

# NEET PG Pharmacology Sample Paper-8

Duration: 15 Minutes

Maximum Marks: 80

## Instructions

- This paper contains **20** Multiple Choice Questions.
- Each correct answer carries **+4** mark. Incorrect answer: **-1** marks. Only **one** correct option.
- Unattempted questions carry **0** marks.
- Use of mobile phones, smartwatches, or any electronic gadgets is strictly prohibited.

**Q1.** A patient presenting with acute organophosphate poisoning is treated with high-dose atropine. While the muscarinic symptoms resolve, the patient continues to experience severe skeletal muscle fasciculations and diaphragmatic weakness. This persistent effect is mediated by the continuous activation of which receptor type, and why was it unaffected by atropine?

- (A)  $M_2$  receptors; atropine is selective only for  $M_1$  and  $M_3$  subtypes
- (B) Nicotinic receptors ( $N_m$ ); atropine does not block nicotinic cholinceptors
- (C)  $\alpha_1$ -adrenergic receptors; organophosphates cause secondary sympathetic overactivity
- (D) Muscarinic autoreceptors ( $M_4$ ); they require pralidoxime exclusively for competitive reversal

**Q2.** A 49-year-old male is prescribed a new prodrug that requires extensive metabolic activation. During genetic screening, he is found to be a CYP2D6 ultra-rapid metabolizer. If this drug is a substrate for CYP2D6, what is the most likely clinical consequence regarding its therapeutic effect and plasma concentration of the active metabolite?

- (A) Markedly increased therapeutic efficacy and higher peak plasma levels of the active metabolite



- (B) Complete therapeutic failure due to rapid clearance of the parent compound before activation
- (C) Severe systemic toxicity due to accumulated parent drug levels in tissue reservoirs
- (D) No change in clinical response because phase II conjugation pathways will instantly compensate

**Q3.** A 28-year-old male diagnosed with schizophrenia is started on haloperidol. Within 48 hours, he develops severe, painful spasms of the neck muscles (torticollis) and oculogyric crisis. Which of the following mechanisms explains this acute extrapyramidal side effect, and what is the immediate treatment of choice?

- (A) Dopaminergic hypersensitivity in the mesolimbic pathway; treat with bromocriptine
- (B) Acute dopamine  $D_2$  receptor blockade in the nigrostriatal pathway; treat with central anticholinergics like benztropine
- (C) Serotonin 5-HT<sub>2A</sub> receptor antagonism in the frontal cortex; treat with dantrolene
- (D) GABAergic inhibition in the substantia nigra; treat with diazepam

**Q4.** A 34-year-old pregnant woman in her third trimester is diagnosed with a severe lower urinary tract infection. Which of the following antimicrobial agents is contraindicated in this patient due to the specific risk of causing grey baby syndrome if delivery occurs prematurely, or hemolytic anemia in the newborn if G6PD deficient?

- (A) Amoxicillin-clavulanate
- (B) Nitrofurantoin
- (C) Chloramphenicol
- (D) Azithromycin

**Q5.** A 62-year-old patient with long-standing Type 2 Diabetes Mellitus and chronic kidney disease (eGFR = 28 mL/min/1.73 m<sup>2</sup>) requires optimization



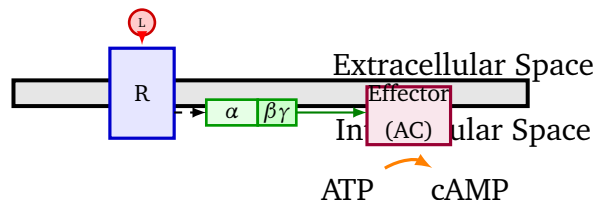
of glycemic control. Which of the following antidiabetic agents is contraindicated at this stage of renal function due to an increased risk of lactic acidosis?

- (A) Linagliptin
- (B) Metformin
- (C) Empagliflozin
- (D) Pioglitazone

**Q6.** An experimental drug 'X' is evaluated for its pharmacokinetics. It is administered as an intravenous bolus of 500 mg. The plasma concentration extrapolated to time zero is found to be 12.5 mg/L. What is the apparent volume of distribution ( $V_d$ ) of this drug, and what does this value imply about its tissue distribution?

- (A) 40 L; the drug distributes widely into total body water but does not sequester highly in tissues
- (B) 4 L; the drug is largely confined to the vascular compartment
- (C) 400 L; the drug is extensively bound to peripheral tissue proteins
- (D) 0.025 L; the drug has been rapidly eliminated by first-pass hepatic clearance

**Q7.** Consider the schematic representing a signal transduction pathway across a cellular membrane. Identify the specific receptor type and its immediate downstream effector mechanism depicted in the diagram:



- (A)  $G_q$ -coupled protein receptor; activates Phospholipase C to generate  $IP_3$  and DAG
- (B)  $G_s$ -coupled protein receptor; activates Adenylyl Cyclase to increase cAMP levels



- (C) Ligand-gated ion channel; permits rapid influx of extracellular calcium ions
- (D) Receptor tyrosine kinase; initiates autophosphorylation of intracellular domains

**Q8.** A 60-year-old male with chronic open-angle glaucoma is prescribed topical timolol drops. He has a co-existing medical history of moderate bronchial asthma. He presents to the emergency department with severe wheezing and respiratory distress shortly after starting the drops. What is the mechanism behind this presentation?

- (A) Topical timolol causes systemic  $\alpha_1$  blockade leading to pulmonary congestion
- (B) Timolol is a non-selective  $\beta$ -blocker that passes through the nasolacrimal duct and causes bronchoconstriction via  $\beta_2$  blockade
- (C) Timolol stimulates muscarinic  $M_3$  receptors in the bronchioles causing mucus hypersecretion
- (D) Systemic absorption of timolol causes direct mast cell degranulation in the respiratory tract

**Q9.** A patient with severe major depressive disorder is treated with phenelzine, a Monoamine Oxidase Inhibitor (MAOI). During a family dinner, they consume aged cheese and red wine, and shortly after develop a throbbing headache, palpitations, and a blood pressure reading of 210/120 mmHg. This acute hypertensive crisis is due to the displacement of which neurotransmitter by what dietary amine?

- (A) Displacement of Norepinephrine by Tyramine
- (B) Displacement of Serotonin by Tryptophan
- (C) Displacement of Dopamine by Phenylalanine
- (D) Displacement of Acetylcholine by Choline

**Q10.** A 53-year-old woman undergoing chemotherapy for breast cancer experiences severe, refractory nausea and vomiting. She is prescribed aprepitant.



What is the mechanism of action of aprepitant in managing chemotherapy-induced nausea and vomiting (CINV)?

- (A) Antagonism of Serotonin 5-HT<sub>3</sub> receptors in the chemoreceptor trigger zone
- (B) Antagonism of Neurokinin-1 (NK<sub>1</sub>) receptors in the central nervous system
- (C) Agonism of Cannabinoid CB<sub>1</sub> receptors in the solitary tract nucleus
- (D) Antagonism of Dopamine D<sub>2</sub> receptors in the area postrema

**Q11.** An intensive care unit patient has developed a nosocomial lower respiratory tract infection caused by *Pseudomonas aeruginosa*. Which of the following beta-lactam antibiotics offers reliable empiric coverage against this specific pathogen?

- (A) Ceftriaxone
- (B) Cefotaxime
- (C) Ceftazidime
- (D) Ertapenem

**Q12.** A 45-year-old male is initiated on high-dose therapy with an anti-arrhythmic agent for refractory ventricular tachycardia. During follow-up, he describes symptoms of weight gain, cold intolerance, dry skin, and lethargy. Thyroid function tests confirm drug-induced hypothyroidism. Which of the following drugs is most likely responsible?

- (A) Amiodarone
- (B) Procainamide
- (C) Lidocaine
- (D) Verapamil

**Q13.** A drug with a narrow therapeutic index is eliminated strictly via first-order kinetics. If the steady-state plasma concentration is reached after 4 to 5 half-



lives, what change occurs to the time required to reach a new steady-state if the daily maintenance dose is doubled?

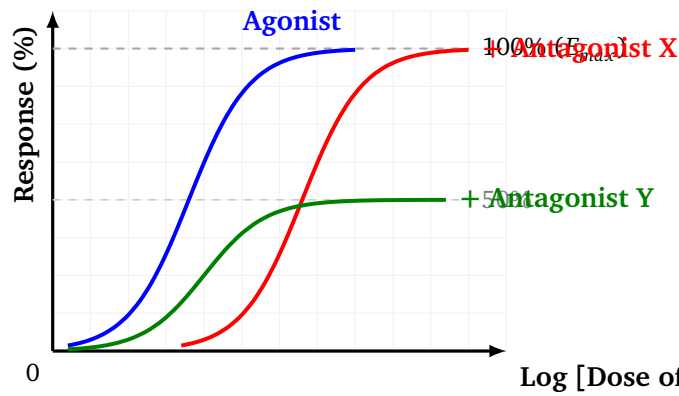
- (A) The time to reach the new steady-state will double because the drug load is twice as high
- (B) The time to reach the new steady-state remains unchanged because it depends solely on the half-life
- (C) The time to reach the new steady-state will decrease by half due to saturation of clearance mechanisms
- (D) The time to reach the new steady-state becomes unpredictable as the system switches to zero-order clearance

**Q14.** A 68-year-old male with benign prostatic hyperplasia (BPH) and hypertension is prescribed tamsulosin. He reports significant improvement in urinary flow and minimal postural dizziness compared to his previous treatment with doxazosin. This clinical advantage of tamsulosin is explained by its high selectivity for which specific receptor subtype?

- (A)  $\alpha_{1B}$  adrenergic receptors located predominantly in vascular smooth muscle
- (B)  $\alpha_{1A}$  adrenergic receptors located predominantly in the prostate and bladder neck
- (C)  $\alpha_2$  adrenergic autoreceptors located in the central nervous system
- (D)  $\beta_2$  adrenergic receptors located in the detrusor muscle

**Q15.** The given dose-response curve illustrates the effect of an agonist alone, and in the presence of two different antagonists (X and Y). Identify the natures of antagonist X and antagonist Y based on the profile of the curves:





- (A) X is a competitive antagonist; Y is a non-competitive antagonist  
 (B) X is a non-competitive antagonist; Y is a competitive antagonist  
 (C) X is an irreversible antagonist; Y is a chemical antagonist  
 (D) X is a physiological antagonist; Y is a competitive antagonist

**Q16.** A patient diagnosed with severe bipolar disorder is managed with lithium carbonate. She presents with polyuria, polydipsia, and dilute urine with low osmolality that fails to concentrate following administration of exogenous desmopressin. Which mechanism explains this lithium-induced adverse effect?

- (A) Downregulation of central  $V_1$  vasopressin receptors in the hypothalamus  
 (B) Blockade of Aquaporin-2 vesicle insertion in the renal collecting duct via interference with cAMP signaling  
 (C) Hyperresponsiveness of Henle's loop to thick ascending limb sodium reabsorption  
 (D) Excessive stimulation of renal proximal tubular sodium-hydrogen exchangers

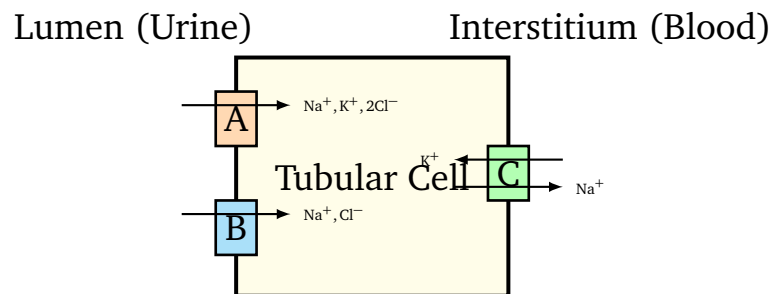
**Q17.** A 24-year-old female presents to the clinic with a history of recurrent vulvovaginal candidiasis. She is prescribed a single oral dose of fluconazole. Fluconazole exerts its antifungal action by inhibiting which of the following essential fungal enzymes?

- (A) Squalene epoxidase



- (B)  $14\alpha$ -demethylase
- (C)  $\beta$ -(1,3)-D-glucan synthase
- (D) Topoisomerase II

**Q18.** The accompanying diagram illustrates the molecular site of action of four distinct classes of loop and thiazide diuretics within a renal tubular epithelial cell. Which lettered transporter or channel represents the primary site of inhibition for chlorthalidone?



- (A) Transporter A ( $\text{Na}^+/\text{K}^+ / 2\text{Cl}^-$  symporter)
  - (B) Transporter B ( $\text{Na}^+/\text{Cl}^-$  symporter)
  - (C) Transporter C ( $\text{Na}^+/\text{K}^+$ -ATPase pump)
  - (D) None of the above; chlorthalidone acts intracellularly on carbonic anhydrase
- Q19.** A 40-year-old HIV-positive patient is initiating a highly active antiretroviral therapy (HAART) regimen containing dolutegravir. Dolutegravir belongs to which class of antiretroviral drugs?
- (A) Nucleoside Reverse Transcriptase Inhibitors (NRTIs)
  - (B) Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs)
  - (C) Integrase Strand Transfer Inhibitors (INSTIs)
  - (D) Protease Inhibitors (PIs)
- Q20.** A 58-year-old female with a mechanical heart valve has been maintained on warfarin terminated anticoagulation for three years. She is recently treated for an acute infection and started on a 7-day course of rifampin. What adjustment should be anticipated regarding her warfarin dosing schedule?



- (A) Warfarin dose must be decreased because rifampin inhibits CYP2C9 metabolic pathways
- (B) Warfarin dose must be increased because rifampin is a potent inducer of hepatic CYP2C9 enzymes
- (C) Warfarin should be discontinued immediately due to irreversible competitive displacement from plasma albumin
- (D) No change is needed as rifampin selectively interacts only with low molecular weight heparin clearings



**Detailed Solutions**

Q1.

**Solution**

**Concept:** Organophosphate compounds irreversibly inhibit acetylcholinesterase, leading to an extreme accumulation of acetylcholine within the synaptic clefts of both muscarinic and nicotinic receptors. While atropine acts as a highly effective competitive antagonist at muscarinic receptors to alleviate autonomic symptoms, it lacks efficacy at nicotinic receptors found at neuromuscular junctions.

**Solution:**

- (a) Organophosphates cause toxic accumulations of acetylcholine at all cholinergic synapses throughout the central and peripheral nervous systems.
- (b) Atropine selectively blocks muscarinic receptors, effectively reversing life-threatening symptoms such as severe bradycardia, excessive bronchial secretions, and bronchoconstriction.
- (c) Overstimulation of nicotinic receptors ( $N_m$ ) at the neuromuscular junction causes skeletal muscle fasciculations, continuous depolarization, and eventual muscle weakness or diaphragmatic paralysis.
- (d) Atropine has no pharmacological activity at nicotinic receptors; therefore, neuromuscular symptoms remain completely unaffected by its administration.
- (e) Pralidoxime or other oxime reactivators are required alongside atropine to regenerate functional acetylcholinesterase and relieve these nicotinic manifestations.

**Final Answer:** Nicotinic receptors ( $N_m$ ); atropine does not block nicotinic cholinceptors

**Answer: (B)**

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Q2.

**Solution**

**Concept:** Prodrugs are pharmacologically inactive compounds that require enzymatic biotransformation within the body to convert into their active, therapeutic forms. Genetic polymorphisms that alter cytochrome P450 enzyme activity can profoundly impact the rate of this activation process and dictate overall clinical outcomes.

**Solution:**

- (a) CYP2D6 ultra-rapid metabolizers possess multiple functional copies of the CYP2D6 gene, causing exceptionally high hepatic enzyme expression and activity.
- (b) When a patient with this phenotype receives a prodrug requiring CYP2D6, the parent drug undergoes accelerated biotransformation into its active state.
- (c) This rapid metabolism causes a swift drop in parent drug levels alongside a steep rise in active metabolite plasma concentrations.
- (d) The elevated active metabolite concentrations markedly amplify the primary therapeutic effects and increase the risk of dose-dependent toxicities.
- (e) A classic clinical example is the rapid conversion of codeine into morphine, which can lead to life-threatening opioid toxicity.

**Final Answer:** Markedly increased therapeutic efficacy and higher peak plasma levels of the active metabolite

**Answer: (A)**

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Q3.

**Solution**

**Concept:** Acute dystonia is an extrapyramidal side effect typically triggered by high-potency first-generation antipsychotics like haloperidol. It manifests as sudden, painful, and involuntary contractions of distinct muscle groups shortly after starting the medication or following a significant dose increase.

**Solution:**

- (a) Haloperidol exerts strong antagonism at dopamine  $D_2$  receptors within the nigrostriatal pathway to cause a dramatic reduction in dopaminergic neurotransmission.
- (b) This sudden blockade disrupts the basal ganglia's functional equilibrium, leading to a state of relative cholinergic excess.
- (c) The resulting overactivity of cholinergic neurons drives the sudden, involuntary muscle contractions characteristic of acute dystonic reactions.
- (d) Central anticholinergic agents, such as benztropine or diphenhydramine, quickly restore the essential balance between dopamine and acetylcholine.
- (e) Implementing these anticholinergics via intravenous or intramuscular injection provides rapid, highly effective relief from painful muscle spasms.

**Final Answer:** Acute dopamine  $D_2$  receptor blockade in the nigrostriatal pathway; treat with central anticholinergics like benztropine

**Answer: (B)**

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Q4.

**Solution**

**Concept:** Antimicrobial selection during pregnancy requires careful evaluation of potential teratogenic risks and neonatal toxicities. Certain antibiotics can easily cross the placental barrier and cause severe biochemical disruption or hematological damage in developing fetuses and newborns.

**Solution:**

- (a) Chloramphenicol can cross the placenta and accumulate significantly in neonates due to their immature hepatic and renal clearance pathways.
- (b) Neonates possess insufficient glucuronosyltransferase activity, an enzyme necessary for the metabolic detoxification and clearance of chloramphenicol.
- (c) Drug accumulation impairs mitochondrial protein synthesis, culminating in cardiovascular collapse and the characteristic presentation of grey baby syndrome.
- (d) Chloramphenicol induces significant oxidative stress, triggering acute hemolytic anemia in newborns with glucose-6-phosphate dehydrogenase (G6PD) deficiency.
- (e) Consequently, chloramphenicol is strictly contraindicated during the third trimester of pregnancy to ensure neonatal safety.

**Final Answer:** Chloramphenicol

**Answer:** (C)

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Q5.

**Solution**

**Concept:** Selecting appropriate antidiabetic pharmacotherapy for Type 2 Diabetes Mellitus requires careful adjustment based on the patient's renal function. Several medications rely extensively on renal clearance or can drastically amplify metabolic complications when kidney function falls below specific thresholds.

**Solution:**

- (a) Metformin reduces hepatic gluconeogenesis and increases peripheral glucose uptake while promoting intracellular lactate production.
- (b) Under normal physiological conditions, metformin is cleared efficiently by the kidneys via active tubular secretion.
- (c) In severe chronic kidney disease, diminished renal clearance causes systemic accumulation of metformin.
- (d) High systemic concentrations of metformin inhibit mitochondrial oxidative phosphorylation, driving significant overproduction and accumulation of lactic acid.
- (e) Metformin is strictly contraindicated when the eGFR drops below 30 mL/min/1.73 m<sup>2</sup> to minimize the risk of fatal lactic acidosis.

**Final Answer:** Metformin

**Answer: (B)**

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Q6.

**Solution**

**Concept:** The apparent volume of distribution ( $V_d$ ) is a fundamental pharmacokinetic parameter that relates the total amount of drug in the body to its measured plasma concentration. It provides essential insight into whether a drug remains confined within the vascular space or distributes into peripheral tissues.

**Solution:**

- (a) The volume of distribution is calculated using the standard equation:  $V_d = \text{Dose}/C_0$ , where  $C_0$  represents the plasma concentration extrapolated to time zero.
- (b) Substituting the given values into the equation yields:  $V_d = 500 \text{ mg}/12.5 \text{ mg/L} = 40 \text{ L}$ .
- (c) A  $V_d$  value near 40 L matches the average volume of total body water in an adult.
- (d) This indicates that the drug distributes widely through total body water but lacks significant sequestration or trapping within peripheral tissue proteins.
- (e) In contrast, a low  $V_d$  signifies vascular restriction, whereas a high  $V_d$  indicates extensive tissue binding.

**Final Answer:** 40 L; the drug distributes widely into total body water but does not sequester highly in tissues

**Answer: (A)**

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Q7.

**Solution**

**Concept:** G-protein coupled receptors (GPCRs) represent a major class of cell surface receptors that translate extracellular signals into intracellular responses. They utilize specialized heterotrimeric G-proteins to regulate specific downstream effector enzymes and alter second messenger concentrations.

**Solution:**

- (a) The provided schematic displays a classic ligand-activated transmembrane receptor operating in conjunction with a heterotrimeric G-protein ( $\alpha\beta\gamma$  subunits).
- (b) Upon ligand binding, the activated  $\alpha$  subunit detaches and interacts directly with the membrane-bound enzyme, adenylyl cyclase (AC).
- (c) This explicit stimulation of adenylyl cyclase is the definitive characteristic of a  $G_s$ -coupled protein receptor pathway.
- (d) Adenylyl cyclase catalyzes the biochemical conversion of adenosine triphosphate (ATP) into cyclic adenosine monophosphate (cAMP).
- (e) The generated cAMP functions as a key second messenger, initiating intracellular cascades through the activation of protein kinase A (PKA).

**Final Answer:**  $G_s$ -coupled protein receptor; activates Adenylyl Cyclase to increase cAMP levels

**Answer: (B)**

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Q8.

**Solution**

**Concept:** Topical ophthalmic medications can undergo significant systemic absorption, bypassing first-pass hepatic metabolism and exerting potent systemic effects. When administering non-selective autonomic agents, clinicians must consider potential systemic contraindications and adverse reactions.

**Solution:**

- (a) Topical ophthalmic drops drain through the nasolacrimal duct into the nasal mucosa, where rapid systemic absorption occurs via the local capillary network.
- (b) Timolol is a highly potent, non-selective  $\beta$ -adrenergic antagonist used to reduce intraocular pressure in glaucoma management.
- (c) Systemically absorbed timolol exerts a strong antagonistic effect on  $\beta_2$ -adrenergic receptors located within bronchial smooth muscle.
- (d) Blocking these  $\beta_2$  receptors prevents epinephrine-mediated bronchodilation, precipitating acute bronchoconstriction in patients with reactive airway disease.
- (e) Consequently, non-selective topical  $\beta$ -blockers are contraindicated in patients with moderate-to-severe bronchial asthma due to the risk of respiratory distress.

**Final Answer:** Timolol is a non-selective  $\beta$ -blocker that passes through the nasolacrimal duct and causes bronchoconstriction via  $\beta_2$  blockade

**Answer: (B)**

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Q9.

**Solution**

**Concept:** Monoamine oxidase inhibitors (MAOIs) block the degradation of biogenic amines within both the gastrointestinal tract and the central nervous system. Ingesting foods rich in dietary amines during MAOI therapy can prevent normal intestinal clearance, triggering severe cardiovascular complications.

**Solution:**

- (a) Phenelzine is an MAOI that inhibits the monoamine oxidase enzymes responsible for breaking down catecholamines and dietary amines.
- (b) Aged cheese and red wine contain high levels of tyramine, an amine normally metabolized by MAO-A in the intestinal wall.
- (c) When MAO-A is inhibited, tyramine is absorbed intact into the systemic circulation and travels to sympathetic nerve terminals.
- (d) Systemic tyramine enters sympathetic vesicles via the vesicular monoamine transporter, displacing vast amounts of stored norepinephrine into the synaptic cleft.
- (e) This massive release of norepinephrine causes widespread vasoconstriction and intense cardiac stimulation, resulting in a severe hypertensive crisis.

**Final Answer:** Displacement of Norepinephrine by Tyramine

**Answer: (A)**

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Q10.

**Solution**

**Concept:** Chemotherapy-induced nausea and vomiting (CINV) involves complex neurochemical pathways in both the peripheral abdominal vagal afferents and the central emetic centers. Managing refractory emesis often requires targeted antagonism of specific neurotransmitter receptors involved in the vomiting reflex.

**Solution:**

- (a) Highly emetogenic chemotherapy triggers the release of substance P, an essential tachykinin neurotransmitter involved in delayed emetic pathways.
- (b) Substance P binds to neurokinin-1 ( $NK_1$ ) receptors located within the solitary tract nucleus and the area postrema of the central nervous system.
- (c) Aprepitant functions as a highly selective, central antagonist at these  $NK_1$  receptors, blocking the emetic actions of substance P.
- (d) By preventing substance P binding, aprepitant significantly reduces both acute and delayed phases of chemotherapy-induced emesis.
- (e) This agent is typically combined with 5-HT<sub>3</sub> receptor antagonists and corticosteroids to optimize antiemetic efficacy.

**Final Answer:** Antagonism of Neurokinin-1 ( $NK_1$ ) receptors in the central nervous system

**Answer: (B)**

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Q11.

**Solution**

**Concept:** Nosocomial lower respiratory tract infections often involve highly opportunistic and multi-drug resistant pathogens. Among these, *Pseudomonas aeruginosa* is a notorious Gram-negative bacterium requiring targeted, specific beta-lactam therapy. Proper selection involves identifying antibiotics structurally modified to penetrate its outer membrane and withstand its defensive beta-lactamases.

**Solution:**

- (a) *Pseudomonas aeruginosa* is an aggressive healthcare-associated pathogen capable of causing severe pneumonia in critically ill intensive care unit patients.
- (b) Standard third-generation cephalosporins, including ceftriaxone and cefotaxime, lack the structural affinity required to overcome the intrinsic resistance mechanisms of this organism.
- (c) Ceftazidime is an advanced third-generation cephalosporin with unique side chains that grant high-affinity binding to pseudomonal penicillin-binding proteins.
- (d) Carbapenem selection must be approached cautiously, as ertapenem stands out as the solitary carbapenem lacking any clinical efficacy against pseudomonal strains.
- (e) Therefore, ceftazidime provides reliable empiric coverage, often combined with an aminoglycoside or fluoroquinolone to guarantee adequate treatment and prevent resistance.

**Final Answer:** Ceftazidime provides reliable empiric coverage against *Pseudomonas aeruginosa*.

Answer: (C)

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Q12.

**Solution**

**Concept:** Anti-arrhythmic drugs are powerful therapeutic agents possessing diverse pharmacodynamic profiles that can inadvertently influence system-wide metabolic pathways. Chronic administration of agents containing dense halogenated structures often alters systemic glandular function, resulting in drug-induced organ toxicity.

**Solution:**

- (a) Amiodarone is a highly effective Class III anti-arrhythmic medication used to suppress severe, refractory ventricular tachyarrhythmias.
- (b) The chemical structure of amiodarone contains a high percentage of inorganic iodine by weight, mimicking thyroid hormone structures.
- (c) Metabolism of a standard daily dose releases massive quantities of free iodine into the systemic circulation, disrupting physiological homeostasis.
- (d) This excess iodine can trigger the Wolff-Chaikoff effect, an autoregulatory mechanism where high iodine levels acutely inhibit thyroid hormone synthesis.
- (e) The patient's clinical presentation of unexpected weight gain, persistent cold intolerance, dry skin, and profound lethargy strongly confirms amiodarone-induced hypothyroidism.

**Final Answer:** Amiodarone is responsible for causing drug-induced hypothyroidism.

**Answer: (A)**

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Q13.

**Solution**

**Concept:** Steady-state pharmacokinetics describe the equilibrium condition where the rate of systemic drug administration matches the rate of drug elimination. Under fundamental first-order kinetic principles, the time necessary to achieve this steady-state concentration is governed entirely by intrinsic biological parameters.

**Solution:**

- (a) In first-order elimination kinetics, a constant fraction of the total drug load is metabolized and cleared per unit time.
- (b) The mathematical time required to achieve steady-state is defined strictly as four to five biological half-lives ( $t_{1/2}$ ) of the molecule.
- (c) When a maintenance dose is doubled, the ultimate concentration achieved at steady-state will also double in a linear fashion.
- (d) However, because clearance mechanisms are operating far below saturation, the intrinsic half-life of the drug remains completely unchanged.
- (e) Consequently, the absolute time required to reach the new, higher steady-state concentration remains exactly four to five half-lives.

**Final Answer:** The time to reach the new steady-state remains unchanged because it depends solely on the half-life.

**Answer: (B)**

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Q14.

**Solution**

**Concept:** Selective adrenergic receptor antagonism allows for targeted clinical management of localized smooth muscle hypertonicity while mitigating systemic side effects. Distinguishing between receptor subtypes distributed in different tissues is critical for optimizing patient compliance and safety profiles.

**Solution:**

- (a) Benign prostatic hyperplasia results in mechanical urinary obstruction mediated heavily by contraction of smooth muscle fibers within the prostate.
- (b) These localized smooth muscle contractions are driven primarily through the activation of specialized alpha-1A adrenergic receptor subtypes.
- (c) Older alpha-blockers like doxazosin non-selectively block alpha-1B receptors in blood vessels, frequently causing systemic vasodilation and orthostatic hypotension.
- (d) Tamsulosin exhibits high selective affinity for alpha-1A receptors, isolating its relaxant effects to the prostate and bladder neck.
- (e) This targeted action significantly enhances lower urinary tract flow rates while avoiding significant vascular effects, explaining the absence of dizziness.

**Final Answer:** Tamsulosin is selective for alpha-1A adrenergic receptors located predominantly in the prostate and bladder neck.

**Answer: (B)**

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Q15.

**Solution**

**Concept:** Dose-response relationships provide critical quantitative visual metrics regarding the pharmacological mechanisms and binding interactions of antagonist molecules. Analyzing changes in maximum efficacy versus structural shifts in potency clarifies whether an inhibitor can be surmountably displaced.

**Solution:**

- (a) The control agonist curve displays a classic sigmoidal shape with a maximum response reaching full saturation at one hundred percent efficacy.
- (b) Antagonist X causes a parallel rightward shift of the curve, keeping maximum response intact while increasing the apparent EC<sub>50</sub> value.
- (c) This surmountable inhibition is the definitive classic hallmark of a competitive antagonist competing directly for the active receptor site.
- (d) Antagonist Y produces a depressed curve characterized by a notable decrease in maximum response without a significant parallel shift.
- (e) This irreversible or allosteric reduction in efficacy confirms a non-competitive mechanism, where ligand binding cannot overcome the inhibition.

**Final Answer:** X is a competitive antagonist and Y is a non-competitive antagonist.

**Answer: (A)**

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Q16.

**Solution**

**Concept:** Drug-induced nephrogenic diabetes insipidus is a classic adverse effect characterized by an inability of the renal collecting ducts to concentrate urine. This occurs when an exogenous substance interferes directly with the intracellular cascade downstream of antidiuretic hormone receptors.

**Solution:**

- (a) Bipolar disorder management with lithium carbonate carries an inherent risk of renal accumulation within the principal cells of collecting ducts.
- (b) Lithium enters these cells via epithelial sodium channels and directly blocks the activity of the enzyme adenylyl cyclase.
- (c) This enzymatic blockade inhibits the generation of cyclic adenosine monophosphate following vasopressin V2 receptor activation by endogenous ADH.
- (d) Deprived of adequate second messenger signaling, Aquaporin-2 storage vesicles fail to undergo exocytosis and insert into the luminal membrane.
- (e) The kidney loses its water permeability, rendering exogenous desmopressin completely ineffective and causing profound polyuria and dilute urine.

**Final Answer:** Lithium blocks Aquaporin-2 vesicle insertion in the renal collecting duct via interference with cAMP signaling.

**Answer: (B)**

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Q17.

**Solution**

**Concept:** Antifungal pharmacotherapy relies on exploiting structural differences between host cellular membranes and fungal cell walls. Disrupting the biosynthetic pathways responsible for maintaining fungal cell membrane integrity serves as an effective mechanism for stopping fungal proliferation.

**Solution:**

- (a) Fungal cell membranes depend strictly on ergosterol to maintain appropriate fluidity, structural rigidity, and vital transport channel functions.
- (b) Fluconazole belongs to the azole class of antifungal medications, widely utilized for handling local and systemic Candida infections.
- (c) The primary mechanism of fluconazole involves selective, high-affinity inhibition of the microsomal cytochrome P450 enzyme, 14 $\alpha$ -demethylase.
- (d) This specific enzyme is essential for converting the precursor molecule lanosterol into functional ergosterol inside fungal cells.
- (e) Inhibiting 14 $\alpha$ -demethylase depletes ergosterol and causes toxic methylated sterols to accumulate, destabilizing the fungal membrane and halting growth.

**Final Answer:** Fluconazole exerts its antifungal action by inhibiting 14 $\alpha$ -demethylase.

**Answer: (B)**

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Q18.

**Solution**

**Concept:** Diuretics are categorized based on their specific anatomical site of action along the nephron and the corresponding transport proteins they inhibit. Matching a drug class to its appropriate molecular target clarifies its physiological efficacy and secondary electrolyte disturbances.

**Solution:**

- (a) The provided renal epithelial cell schematic illustrates distinct ionic transport systems operating on the luminal and basolateral cell membranes.
- (b) Transporter A represents the sodium-potassium-two-chloride symporter located exclusively in the thick ascending limb, which is inhibited by loop diuretics.
- (c) Transporter B represents the luminal sodium-chloride symporter, responsible for electrolyte reabsorption within the early distal convoluted tubule.
- (d) Chlorthalidone is a potent thiazide-like diuretic that selectively binds and blocks this specific sodium-chloride symporter to promote diuresis.
- (e) This inhibition increases luminal delivery of sodium and water to distal segments, matching the functional localization of Transporter B.

**Final Answer:** Chlorthalidone primary site of inhibition is Transporter B, the sodium-chloride symporter.

**Answer: (B)**

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Q19.

**Solution**

**Concept:** Highly active antiretroviral therapy for human immunodeficiency virus utilizes distinct drug combinations to target multiple stages of the viral replication cycle. Inhibiting the specific viral enzymes necessary for integrating viral genetic material prevents permanent host genome modification.

**Solution:**

- (a) Following reverse transcription, HIV retroviral genetic material exists as a double-stranded complementary DNA molecule within the host cytoplasm.
- (b) The replication process requires this viral DNA to cross the nuclear membrane and insert directly into the host chromosomes.
- (c) This insertion process is catalyzed exclusively by the viral integrase enzyme, a key target in antiretroviral therapy.
- (d) Dolutegravir is an advanced agent belonging to the Integrase Strand Transfer Inhibitor class of antiretroviral drugs.
- (e) By binding to the integrase active site, dolutegravir effectively blocks the strand transfer step, successfully halting viral replication.

**Final Answer:** Dolutegravir belongs to the Integrase Strand Transfer Inhibitor class.

**Answer: (C)**

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Q20.

**Solution**

**Concept:** Concomitant administration of multiple medications frequently introduces significant drug-drug interactions mediated by hepatic enzyme induction or inhibition. Anticipating changes in cytochrome P450 enzyme expression is vital for preserving therapeutic efficacy and preventing severe thromboembolic complications.

**Solution:**

- (a) Warfarin is an oral anticoagulant requiring precise dosing maintenance, metabolized primarily by hepatic cytochrome P450 2C9 enzymes.
- (b) Rifampin is a powerful antimicrobial agent that acts as a highly potent inducer of multiple hepatic CYP450 isoenzymes, including CYP2C9.
- (c) Co-administration of rifampin upregulates expression of these enzymes, rapidly accelerating the biotransformation and clearance of circulating warfarin.
- (d) The accelerated clearance causes a steep drop in warfarin plasma levels, risking inadequate anticoagulation and artificial valve thrombosis.
- (e) To compensate for this interaction and preserve a therapeutic international normalized ratio, the maintenance dose of warfarin must be increased.

**Final Answer:** Warfarin dose must be increased because rifampin is a potent inducer of hepatic CYP2C9 enzymes.

**Answer: (B)**

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**Answer Key**

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	B	2	A	3	B	4	C	5	B
6	A	7	B	8	B	9	A	10	B
11	C	12	A	13	B	14	B	15	A
16	B	17	B	18	B	19	C	20	B

