

# NEET PG Pharmacology Sample Paper-10

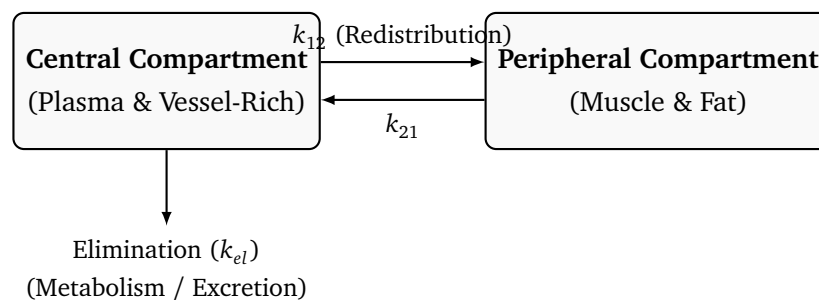
Duration: 15 Minutes

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.** An experimental drug follows the multi-compartment distribution model illustrated below. After a single rapid intravenous bolus injection, the initial rapid decline in plasma concentration is primarily driven by redistribution.

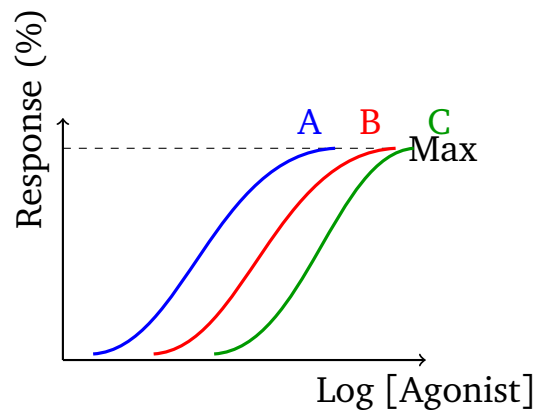


For which of the following highly lipid-soluble drugs does this redistribution mechanism predominantly terminate its central nervous system action?

- (A) Phenobarbital
- (B) Thiopental
- (C) Midazolam
- (D) Morphine

**Q2.** A new competitive antagonist 'Drug X' is studied in an isolated tissue preparation. Refer to the log dose-response diagram below representing the effect of an agonist alone (Curve A) and in the presence of increasing concentrations of Drug X (Curves B and C).





Which of the following parameters remains unchanged for the agonist in Curve C compared to Curve A?

- (A)  $EC_{50}$
- (B) Efficacy ( $E_{max}$ )
- (C) Potency
- (D) Dissociation constant ( $K_d$ ) of the agonist-receptor complex

**Q3.** A 45-year-old patient with chronic kidney disease is prescribed a drug that is cleared strictly by first-order glomerular filtration. If the patient's remaining functional GFR drops by exactly 50%, what will be the new steady-state plasma concentration ( $C_{ss}$ ) of the drug if the dose and dosing interval remain unchanged?

- (A) Unchanged
- (B) Decreased by 50%
- (C) Increased by 50%
- (D) Increased by 100%

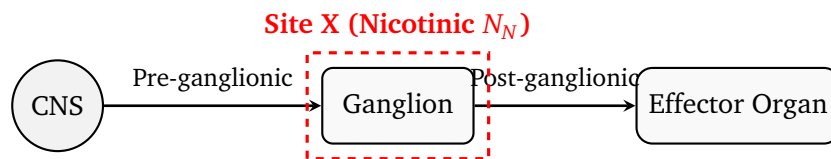
**Q4.** Which of the following statements best defines the term 'inverse agonist' in molecular pharmacology?

- (A) A drug that binds to the receptor but produces no conformational change or intrinsic biological activity.
- (B) A drug that binds to a distinct allosteric site to non-competitively inhibit the primary agonist.



- (C) A drug that stabilizes the inactive conformation ( $R$ ) of a constitutively active receptor, reducing basal activity.
- (D) A drug that binds to the active state ( $R^*$ ) but lacks the structural elements needed to activate downstream G-proteins.

**Q5.** Consider the basic schematic of the autonomic nervous system pathways shown below. An unknown experimental compound blocks the transmission at the site labeled 'Site X'.



If the compound is classified specifically as a ganglionic blocker acting at Site X, which of the following physiological manifestations is most likely to be observed due to the elimination of dominant autonomic tone?

- (A) Severe miosis
- (B) Marked diarrhea and hypermotility
- (C) Orthostatic hypotension and tachycardia/mydriasis depending on dominant resting tone
- (D) Profuse generalized diaphoresis
- Q6.** A 62-year-old male with a history of severe bronchial asthma and benign prostatic hyperplasia (BPH) presents with acute angle-closure glaucoma. Which of the following ophthalmic agents is absolutely contraindicated in this patient due to systemic absorption risks altering his comorbid pulmonary pathology?
- (A) Pilocarpine
- (B) Apraclonidine
- (C) Latanoprost
- (D) Timolol



- Q7.** A patient accidentally ingests an insecticide containing an irreversible acetylcholinesterase inhibitor. He is brought to the emergency department displaying profound bradycardia, bronchorrhea, and skeletal muscle fasciculations. To reverse both the central/muscarinic signs AND the nicotinic neuromuscular junction signs, which agent must be administered promptly before enzyme aging occurs?
- (A) Atropine
  - (B) Pralidoxime
  - (C) Physostigmine
  - (D) Neostigmine
- Q8.** A selective  $\beta_1$ -adrenergic receptor antagonist with membrane-stabilizing activity and high intrinsic sympathomimetic activity (ISA) is ideal for treating certain patients prone to excessive bradycardia. Which of the following drugs fits this description?
- (A) Metoprolol
  - (B) Atenolol
  - (C) Acebutolol
  - (D) Propranolol
- Q9.** A 34-year-old pregnant woman in her first trimester requires maintenance therapy for generalized tonic-clonic seizures. Which anticonvulsant should be strictly avoided due to its high association with neural tube defects like spina bifida bifida and facial dysmorphism?
- (A) Levetiracetam
  - (B) Valproic acid
  - (C) Lamotrigine
  - (D) Carbamazepine
- Q10.** A patient being treated for Parkinson's disease with a combination regimen experiences severe visual hallucinations and dyskinesia. The physician decides



to reduce the central conversion rate of levodopa without losing peripheral dopa-decarboxylase inhibition. Which of the following mechanisms correctly matches Selegiline's role if it were added to modify dopamine levels?

- (A) Irreversible inhibition of Peripheral COMT
- (B) Reversible activation of central D3 receptors
- (C) Selective, irreversible inhibition of Monoamine Oxidase B (MAO-B)
- (D) Blockade of central dopamine reuptake transporters (DAT)

**Q11.** An emergency room patient presents with severe respiratory depression, pinpoint pupils, and coma following an intentional overdose. His friend notes he took several "yellow pills" from an old prescription bottle. After administration of an intravenous antagonist, the patient abruptly awakens and displays signs of acute withdrawal. What was the mechanism of action of the drug used to reverse this state?

- (A) Competitive antagonist at  $GABA_A$  receptors
- (B) Competitive antagonist at  $\mu$ -opioid receptors
- (C) Functional antagonist through NMDA receptor blockade
- (D) Partial agonist at  $\alpha_2$ -adrenergic receptors

**Q12.** Among the modern atypical antipsychotics, which agent carries the highest risk of causing metabolic syndrome, profound weight gain, and dyslipidemia, while possessing a relatively low propensity for extrapyramidal side effects (EPS)?

- (A) Aripiprazole
- (B) Ziprasidone
- (C) Olanzapine
- (D) Haloperidol

**Q13.** A 28-year-old male presents with a painful urethral discharge. Microscopic evaluation confirms a *Neisseria gonorrhoeae* infection. Given increasing patterns of beta-lactamase mediated resistance, which of the following



options represents the current empiric structural class or drug of choice recommended for treating uncomplicated gonococcal urethritis?

- (A) Oral Amoxicillin
- (B) Intramuscular Ceftriaxone
- (C) Oral Doxycycline
- (D) Intravenous Aztreonam

**Q14.** A hospitalized patient receiving high-dose intravenous antibiotic therapy for a severe methicillin-resistant \*Staphylococcus aureus\* (MRSA) infection begins to complain of tinnitus and high-frequency hearing loss. Laboratory values also indicate a rising serum creatinine level. Which drug is most likely responsible for these toxicities?

- (A) Linezolid
- (B) Daptomycin
- (C) Vancomycin
- (D) Tigecycline

**Q15.** A 62-year-old diabetic female is treated for a complex urinary tract infection with a fluoroquinolone. While on this therapy, she must be advised about potential musculoskeletal adverse effects. What is the classic structural/connective tissue toxicity associated with fluoroquinolones like Ciprofloxacin?

- (A) Acute rhabdomyolysis
- (B) Tendonitis and Achilles tendon rupture
- (C) Osteonecrosis of the jaw
- (D) Drug-induced systemic lupus erythematosus (DILE)

**Q16.** A patient undergoing an intensive multi-drug regimen for pulmonary Tuberculosis complains of an inability to distinguish between red and green colors during a routine driving screening test. Which component of first-line



anti-tubercular therapy (ATT) is notorious for inducing this dose-dependent optic neuritis?

- (A) Isoniazid
- (B) Rifampicin
- (C) Pyrazinamide
- (D) Ethambutol

**Q17.** Macrolide antibiotics like Erythromycin and Clarithromycin inhibit bacterial protein synthesis by binding reversibly to the 50S ribosomal subunit. Which of the following mechanisms represents a major pathway of acquired plasmid-mediated bacterial resistance to this class?

- (A) Methylation of the 23S rRNA binding site
- (B) Acetylation of the antibiotic by an aminoglycoside acetyltransferase
- (C) Mutation in the RNA polymerase beta subunit
- (D) Downregulation of OmpF porin channels

**Q18.** A 55-year-old male with chronic heart failure and a history of gout is started on an optimal medical regimen. Weeks later, he presents with an excruciating, swollen, and red first metatarsophalangeal joint. A blood test confirms acute hyperuricemia. Which of the following cardiovascular drugs most likely precipitated this attack by competing with uric acid transporters in the proximal convoluted tubule?

- (A) Furosemide
- (B) Enalapril
- (C) Spironolactone
- (D) Carvedilol

**Q19.** A 44-year-old female presenting with profound tachycardia, heat intolerance, and exophthalmos is diagnosed with Graves' hyperthyroidism. She is started on antithyroid therapy. Which mechanism explains why Propylthiouracil



(PTU) is preferred over Methimazole during severe thyroid storm situations?

- (A) It prevents the structural synthesis of thyroglobulin molecules.
- (B) It directly blocks the follicular lumen sodium-iodide symporter (*NIS*).
- (C) It inhibits the peripheral conversion of thyroxine ( $T_4$ ) to triiodothyronine ( $T_3$ ).
- (D) It stimulates thyroid-stimulating hormone (TSH) degradation pathways.

**Q20.** A patient receiving an oncology chemotherapy protocol containing high-dose Cyclophosphamide for non-Hodgkin lymphoma develops gross hematuria accompanied by severe lower abdominal cramping. To minimize the cumulative bladder urothelial toxicity mediated by the metabolite acrolein, which adjuvant drug should be co-administered?

- (A) Amifostine
- (B) Leucovorin
- (C) Dexrazoxane
- (D) Mesna



**Detailed Solutions**

Q1.

**Solution****Concept:**

Pharmacokinetics dictates how a drug moves through various physiological compartments based on blood flow and tissue lipid solubility. A multi-compartment distribution model describes drugs that rapidly enter highly vascularized tissues before slowly transferring into less vascularized tissues. For highly lipophilic substances administered via an intravenous bolus, the termination of central nervous system activity depends heavily on this inter-compartmental movement rather than metabolic clearance.

**Solution:**

- (a) When an ultra-short-acting, highly lipid-soluble drug is injected as an intravenous bolus, it immediately enters the central compartment, which includes the blood volume and highly perfused organs such as the brain, heart, liver, and kidneys.
- (b) Due to its high lipid solubility and the high blood flow to the brain, the drug rapidly crosses the blood-brain barrier, achieving peak central nervous system concentrations and producing immediate anesthesia or sedation.
- (c) Subsequently, the drug begins to move along its concentration gradient from the central compartment into the peripheral compartment, which consists of moderately perfused tissues like skeletal muscle and poorly perfused tissues like adipose tissue.
- (d) This process, known as redistribution, rapidly lowers the concentration of the drug in the plasma and the brain. Consequently, the drug diffuses out of the brain back into the systemic circulation to fill the peripheral tissues.
- (e) Thiopental is the quintessential example of a drug whose rapid termination of action (within minutes) is entirely due to redistribution into muscle and fat rather than hepatic metabolism or renal excretion.

**Final Answer:** Thiopental**Answer: (B)**[Go Back to Question 1](#)

Q2.

**Solution****Concept:**

Receptor pharmacology analyzes how drugs interact with receptors to produce biological responses. Graded dose-response curves plot the log of the agonist concentration against the percentage of maximal response. This allows for the calculation of potency via the median effective concentration ( $EC_{50}$ ) and efficacy via the maximal response ( $E_{max}$ ). Competitive antagonists bind reversibly to the same active site as the endogenous agonist, creating a classic surmountable blockade that alters specific curve parameters.

**Solution:**

- (a) A competitive antagonist binds reversibly to the active site of the receptor, directly competing with the agonist for receptor occupancy. Because the binding is reversible, increasing the concentration of the agonist can overcome the inhibitory effect of the antagonist.
- (b) As the concentration of the competitive antagonist increases (moving from Curve A to Curve B and then Curve C), more agonist is required to achieve the same level of receptor occupancy and subsequent biological effect. This results in a parallel rightward shift of the log dose-response curve.
- (c) The parallel rightward shift signifies an increase in the  $EC_{50}$  value, which means the apparent potency of the agonist decreases in the presence of the antagonist.
- (d) Since the antagonism is completely surmountable at sufficiently high agonist concentrations, the maximal attainable response remains entirely unchanged. Therefore, the maximal efficacy ( $E_{max}$ ) of the agonist in Curve C is identical to that in Curve A.
- (e) The dissociation constant ( $K_d$ ) represents an intrinsic chemical property of the isolated agonist-receptor pair, but the presence of a competitor alters the apparent binding affinity, making efficacy the correct unchanged parameter.

**Final Answer:** Efficacy ( $E_{max}$ )

**Answer: (B)**

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Q3.

**Solution****Concept:**

Steady-state plasma concentration ( $C_{ss}$ ) represents the dynamic equilibrium where the rate of drug administration equals the rate of drug elimination. In first-order elimination kinetics, the clearance ( $CL$ ) of a drug is constant and independent of the dose. When a drug is cleared solely by renal glomerular filtration, its clearance is directly proportional to the patient's glomerular filtration rate (GFR). Alterations in renal physiology directly impact the systemic accumulation of such medications.

**Solution:**

- (a) The mathematical relationship governing the steady-state plasma concentration during continuous or regular intermittent dosing is defined by the equation where steady-state concentration equals the dosing rate divided by total body clearance.
- (b) Since the drug in this scenario is cleared exclusively via glomerular filtration, the total systemic clearance is functionally equivalent to the patient's operational glomerular filtration rate.
- (c) When the patient's functional GFR decreases by exactly 50%, the clearance of the drug is also halved because fewer functional nephrons are filtering the drug out of the plasma per unit of time.
- (d) Looking at the steady-state equation, clearance sits in the denominator. If the dosing rate and interval remain completely unaltered while the denominator is reduced by half, the resulting steady-state concentration must double.
- (e) A doubling of the plasma concentration corresponds mathematically to an increase of exactly 100% from the original baseline value, emphasizing why dosage adjustments are mandatory in renal failure.

**Final Answer:** Increased by 100%

**Answer: (D)**

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Q4.

**Solution****Concept:**

The receptor theory of pharmacology recognizes that many receptors exhibit intrinsic baseline activity even in the absolute absence of an explicit ligand. This phenomenon is known as constitutive activity. Ligands are classified based on their ability to alter this baseline equilibrium between the active conformation ( $R^*$ ) and the inactive conformation ( $R$ ) of the receptor complex. This distinction separates neutral antagonists from agents that suppress basal signaling.

**Solution:**

- (a) Receptors exist in a dynamic equilibrium between an inactive state and a spontaneously active state. In systems with high constitutive activity, significant downstream cellular signaling occurs without any agonist binding.
- (b) A conventional or full agonist binds preferentially to the active state, shifting the equilibrium toward the active conformation and maximizing the downstream biological response.
- (c) A neutral competitive antagonist binds equally to both the active and inactive states, preventing other ligands from binding but causing no shift in the baseline equilibrium or constitutive signaling of the cell.
- (d) An inverse agonist exhibits a selective binding affinity for the inactive conformation of the receptor. By binding to and stabilizing this inactive state, it drives the dynamic equilibrium away from the active state.
- (e) This stabilization effectively reduces the baseline constitutive activity of the receptor system below the resting level, producing a pharmacological response that is directionally opposite to that of a traditional agonist.

**Final Answer:** A drug that stabilizes the inactive conformation ( $R$ ) of a constitutively active receptor, reducing basal activity.

**Answer: (C)**

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Q5.

**Solution****Concept:**

Autonomic pharmacology relies on understanding the dominant resting tone of various organ systems. Preganglionic fibers of both the sympathetic and parasympathetic nervous systems release acetylcholine, which acts on nicotinic ( $N_N$ ) receptors located at autonomic ganglia. Ganglionic blockers interrupt neurotransmission at these sites, and the resulting physiological effect in any given organ is determined by which division of the autonomic nervous system exerts primary baseline control over that organ.

**Solution:**

- (a) Ganglionic blockers non-selectively antagonize nicotinic receptors at both sympathetic and parasympathetic ganglia, functionally isolating effector organs from central autonomic control and eliminating baseline autonomic tone.
- (b) In the cardiovascular system, arterioles and veins are dominated by sympathetic vasoconstrictor tone. Blocking this pathway removes the sympathetic restriction, leading to profound vasodilation, venous pooling, and severe orthostatic hypotension.
- (c) The sinoatrial node of the heart is dominated by parasympathetic vagal tone, which acts to maintain a low resting heart rate. Eliminating this parasympathetic brake results in a predictable increase in heart rate, known as tachycardia.
- (d) In the eye, the iris circular muscle is controlled by dominant parasympathetic tone. Ganglionic blockade prevents pupillary constriction, allowing the sympathetic system or passive relaxation to cause mydriasis rather than miosis.
- (e) Since gastrointestinal motility is dominated by the parasympathetic system, blocking the ganglia results in constipation and urinary retention, ruling out diarrhea, while sweat glands lose their sympathetic cholinergic activation, eliminating diaphoresis.

**Final Answer:** Orthostatic hypotension and tachycardia/mydriasis depending on dominant resting tone

**Answer: (C)**

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Q6.

**Solution****Concept:**

Ophthalmic pharmacology requires a careful evaluation of a patient's systemic comorbidities, as topically applied eye drops can undergo significant systemic absorption through the nasolacrimal duct. This absorption bypasses first-pass hepatic metabolism and can cause profound systemic adverse effects. Managing acute angle-closure glaucoma demands lowering intraocular pressure rapidly, but the medications chosen must not exacerbate underlying respiratory or cardiovascular conditions.

**Solution:**

- (a) Acute angle-closure glaucoma is an ocular emergency caused by an anatomical blockage of the trabecular meshwork, requiring immediate medical intervention to reduce the production of aqueous humor or enhance its outflow.
- (b) Timolol is a non-selective beta-adrenergic receptor antagonist commonly formulated as an eye drop to decrease aqueous humor production by blocking beta receptors on the ciliary epithelium.
- (c) When administered topically, timolol can drain through the nasolacrimal duct into the nasal mucosa, where it is rapidly absorbed directly into the systemic venous circulation.
- (d) In patients with severe bronchial asthma, systemic exposure to a non-selective beta-blocker leads to the blockade of  $\beta_2$ -adrenergic receptors on bronchial smooth muscle, triggering life-threatening bronchospasm.
- (e) While pilocarpine must be used cautiously in acute glaucoma, it does not carry the absolute systemic pulmonary contraindication that timolol does, and apraclonidine or latanoprost do not cause significant bronchoconstriction.

**Final Answer:** Timolol**Answer: (D)**[Go Back to Question 6](#)

Q7.

**Solution****Concept:**

Toxicology of organophosphate compounds involves the irreversible inhibition of the acetylcholinesterase enzyme via phosphorylation. This results in an excessive accumulation of acetylcholine at all cholinergic synapses, including muscarinic receptors on effector organs and nicotinic receptors at autonomic ganglia and the neuromuscular junction. Effective treatment strategies must address both receptor populations before the enzyme undergoes a chemical stabilization process known as aging.

**Solution:**

- (a) Organophosphate poisoning presents with a toxidrome characterized by overstimulation of muscarinic receptors (causing bradycardia, bronchorrhea, miosis, and diarrhea) and nicotinic receptors (causing muscle fasciculations and weakness).
- (b) Atropine is a highly effective competitive muscarinic receptor antagonist. It successfully reverses life-threatening muscarinic symptoms such as severe bradycardia and excessive bronchial secretions, but it has no activity at nicotinic receptors.
- (c) To reverse the nicotinic manifestations, such as skeletal muscle fasciculations and potential diaphragmatic paralysis, the phosphorylated acetylcholinesterase enzyme must be chemically reactivated.
- (d) Pralidoxime is a cholinesterase reactivator that binds to the organophosphate-enzyme complex. It exerts a nucleophilic attack on the phosphate group, pulling the toxin off the acetylcholinesterase molecule and restoring enzyme function.
- (e) Because pralidoxime restores the enzyme's ability to degrade acetylcholine, it successfully alleviates both muscarinic and nicotinic overstimulation, provided it is administered early before enzyme aging occurs.

**Final Answer:** Pralidoxime

**Answer: (B)**

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Q8.

**Solution****Concept:**

Beta-adrenergic blockers are categorized based on their cardioselectivity, membrane-stabilizing activity, and intrinsic sympathomimetic activity (ISA). Intrinsic sympathomimetic activity describes a drug's capacity to act as a partial agonist at the beta-receptor site. This unique property prevents excessive depression of resting heart rate, making these agents advantageous for patients who require beta-blockade but are highly susceptible to severe bradycardia.

**Solution:**

- (a) Beta-blockers with intrinsic sympathomimetic activity (ISA) bind to beta receptors and stimulate them weakly while simultaneously blocking the binding of more potent endogenous catecholamines like epinephrine and norepinephrine.
- (b) At rest, when sympathetic tone is low, the partial agonist activity provides a baseline level of receptor stimulation, preventing the profound bradycardia that typically occurs with standard, non-ISA beta-antagonists.
- (c) During exercise or stress, when sympathetic tone is naturally high, these drugs act as true antagonists, preventing excessive tachycardia and protecting the myocardium.
- (d) Acebutolol is a highly selective  $\beta_1$ -receptor antagonist that possesses both membrane-stabilizing activity and significant intrinsic sympathomimetic activity, fitting the precise clinical description required.
- (e) Metoprolol and atenolol are selective  $\beta_1$  blockers but lack ISA, whereas propranolol is a non-selective beta-blocker that lacks ISA and causes significant reductions in resting heart rate.

**Final Answer:** Acebutolol

**Answer:** (C)

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Q9.

**Solution****Concept:**

Teratogenicity is a critical consideration in neuropharmacology, as several antiepileptic drugs can cross the placental barrier and disrupt fetal development. Managing epilepsy during pregnancy requires balancing maternal seizure control with the risk of congenital malformations. Neural tube defects occur during early embryonic development due to alterations in folate metabolism or direct interference with cellular differentiation pathways.

**Solution:**

- (a) Valproic acid is a broad-spectrum antiepileptic drug that functions by blocking voltage-gated sodium channels, increasing gamma-aminobutyric acid (GABA) levels, and inhibiting T-type calcium channels.
- (b) Despite its high efficacy in managing generalized tonic-clonic seizures, valproic acid carries the highest risk of major congenital malformations among all commonly utilized anticonvulsant medications.
- (c) The primary mechanism of valproate-induced teratogenicity involves the inhibition of histone deacetylases and interference with maternal folate metabolism, which disrupts the closing of the neural tube.
- (d) Exposure to valproic acid during the first trimester of pregnancy is strongly associated with a ten-fold increase in the incidence of neural tube defects, such as spina bifida aperta, as well as distinct craniofacial dysmorphisms.
- (e) While carbamazepine also carries a mild risk of neural tube defects, valproic acid is statistically much more dangerous and should be strictly avoided in pregnant women unless no clinical alternative exists.

**Final Answer:** Valproic acid

**Answer: (B)**

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Q10.

**Solution****Concept:**

The management of Parkinson's disease focuses on restoring dopamine levels within the striatum while minimizing peripheral side effects and central complications like dyskinesias or hallucinations. Dopamine itself cannot cross the blood-brain barrier, so its immediate precursor, levodopa, is administered. Optimizing the duration of action of central dopamine involves inhibiting the specific enzymes responsible for its catabolism within the central nervous system.

**Solution:**

- (a) Levodopa is routinely co-administered with carbidopa, a peripheral dopa-decarboxylase inhibitor that prevents the premature conversion of levodopa to dopamine in systemic tissues, reducing nausea and cardiovascular side effects.
- (b) Once levodopa successfully crosses the blood-brain barrier, it is converted into active dopamine by central dopa-decarboxylase within surviving dopaminergic neurons.
- (c) Dopamine in the brain is primarily metabolized by two enzymatic pathways: Catechol-O-methyltransferase (*COMT*) and Monoamine Oxidase (*MAO*), specifically the MAO-B isoform which predominates in the striatum.
- (d) Selegiline is a potent, selective, and irreversible inhibitor of the Monoamine Oxidase B enzyme. By permanently inactivating this enzyme, selegiline prevents the breakdown of dopamine within the synaptic cleft.

**Final Answer:** Selective, irreversible inhibition of Monoamine Oxidase B (MAO-B)

**Answer: (C)**

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Q11.

**Solution****Concept:**

Opioid overdose toxicology centers on the overactivation of opioid receptors located throughout the central and peripheral nervous systems. The classical triad consists of respiratory depression, miosis, and a comatose state. Managing this life-threatening emergency requires immediate restoration of ventilatory drive using a fast-acting parenteral antagonist that can displace highly potent agonists from the targeted opioid receptor subpopulations.

**Solution:**

- (a) The patient demonstrates classic clinical features of severe opioid poisoning, specifically respiratory depression, extreme pupillary constriction, and unconsciousness resulting from exogenous ligand overstimulation.
- (b) Naloxone is a competitive opioid receptor antagonist that exhibits a very high binding affinity for mu-opioid receptors, as well as kappa and delta opioid receptors.
- (c) When administered intravenously, naloxone rapidly crosses the blood-brain barrier and competitively displaces the offending opioid molecules from the receptor binding sites.
- (d) This dynamic reversal restores the normal sensitivity of brainstem respiratory centers to carbon dioxide concentrations, effectively reversing respiratory depression and awakening the patient.
- (e) In individuals with severe physical dependence on opioids, this immediate clearance of receptor activation precipitously unmasks the underlying neuroadaptations, resulting in the acute onset of a severe withdrawal syndrome.

**Final Answer:** Competitive antagonist at  $\mu$ -opioid receptors

**Answer: (B)**

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Q12.

**Solution****Concept:**

Atypical or second-generation antipsychotics are distinguished by their complex receptor-binding profiles, which characteristically involve combined serotonin-dopamine antagonism. While these agents offer a significant clinical advantage by drastically reducing the incidence of extrapyramidal motor side effects, they exhibit highly varied metabolic profiles. Certain medications within this class can cause profound metabolic dysregulation through distinct central mechanisms.

**Solution:**

- (a) Second-generation antipsychotics minimize extrapyramidal symptoms due to their lower binding affinity for dopamine D2 receptors and concurrent blockade of serotonin 5-HT<sub>2A</sub> receptors.
- (b) However, various molecules within this structural class interact strongly with histamine H<sub>1</sub> and serotonin 5-HT<sub>2C</sub> receptors, which are responsible for regulating hypothalamic appetite signaling.
- (c) Olanzapine exhibits an exceptionally high binding affinity for these specific histaminergic and serotonergic receptors, which leads to intense, unremitting hyperphagia and substantial weight gain.
- (d) Beyond weight gain, olanzapine directly interferes with peripheral insulin sensitivity and alters adipokine secretion patterns, which accelerates the development of profound hypertriglyceridemia and metabolic syndrome.
- (e) In direct contrast, agents such as ziprasidone and aripiprazole exhibit minimal affinity for these metabolic pathways, while traditional haloperidol induces high extrapyramidal toxicity instead.

**Final Answer:** Olanzapine

**Answer:** (C)

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Q13.

**Solution****Concept:**

Antimicrobial selection for gonococcal infections must account for the rapid evolution of plasmid-mediated and chromosomal beta-lactamase resistance mechanisms. Pathogens such as *Neisseria gonorrhoeae* have developed robust mechanisms to render traditional penicillins ineffective. Modern management algorithms rely on advanced generation cephalosporins that exhibit high stability against bacterial enzymatic degradation.

**Solution:**

- (a) *Neisseria gonorrhoeae* isolates globally have acquired high levels of resistance to traditional agents like amoxicillin via the production of plasmid-encoded TEM-1 beta-lactamase enzymes.
- (b) Due to widespread resistance and treatment failures, international guidelines recommend a highly effective, structurally stable bactericidal agent that can overcome these beta-lactamase defenses.
- (c) Ceftriaxone is a broad-spectrum, third-generation cephalosporin that demonstrates exceptional stability against the beta-lactamases produced by these Gram-negative diplococci.
- (d) It acts by binding irreversibly to penicillin-binding proteins, thereby disrupting bacterial cell wall synthesis and causing rapid bacterial cell lysis.
- (e) A single intramuscular injection of ceftriaxone achieves prolonged therapeutic concentrations in mucosal tissues, making it the definitive first-line recommendation for treating uncomplicated gonococcal urethritis.

**Final Answer:** Intramuscular Ceftriaxone

**Answer: (B)**

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Q14.

**Solution****Concept:**

Glycopeptide antibiotics are critical for treating multi-drug resistant Gram-positive infections, including methicillin-resistant *Staphylococcus aureus* (MRSA). However, their clinical utility is limited by a narrow therapeutic index. Tracking serum trough concentrations is essential to minimize systemic toxicity, particularly in renal structures and specialized sensory organs where drug accumulation can cause permanent functional damage.

**Solution:**

- (a) Vancomycin is a glycopeptide antibiotic that selectively inhibits bacterial cell wall synthesis by binding directly to the D-alanyl-D-alanine terminus of cell wall peptidoglycan precursors.
- (b) It serves as a cornerstone therapy for severe, invasive infections caused by MRSA, but prolonged high-dose therapy can lead to significant organ-specific toxicities.
- (c) Nephrotoxicity occurs due to drug accumulation within proximal renal tubule cells, leading to cellular necrosis and causing a progressive rise in serum creatinine values.
- (d) Concurrently, vancomycin can exert direct ototoxic effects on the auditory system, damaging the delicate hair cells within the cochlea and presenting clinically as tinnitus and high-frequency hearing loss.
- (e) While linezolid can cause bone marrow suppression and daptomycin can induce severe myopathy, vancomycin is uniquely associated with this specific presentation of concurrent nephrotoxicity and ototoxicity.

**Final Answer:** Vancomycin

**Answer:** (C)

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Q15.

**Solution****Concept:**

Fluoroquinolones are highly effective, broad-spectrum bactericidal agents that target bacterial topoisomerases. Despite their excellent tissue penetration and bioavailability, they are associated with distinct, class-specific connective tissue toxicities. These adverse effects result from the chelation of divalent cations and the upregulation of specific matrix-degrading enzymes within dense musculoskeletal structures.

**Solution:**

- (a) Fluoroquinolones like ciprofloxacin function by inhibiting bacterial DNA gyrase and topoisomerase IV, preventing the normal replication and transcription of bacterial genetic material.
- (b) Beyond their antimicrobial activity, fluoroquinolones exert detrimental effects on mammalian connective tissue structures by chelating magnesium ions and disrupting normal tenocyte metabolism.
- (c) This chelation leads to a direct reduction in collagen synthesis and triggers a localized upregulation of matrix metalloproteinases within tendinous structures.
- (d) The resulting degradation weakens the structural integrity of dense connective tissues, predisposing patients to painful tendonitis and acute, spontaneous Achilles tendon rupture.
- (e) This risk is significantly elevated in older adults, individuals with concurrent corticosteroid use, and patients with underlying renal impairment, necessitating careful patient education.

**Final Answer:** Tendonitis and Achilles tendon rupture

**Answer: (B)**

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Q16.

**Solution****Concept:**

First-line anti-tubercular therapy (ATT) involves a combination of specialized medications designed to prevent the emergence of drug resistance. Each agent within this standard regimen possesses a distinct mechanism of action and a characteristic side-effect profile. Successful clinical management requires regular screening for organ-specific toxicities, particularly those affecting sensory systems and metabolic pathways.

**Solution:**

- (a) The standard first-line regimen for treating pulmonary tuberculosis consists of isoniazid, rifampicin, pyrazinamide, and ethambutol, with each drug targeting different aspects of mycobacterial replication.
- (b) Ethambutol functions by inhibiting the mycobacterial enzyme arabinosyl transferase, which disrupts the polymerization of arabinogalactan and impairs cell wall synthesis.
- (c) A well-documented, dose-dependent adverse effect of ethambutol is the development of retrobulbar optic neuritis, which causes a progressive decrease in visual acuity.
- (d) This inflammatory condition selectively damages macular fibers, leading to a characteristic impairment in color vision, specifically rendering patients unable to distinguish between red and green.
- (e) Regular visual acuity and color discrimination assessments are mandatory for patients on ethambutol therapy to detect early signs of optic nerve toxicity and prevent permanent visual loss.

**Final Answer:** Ethambutol

**Answer: (D)**

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Q17.

**Solution****Concept:**

Bacterial resistance to protein synthesis inhibitors often involves structural modifications of the ribosomal target site. Macrolides, lincosamides, and streptogramins share overlapping binding regions on the large ribosomal subunit. Plasmid-mediated acquisition of specific methyltransferase enzymes can alter these target sites, reducing drug affinity and leading to cross-resistance across multiple antibiotic classes.

**Solution:**

- (a) Macrolides like erythromycin exert their antimicrobial action by binding reversibly to the 50S ribosomal subunit, specifically interacting with the 23S ribosomal RNA component.
- (b) This binding physically obstructs the translocation step during protein synthesis, preventing the elongation of the growing polypeptide chain and exerting a bacteriostatic effect.
- (c) A primary mechanism of acquired resistance involves the horizontal transfer of plasmid-borne erm genes, which encode specialized RNA methyltransferase enzymes.
- (d) These enzymes catalyze the post-transcriptional methylation of a specific adenine residue within the 23S rRNA structure of the 50S subunit.
- (e) This targeted structural modification sterically hinders the macrolide molecule from binding to the ribosome, completely neutralizing its inhibitory effects while preserving normal bacterial translation functions.

**Final Answer:** Methylation of the 23S rRNA binding site

**Answer: (A)**

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Q18.

**Solution****Concept:**

Diuretic therapies can significantly alter the renal handling of metabolic waste products, including uric acid. The proximal convoluted tubule utilizes specialized transport proteins to regulate the filtration, reabsorption, and secretion of organic anions. Medications that compete for these organic anion transporters can impair uric acid clearance, raising systemic levels and triggering acute crystalline arthropathies.

**Solution:**

- (a) Loop diuretics such as furosemide are highly effective agents used to manage volume overload in patients suffering from chronic congestive heart failure.
- (b) Furosemide undergoes active secretion into the proximal convoluted tubule via the organic anion transporting polypeptide pathway to reach its site of action in the loop of Henle.
- (c) This active secretion process directly competes with the renal elimination of endogenous uric acid, leading to a significant reduction in net urate excretion.
- (d) Additionally, the resulting volume depletion enhances the compensatory reabsorption of sodium and fluid in the proximal tubule, which indirectly promotes concurrent urate reabsorption.
- (e) The resulting hyperuricemia causes monosodium urate crystals to precipitate within peripheral joints, presenting clinically as an acute, painful gouty flare in the first metatarsophalangeal joint.

**Final Answer:** Furosemide**Answer: (A)**[Go Back to Question 18](#)

Q19.

**Solution****Concept:**

Thionamide antithyroid medications are used to suppress thyroid hormone synthesis by inhibiting key enzymatic processes within thyroid follicular cells. In life-threatening thyrotoxic crises, such as a thyroid storm, immediate management requires blocking both the synthesis of new hormone and the peripheral conversion of circulating prohormones into their highly active metabolic forms.

**Solution:**

- (a) Both propylthiouracil and methimazole lower thyroid hormone levels by inhibiting the thyroid peroxidase enzyme, preventing the iodination of tyrosine residues on thyroglobulin.
- (b) However, in a severe thyroid storm, large quantities of thyroxine ( $T_4$ ) are already circulating in the bloodstream and contributing to hypermetabolic symptoms.
- (c) Propylthiouracil possesses a distinct secondary pharmacological effect: it inhibits the peripheral 5'-deiodinase enzyme located in peripheral tissues.
- (d) This peripheral enzyme is responsible for converting the relatively inactive prohormone  $T_4$  into triiodothyronine ( $T_3$ ), which is the highly active form responsible for metabolic overstimulation.
- (e) By rapidly suppressing this peripheral conversion, propylthiouracil reduces active  $T_3$  levels faster than methimazole, making it the preferred thionamide for managing acute thyrotoxic emergencies.

**Final Answer:** It inhibits the peripheral conversion of thyroxine ( $T_4$ ) to triiodothyronine ( $T_3$ ).

**Answer: (C)**

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Q20.

**Solution****Concept:**

The metabolic breakdown of certain alkylating chemotherapeutic agents can generate toxic secondary metabolites. When these reactive byproducts accumulate in the urinary tract, they can cause significant urothelium injury and hemorrhagic cystitis. Preventing this systemic toxicity requires the co-administration of a chemoprotective sulfhydryl compound that selectively neutralizes the toxic metabolite within the bladder.

**Solution:**

- (a) Cyclophosphamide is an alkylating agent that undergoes hepatic biotransformation to produce its active antineoplastic components, along with a toxic byproduct known as acrolein.
- (b) Acrolein is excreted by the kidneys and concentrates in the urinary bladder, where it causes direct mucosal irritation, inflammation, and severe hemorrhagic cystitis.
- (c) Mesna (2-mercaptoethane sulfonate) is a synthetic sulfhydryl compound administered concurrently with high-dose cyclophosphamide protocols to provide targeted uroprotection.
- (d) Once filtered into the urine, the free thiol groups of mesna bind directly to the electrophilic acrolein molecule, forming a stable, non-toxic, and water-soluble conjugate.
- (e) This highly localized chemical neutralization prevents acrolein from interacting with the bladder wall, effectively eliminating the risk of bleeding and ulceration without compromising chemotherapeutic efficacy.

**Final Answer:** Mesna

**Answer: (D)**

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**Answer Key**

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

