INI CET Pharmacology

Sample Paper – 5

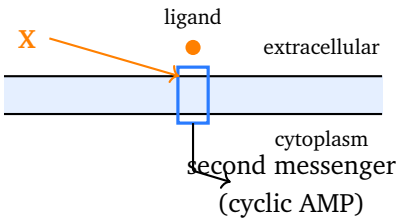
Duration: 14 Minutes **Maximum Marks: 15**

Instructions

- This paper contains **15** Multiple Choice Questions (Single Best Response), modelled on the Pharmacology content of **INI CET** (Institutes of National Importance Combined Entrance Test) conducted by AIIMS New Delhi.
- The **15-question** length matches the number of Pharmacology items you actually meet in the real 200-question paper.
- Each correct answer carries **+1 mark**. There is **negative marking of one-third (0.33) mark** for every incorrect answer; unattempted questions carry no penalty.
- Every question has **exactly one best answer**. Choose the single most appropriate option.
- Attempt this practice paper in one timed sitting of about **14 minutes**, the pace the real computer-based test demands.

General Pharmacology

Q1. In the schematic of transmembrane signalling shown, the receptor marked **X** spans the membrane, is activated by an extracellular ligand, and works through a coupled G protein to raise an intracellular second messenger. This receptor class is:



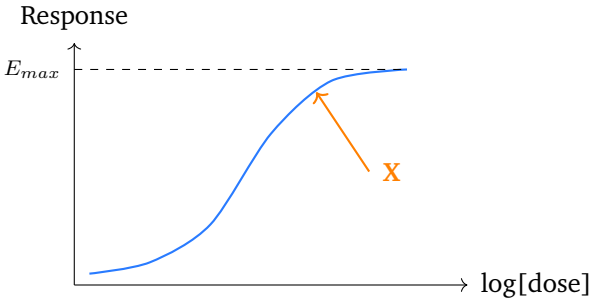
(A) G protein-coupled receptor

(B) Ligand-gated ion channel

(C) Receptor tyrosine kinase

(D) Intracellular nuclear receptor

Q2. In the log dose-response curve shown, the response reaches its ceiling (marked **X**) while a fraction of receptors is still unoccupied, so a maximal effect is produced without full receptor occupancy. This phenomenon is called:



(A) Spare (reserve) receptors

(B) Receptor down-regulation

(C) Tachyphylaxis

(D) Competitive antagonism

Q3. Which of the following ligands acts through an intracellular (nuclear) receptor to regulate gene transcription rather than through a cell-surface receptor?

(A) Adrenaline

(B) Insulin

(C) Cortisol (a glucocorticoid)

(D) Acetylcholine

Autonomic Nervous System

Q4. Which beta-adrenergic blocker is beta-1 selective (cardioselective) and available as an ultra-short-acting intravenous agent, making it useful for acute perioperative tachycardia?

(A) Propranolol

(B) Esmolol

(C) Timolol

(D) Nadolol

Q5. A non-selective beta-blocker is relatively contraindicated in a patient with bronchial asthma because beta-2 blockade can precipitate bronchospasm. Which of the following is the non-selective agent?

(A) Propranolol

(B) Metoprolol

(C) Atenolol

(D) Bisoprolol

Cardiovascular and Renal Pharmacology

Q6. In the Vaughan-Williams classification, which drug is the prototype Class III antiarrhythmic, acting mainly by blocking potassium channels and prolonging repolarisation?

(A) Lidocaine

(B) Verapamil

(C) Metoprolol

(D) Amiodarone

Q7. Lidocaine, given intravenously for ventricular arrhythmias, belongs to which Vaughan-Williams class?

(A) Class III (potassium channel blocker)

(B) Class II (beta-blocker)

(C) Class IB (sodium channel blocker)

(D) Class IV (calcium channel blocker)

Q8. Long-term amiodarone therapy is most characteristically associated with which serious adverse effect?

(A) Pulmonary fibrosis

(B) Gingival hyperplasia

(C) Dry cough from bradykinin accumulation

(D) Cyanide toxicity

Central Nervous System Pharmacology

Q9. Typical antipsychotics such as haloperidol act mainly by blocking which receptor, an action that also underlies their extrapyramidal side effects?

(A) 5-HT2A serotonin receptor

(B) Muscarinic acetylcholine receptor

(C) Histamine H1 receptor

(D) Dopamine D2 receptor

Q10. Which atypical antipsychotic is most strongly associated with agranulocytosis, requiring regular white cell count monitoring?

(A) Haloperidol

(B) Risperidone

(C) Olanzapine

(D) Clozapine

Chemotherapy

Q11. Amphotericin B exerts its antifungal action by binding to which fungal cell membrane component and forming pores that leak intracellular ions?

(A) Beta-1,3-glucan

(B) Ergosterol

(C) Chitin

(D) Cholesterol

Q12. The most important dose-limiting toxicity of amphotericin B, requiring monitoring of renal function and electrolytes, is:

(A) Hepatotoxicity

(B) Ototoxicity

(C) Nephrotoxicity

(D) Pulmonary fibrosis

Q13. Which antifungal is the drug of choice for maintenance and step-down therapy of cryptococcal meningitis and works by inhibiting fungal cytochrome P450 (14-alpha-demethylase)?

(A) Nystatin

(B) Fluconazole

(C) Griseofulvin

(D) Terbinafine

Autacoids, Endocrine and Miscellaneous

Q14. Metformin, the first-line oral drug for type 2 diabetes mellitus, lowers blood glucose mainly by which mechanism?

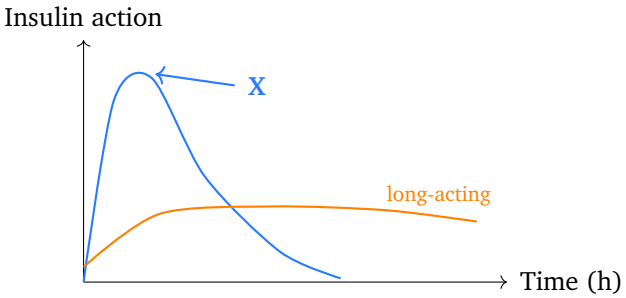
(A) Stimulating insulin release from pancreatic beta cells

(B) Reducing hepatic gluconeogenesis and improving insulin sensitivity

(C) Blocking intestinal alpha-glucosidase enzymes

(D) Increasing renal glucose excretion

Q15. The insulin action-time profiles shown compare a rapid-acting and a long-acting preparation. The curve marked **X** has the fastest onset and shortest duration. It corresponds to which insulin?



(A) Insulin glargine

(B) NPH (isophane) insulin

(C) Insulin lispro

(D) Protamine zinc insulin

Detailed Solutions

Q1.

Solution

Concept – receptor superfamilies: Drug receptors fall into four broad classes, each with its own transduction route.

Step 1 – read the schematic: X spans the membrane and, on binding its ligand, engages a G protein on the cytoplasmic side.

Step 2 – link to the messenger: The G protein activates an effector (e.g. adenylyl cyclase) that raises a second messenger such as cyclic AMP. This defines a **G protein-coupled receptor**.

Why the other options are wrong:

- B, ligand-gated ion channel: opens a pore directly (milliseconds), no G protein or second messenger.
- C, receptor tyrosine kinase: has intrinsic enzyme activity and autophosphorylates (e.g. insulin receptor).
- D, nuclear receptor: is intracellular and alters gene transcription, not a surface G-protein-linked receptor.

Final Answer: G protein-coupled receptor $\Rightarrow$ A

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – spare receptors: A tissue can reach its maximal response before every receptor is occupied.

Step 1 – read the curve: The response plateaus at X (E_{max}) even though occupancy is sub-maximal.

Step 2 – name it: Receptors not needed for the maximal effect are **spare (reserve) receptors**; they increase agonist sensitivity by shifting the dose-response curve leftward.

Why the other options are wrong:

- B, down-regulation: a fall in receptor number after prolonged stimulation, which lowers response.

- C, tachyphylaxis: rapidly declining response on repeated dosing, not a ceiling at low occupancy.
- D, competitive antagonism: shifts the curve to the right, it does not describe a receptor reserve.

Final Answer: Spare (reserve) receptors $\Rightarrow$ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – lipophilic ligands and nuclear receptors: Steroid and thyroid hormones cross the membrane and act inside the cell.

Step 1 – pick the lipophilic ligand: Cortisol, a glucocorticoid, is lipid-soluble and binds an intracellular (nuclear) receptor.

Step 2 – mechanism: The hormone-receptor complex acts as a transcription factor, switching target genes on or off over hours.

Why the other options are wrong:

- A, adrenaline: acts on cell-surface adrenergic (G protein-coupled) receptors.
- B, insulin: acts on a surface receptor tyrosine kinase.
- D, acetylcholine: acts on surface nicotinic (ion channel) and muscarinic (G-protein) receptors.

Final Answer: Cortisol (a glucocorticoid) $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – properties of beta-blockers: They differ in receptor selectivity and duration of action.

Step 1 – match the description: An ultra-short-acting, beta-1 selective, intravenous agent is **esmolol**; it is hydrolysed by red-cell esterases, giving a half-life of only a few minutes.

Step 2 – clinical use: This makes it ideal for titratable rate control of acute perioperative or emergency tachycardia.

Why the other options are wrong:

- A, propranolol: non-selective and longer-acting.
- C, timolol: non-selective, mainly used topically for glaucoma.
- D, nadolol: non-selective with a long duration of action.

Final Answer: Esmolol $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – selectivity and bronchospasm: Blocking beta-2 receptors on bronchial smooth muscle can trigger bronchoconstriction.

Step 1 – find the non-selective drug: Propranolol blocks both beta-1 and beta-2 receptors, so it is relatively contraindicated in asthma.

Step 2 – contrast: Cardioselective agents blunt but do not abolish this risk, and are preferred if a beta-blocker is essential.

Why the other options are wrong:

- B, metoprolol: beta-1 selective.
- C, atenolol: beta-1 selective.
- D, bisoprolol: beta-1 selective (the most cardioselective of these).

Final Answer: Propranolol $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – Vaughan-Williams classes: Class I blocks sodium, II beta-adrenergic, III potassium, IV calcium channels.

Step 1 – identify the Class III prototype: Amiodarone predominantly blocks potassium channels, prolonging repolarisation and the action potential duration (it also has some effect in every class).

Step 2 – effect: This lengthens the QT interval and refractory period, suppressing re-entry.

Why the other options are wrong:

- A, lidocaine: Class IB sodium channel blocker.
- B, verapamil: Class IV calcium channel blocker.
- C, metoprolol: Class II beta-blocker.

Final Answer: Amiodarone $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – subclasses of Class I: Sodium channel blockers split into IA, IB and IC by their effect on the action potential.

Step 1 – place lidocaine: Lidocaine is Class IB; it blocks sodium channels and shortens the action potential duration, acting selectively on depolarised (ischaemic) ventricular tissue.

Step 2 – use: Given intravenously, it treats ventricular arrhythmias, especially after myocardial infarction.

Why the other options are wrong:

- A, Class III: potassium channel blockers such as amiodarone.
- B, Class II: beta-blockers.
- D, Class IV: calcium channel blockers such as verapamil.

Final Answer: Class IB $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – amiodarone toxicity: Its iodine content and lipophilicity cause diverse organ toxicity.

Step 1 – name the classic effect: Pulmonary fibrosis (interstitial pneumonitis) is the most feared long-term toxicity and can be fatal.

Step 2 – monitoring: Baseline and periodic chest imaging, lung, thyroid and liver function tests are recommended.

Why the other options are wrong:

- B, gingival hyperplasia: seen with phenytoin, ciclosporin and nifedipine.
- C, dry cough from bradykinin: caused by ACE inhibitors.
- D, cyanide toxicity: caused by sodium nitroprusside.

Final Answer: Pulmonary fibrosis ⇒ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – antipsychotic mechanism: Blocking dopamine in the mesolimbic pathway relieves positive symptoms.

Step 1 – name the target: Typical agents such as haloperidol are potent **dopamine D2 receptor** antagonists.

Step 2 – side effect link: D2 blockade in the nigrostriatal pathway produces extrapyramidal symptoms; in the tuberoinfundibular pathway it raises prolactin.

Why the other options are wrong:

- A, 5-HT2A: additionally blocked by atypical agents, which lowers extrapyramidal risk.
- B, muscarinic: blockade causes anticholinergic side effects, not the primary antipsychotic action.
- C, histamine H1: blockade causes sedation and weight gain, not the antipsychotic effect.

Final Answer: Dopamine D2 receptor ⇒ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – atypical antipsychotic toxicity: Atypical agents spare movement but carry other hazards.

Step 1 – identify the drug: Clozapine can cause life-threatening agranulocytosis, so regular white cell (neutrophil) counts are mandatory.

Step 2 – role: Because of this monitoring burden it is reserved for treatment-

resistant schizophrenia.

Why the other options are wrong:

- A, haloperidol: a typical agent, its main risk is extrapyramidal symptoms.
- B, risperidone: mainly raises prolactin, agranulocytosis is not characteristic.
- C, olanzapine: chiefly causes weight gain and metabolic syndrome, not agranulocytosis.

Final Answer: Clozapine $\Rightarrow$ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – polyene antifungals: Amphotericin B targets a sterol unique to fungi.

Step 1 – name the target: It binds **ergosterol** in the fungal cell membrane, forming pores that leak potassium and other ions, killing the cell.

Step 2 – selectivity: Fungal membranes use ergosterol whereas human membranes use cholesterol, giving some selectivity.

Why the other options are wrong:

- A, beta-1,3-glucan: the target of echinocandins, which inhibit its synthesis.
- C, chitin: a wall polymer, not the amphotericin target.
- D, cholesterol: the human sterol; weak binding to it underlies amphotericin toxicity, but ergosterol is the intended target.

Final Answer: Ergosterol $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – amphotericin B toxicity: Its use is limited by predictable organ damage.

Step 1 – name the dose-limiting toxicity: Nephrotoxicity is the major dose-limiting effect, causing renal impairment and potassium and magnesium wasting.

Step 2 – reducing it: Saline pre-loading and lipid (liposomal) formulations lessen

renal damage.

Why the other options are wrong:

- A, hepatotoxicity: more typical of the azoles.
- B, ototoxicity: seen with aminoglycosides and loop diuretics, not amphotericin.
- D, pulmonary fibrosis: an amiodarone and bleomycin effect.

Final Answer: Nephrotoxicity $\Rightarrow$ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – azole antifungals: Azoles inhibit ergosterol synthesis and penetrate the CNS well.

Step 1 – identify the drug: Fluconazole has excellent cerebrospinal fluid penetration and is the drug of choice for consolidation and maintenance therapy of cryptococcal meningitis.

Step 2 – mechanism: It inhibits fungal cytochrome P450 14-alpha-demethylase, blocking conversion of lanosterol to ergosterol.

Why the other options are wrong:

- A, nystatin: a polyene used only topically, it is too toxic systemically.
- C, griseofulvin: used for dermatophyte (ringworm) infections, not meningitis.
- D, terbinafine: an allylamine for dermatophyte nail and skin infections.

Final Answer: Fluconazole $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – oral hypoglycaemic mechanisms: Different classes lower glucose by different routes.

Step 1 – state metformin's action: Metformin (a biguanide) mainly **reduces hepatic gluconeogenesis** and improves peripheral insulin sensitivity, without

stimulating insulin release.

Step 2 – clinical points: Because it does not raise insulin, it does not by itself cause hypoglycaemia; its rare but serious risk is lactic acidosis, so it is avoided in renal failure. Sulfonylureas, by contrast, stimulate insulin release and can cause hypoglycaemia.

Why the other options are wrong:

- A, stimulating insulin release: the mechanism of sulfonylureas.
- C, blocking alpha-glucosidase: the mechanism of acarbose.
- D, increasing renal glucose excretion: the mechanism of SGLT2 inhibitors.

Final Answer: Reducing hepatic gluconeogenesis and improving insulin sensitivity $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – insulin pharmacokinetics: Preparations differ by onset, peak and duration.

Step 1 – read the curve: X rises fastest, peaks early and falls quickly, which is the profile of a rapid-acting analogue.

Step 2 – match it: Insulin lispro has the fastest onset (about 15 minutes) and shortest duration, ideal for mealtime (prandial) cover.

Why the other options are wrong:

- A, insulin glargine: a long-acting, nearly peakless basal insulin (the flat curve).
- B, NPH (isophane): an intermediate-acting insulin with a delayed, broad peak.
- D, protamine zinc insulin: a long-acting preparation, not rapid.

Final Answer: Insulin lispro $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	2	A	3	C	4	B	5	A
6	D	7	C	8	A	9	D	10	D
11	B	12	C	13	B	14	B	15	C