response changes in that range. It does not, by itself, establish potency, efficacy, or safety.

15. Answer: A — Genetic variation in receptor or downstream signalling proteins can alter sensitivity and is a source of pharmacodynamic variability.

16. Answer: A — A partial agonist can occupy receptors while generating a smaller response than a full agonist, thereby reducing the response when it displaces the full agonist.

17. Answer: A — Tachyphylaxis is a rapid decrease in response after repeated doses, often due to mediator depletion, receptor desensitisation, or related adaptive processes.

18. Answer: B — Physiological antagonists act through different receptors or pathways to produce opposing effects on the same physiological variable.

19. Answer: B — Chemical antagonism occurs when one substance chemically interacts with another, reducing its biological activity, as with chelation of certain metals.

20. Answer: B — EC50 is system-dependent and can vary with receptor density, coupling efficiency, tissue context, and assay conditions. It does not directly establish efficacy or therapeutic index.

21. Answer: C — Gq-coupled receptors activate phospholipase C, generating IP3, which releases calcium from intracellular stores. The physiological effect depends on the tissue and receptor.

22. Answer: B — A neutral antagonist blocks agonist action but does not alter constitutive receptor activity on its own.

23. Answer: A — Allosteric modulators bind at a distinct site and can alter ligand affinity, efficacy, or both. They may be positive, negative, or neutral modulators.

24. Answer: A — Chronic receptor blockade can cause compensatory up-regulation or increased responsiveness. Removing the antagonist may reveal an exaggerated response.

25. Answer: A — Signalling cascades can amplify a signal because activated enzymes or second messengers can influence many downstream molecules. Dose still matters.

26. Answer: B — Affinity describes binding strength; efficacy describes the ability of a bound ligand to activate the receptor and produce a response.

27. Answer: B — Ligand-gated ion channels directly alter ion conductance and can generate rapid responses, often within milliseconds.

28. Answer: B — In a quantal dose-response population, ED50 is the dose that produces the specified therapeutic effect in 50% of the population. It differs from EC50, which is a concentration measure in a graded response system.

29. Answer: A — Irreversible antagonism can reduce the pool of functional receptors. If spare receptors are insufficient, maximal response falls and cannot be restored simply by adding more agonist.

30. Answer: A — A higher EC50 indicates lower potency in the tested system, while a larger Emax indicates greater maximal efficacy. These are distinct pharmacological properties.

RAPID REVIEW — HIGH-YIELD DISTINCTIONS

Concept	Core distinction
Potency	Often reflected by EC50 in a graded curve; lower EC50 generally means greater potency in the same system.
Efficacy	Ability to produce a response; Emax reflects the maximum response achievable in the tested system.
Competitive antagonist	Usually shifts the agonist curve right; Emax can be restored with sufficient agonist.
Non-competitive / irreversible antagonism	Can reduce Emax when functional receptor capacity becomes limiting.
Partial agonist	Lower intrinsic efficacy than a full agonist; may oppose a full agonist by competing for receptors.
Inverse agonist	Reduces constitutive receptor activity; a neutral antagonist does not change basal activity.
Gs / Gi / Gq	Gs increases cAMP; Gi generally inhibits adenylyl cyclase; Gq activates PLC → IP3/DAG.

Concept	Core distinction
ED50 / EC50 / TD50	ED50: median effective dose in a population; EC50: concentration for 50% of a graded maximum; TD50: toxic dose in 50% of a population.

Educational resource for revision. Clinical decisions should be based on current guidance, patient-specific factors, and appropriate professional judgement.