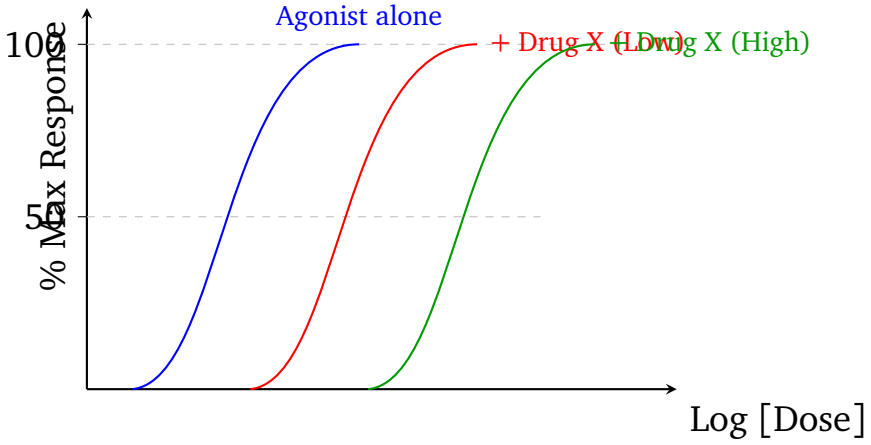
NEET PG Pharmacology Sample Paper-9

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 new investigational drug X is tested in an in vitro tissue preparation. The log dose-response curve of an agonist alone and in the presence of two different concentrations of drug X is shown below:



Based on this pharmacological profile, which of the following best describes the nature of drug X?

(A) Non-competitive antagonist

(B) Competitive antagonist

(C) Irreversible antagonist

(D) Functional antagonist

Q2. An elderly male patient is prescribed a drug that undergoes extensive Phase II metabolism via acetylation. Genetic testing reveals that the patient is a "slow acetylator." Which of the following adverse outcomes is this patient at a significantly higher risk of developing if treated with hydralazine or procainamide?

(A) Acute hepatic necrosis

(B) Drug-induced lupus erythematosus

(C) Hemolytic anemia due to G6PD deficiency

(D) Pseudomembranous colitis

Q3. A clinical trial evaluates a new drug's elimination kinetics. It is observed that a constant fraction of the drug is eliminated per unit of time, regardless of its plasma concentration. Which of the following parameters remains constant for this drug across therapeutic doses?

(A) Rate of elimination

(B) Clearance

(C) Half-life ($t_{1/2}$)

(D) Both Clearance and Half-life ($t_{1/2}$)

Q4. Which of the following transport mechanisms across cell membranes is characterized by saturability, selectivity, movement along a concentration gradient, and a lack of requirement for high-energy phosphate bonds (ATP)?

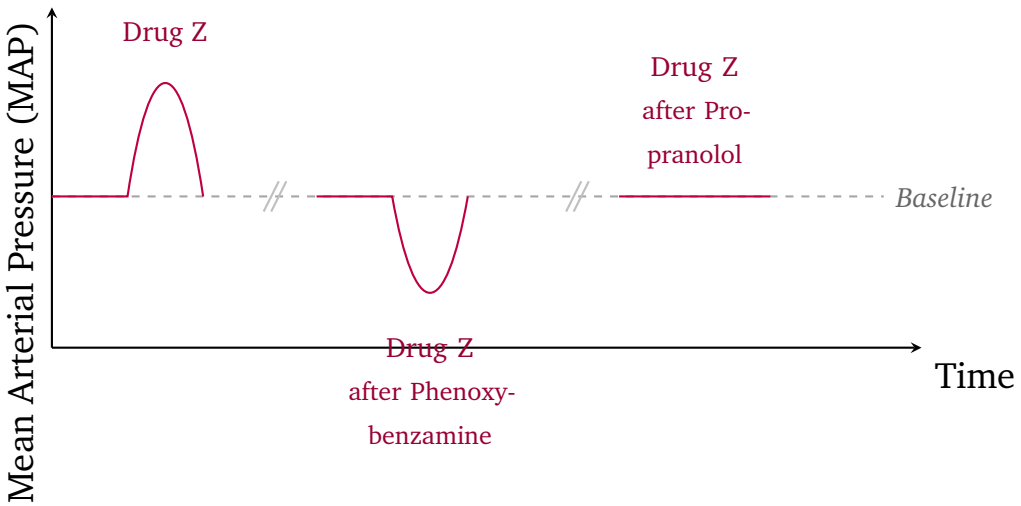
(A) Simple diffusion

(B) Primary active transport

(C) Facilitated diffusion

(D) Secondary active co-transport

Q5. An experimental drug is administered intravenously to an anesthetized animal model while monitoring mean arterial pressure (MAP). The cardiovascular tracing showing responses before and after the administration of specific blockers is represented below:



Which of the following endogenous substances best matches the pharmacodynamic properties of Drug Z?

(A) Norepinephrine

(B) Epinephrine

(C) Isoproterenol

(D) Phenylephrine

Q6. A 45-year-old patient diagnosed with open-angle glaucoma is prescribed a topical ophthalmic medication that acts as a selective α_2-adrenergic receptor agonist. Which of the following medications matches this description and works by reducing aqueous humor production while increasing uveoscleral outflow?

(A) Timolol

(B) Pilocarpine

(C) Brimonidine

(D) Latanoprost

Q7. A patient presents with severe organophosphate poisoning following accidental pesticide exposure. Along with aggressive airway management, standard pharmacological therapy is initiated. Which of the following drugs acts as a specific antidote by reactivating the phosphorylated acetylcholinesterase enzyme, provided it is administered before enzyme aging occurs?

(A) Atropine

(B) Pralidoxime (2-PAM)

(C) Neostigmine

(D) Physostigmine

Q8. A 62-year-old male with benign prostatic hyperplasia (BPH) and comorbid hypertension is started on an oral medication that provides therapeutic benefits for both conditions by selectively blocking post-synaptic α_1-adrenergic receptors. Which drug was most likely prescribed?

(A) Prazosin

(B) Clonidine

(C) Propranolol

(D) Tamsulosin

Q9. A 34-year-old pregnant woman in her third trimester requires pharmacological management for a severe depressive episode. Which of the following antidepressant classes is generally preferred due to a favorable safety profile, but carries a small, specific risk of causing persistent pulmonary hypertension of the newborn (PPHN)?

(A) Tricyclic Antidepressants (TCAs)

(B) Monoamine Oxidase Inhibitors (MAOIs)

(C) Selective Serotonin Reuptake Inhibitors (SSRIs)

(D) Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs)

Q10. A patient undergoing long-term therapy for schizophrenia develops severe, involuntary, repetitive movements of the tongue, face, and jaw after 3 years of treatment with Haloperidol. This condition is diagnosed as tardive dyskinesia. What is the primary underlying pathophysiology responsible for this adverse effect?

(A) Acute blockade of D_2 receptors in the nigrostriatal pathway

(B) Upregulation and supersensitivity of D_2 receptors in the striatum

(C) Cholinergic excess in the basal ganglia

(D) Serotonergic depletion in the frontal cortex

Q11. A 28-year-old individual is brought to the emergency department experiencing severe respiratory depression, pinpoint pupils (miosis), and bradycardia. An opioid overdose is suspected. Which of the following agents should be administered immediately as a pure opioid receptor antagonist with a high affinity for μ receptors?

(A) Methadone

(B) Buprenorphine

(C) Naloxone

(D) Tramadol

Q12. An epileptic patient who has been well-controlled on a stable dose of Phenytoin is started on an active course of treatment for another acute medical condition. Two weeks later, the patient presents with symptoms of Phenytoin toxicity, including nystagmus and ataxia. Which of the following co-administered drugs most likely caused this by inhibiting hepatic CYP2C9/2C19 enzymes?

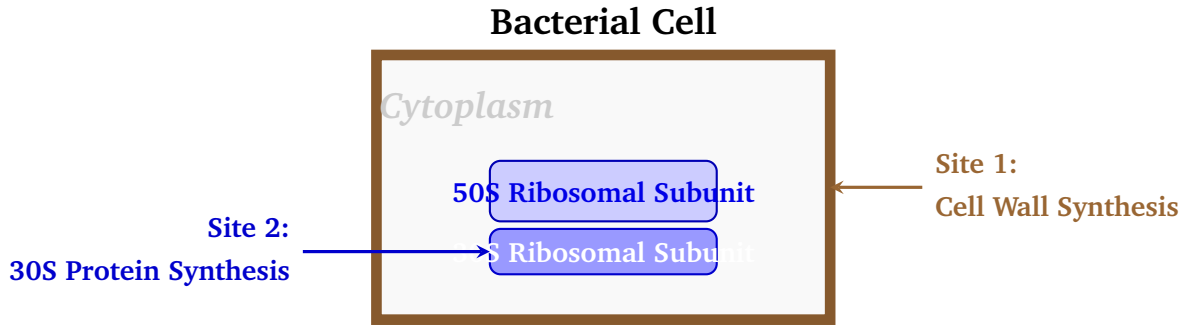
(A) Rifampin

(B) Carbamazepine

(C) Cimetidine

(D) Phenobarbital

Q13. The simplified schematic diagram below outlines a bacterial cell and the primary subcellular molecular sites of action for various antimicrobial agents:



Which of the following pairs of antibiotics correctly corresponds to an inhibitor acting at Site 1 and Site 2 respectively?

(A) Azithromycin; Ciprofloxacin

(B) Ceftriaxone; Gentamicin

(C) Vancomycin; Linezolid

(D) Amoxicillin; Clindamycin

Q14. A 24-year-old female patient presents with a uncomplicated lower urinary tract infection (cystitis). She is prescribed a single-dose oral antibiotic that works by inactivating the enzyme UDP-N-acetylglucosamine enolpyruvyl transferase (MurA), thereby blocking the first step of bacterial peptidoglycan synthesis. Which antibiotic was selected?

(A) Nitrofurantoin

(B) Fosfomycin

(C) Trimethoprim

(D) Cephalexin

Q15. A patient undergoing intensive multi-drug therapy for pulmonary tuberculosis complains of a progressive, bilateral decrease in visual acuity along with an inability to distinguish between the colors red and green. Which of the following anti-tubercular medications is notorious for causing this dose-dependent optic neuritis?

(A) Isoniazid

(B) Rifampin

(C) Ethambutol

(D) Pyrazinamide

Q16. A 40-year-old HIV-positive patient is initiated on highly active antiretroviral therapy (HAART). The regimen includes an integrated viral enzyme inhibitor that specifically prevents the covalent insertion of HIV genomic DNA into

the host cell chromosome. Which of the following drugs represents this mechanism of action?

(A) Efavirenz

(B) Dolutegravir

(C) Atazanavir

(D) Maraviroc

Q17. A clinical culture isolates a strain of Pseudomonas aeruginosa showing high-level resistance to Piperacillin. The laboratory determines that resistance is mediated by the production of an extended-spectrum β-lactamase enzyme. Which of the following agents can be combined with β-lactam antibiotics to physically shield them from hydrolytic inactivation by this enzyme?

(A) Cilastatin

(B) Tazobactam

(C) Probenecid

(D) Avibactam only

Q18. A 55-year-old patient presenting with chronic heart failure with reduced ejection fraction (HFrEF) is prescribed an oral medication combination consisting of a neprilysin inhibitor (Sacubitril) and an angiotensin receptor blocker (Valsartan). What is the primary physiological benefit achieved by inhibiting the neprilysin enzyme in this patient?

(A) Decreasing the degradation of endogenous natriuretic peptides

(B) Selectively blocking the synthesis of aldosterone in the adrenal cortex

(C) Directly reducing the heart rate via hyperpolarization-activated cyclic nucleotide-gated channels

(D) Decreasing the breakdown of Angiotensin II

Q19. A 48-year-old male type 2 diabetic patient with established atherosclerotic cardiovascular disease requires an additional glucose-lowering agent. The physician selects a drug that acts on the proximal convoluted tubules of the

kidney to inhibit glucose reabsorption, leading to glucosuria and providing proven cardioprotective benefits. Which class does this drug belong to?

(A) GLP-1 receptor agonists

(B) DPP-4 inhibitors

(C) SGLT2 inhibitors

(D) Sulfonylureas

Q20. A patient diagnosed with choriocarcinoma is initiated on systemic chemotherapy with high-dose Methotrexate. To protect healthy host tissues from severe bone marrow toxicity and mucosal ulcerations, a rescue agent is administered exactly 24 hours later. This rescue agent bypasses the blocked dihydrofolate reductase enzyme. What is this agent?

(A) Leucovorin (Folinic acid)

(B) Filgrastim (G-CSF)

(C) Mesna

(D) Dexrazoxane

Detailed Solutions

Q1.

Solution

Concept:

In pharmacodynamics, receptor antagonism describes how a drug reduces or blocks the effect of an agonist. Antagonists are broadly classified into competitive and non-competitive types based on whether they bind to the same active site as the agonist and whether their inhibitory effect can be overcome by increasing the agonist concentration.

Solution:

(a) The provided log dose-response curve illustrates the effect of an agonist alone and in the presence of increasing concentrations of drug X.

(b) As the concentration of drug X increases from low to high, the curve undergoes a parallel rightward shift along the log dose axis.

(c) Crucially, the maximal response (the plateau of the curve) remains entirely unchanged across all three curves, indicating that the efficacy of the agonist is fully preserved.

(d) This pattern is highly characteristic of a reversible competitive antagonist. Because it competes for the exact same binding site as the agonist, high concentrations of the agonist can displace the antagonist and achieve the maximum possible biological response.

(e) Consequently, a higher concentration of the agonist is required to produce the same level of response, which increases the median effective dose (ED_{50}) without decreasing the maximal efficacy.

Final Answer: The nature of drug X is a competitive antagonist.

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept:

Pharmacogenetics deeply influences xenobiotic metabolism, particularly during Phase II conjugation reactions. Acetylation is a primary pathway mediated by the hepatic enzyme N-acetyltransferase-2 (NAT2). Genetic polymorphisms divide the human population into rapid and slow acetylators, significantly impacting drug clearance and toxicity profiles.

Solution:

(a) The patient is identified as a slow acetylator, meaning they possess an inherited deficiency or low activity of the NAT2 enzyme.

(b) When treated with medications like hydralazine, procainamide, or isoniazid, slow acetylators cannot efficiently clear these compounds via the normal acetylation pathway.

(c) As a direct result, these drugs accumulate in the body and are shunted toward alternative oxidative metabolic pathways, leading to the formation of reactive metabolites.

(d) These reactive metabolic intermediates interact with host proteins and nuclear components, triggering an autoimmune response characterized by the production of anti-histone antibodies.

(e) This clinical syndrome manifests as drug-induced lupus erythematosus, featuring symptoms like arthralgia, myalgia, fever, and serositis, which typically resolve upon discontinuing the offending drug.

Final Answer: The adverse outcome is drug-induced lupus erythematosus.

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept:

Pharmacokinetics deals with the quantitative processes of drug absorption, distribution, metabolism, and excretion. The elimination of drugs generally follows either first-order or zero-order kinetics, depending on whether the eliminating mechanisms and metabolic enzymes are operating below or at their saturation threshold.

Solution:

(a) The prompt states that a constant fraction of the drug is eliminated per unit of time, which is the defining characteristic of first-order elimination kinetics.

(b) In first-order kinetics, the actual rate of elimination is directly proportional to the plasma concentration of the drug, meaning it changes as the concentration changes.

(c) However, intrinsic clearance (the volume of plasma cleared of the drug per unit time) and the elimination half-life remain completely constant and independent of the dose.

(d) Half-life is mathematically related to clearance and the volume of distribution by a constant ratio, ensuring that the time taken for the plasma concentration to decline by fifty percent does not fluctuate within the therapeutic window.

(e) Therefore, both clearance and half-life remain constant parameters across therapeutic doses under first-order elimination mechanisms.

Final Answer: Both Clearance and Half-life remain constant.

Answer: (D)

Go Back to Question 3

Q4.

Solution

Concept:

The cellular membrane serves as a semi-permeable barrier regulating the entry and exit of molecules. Transport mechanisms are categorized based on their energetic requirements and whether they utilize specialized transmembrane proteins to facilitate the movement of specific chemical substrates.

Solution:

(a) Passive transport mechanisms move solutes down their electrochemical or concentration gradients without requiring metabolic energy in the form of adenosine triphosphate (ATP) hydrolysis.

(b) Simple diffusion allows small, non-polar molecules to pass directly through the lipid bilayer, which lacks saturability or high chemical selectivity.

(c) Facilitated diffusion, however, relies on specialized transmembrane carrier proteins or channels to transport larger or polar molecules across the membrane.

(d) Because it depends on a finite number of transport proteins, facilitated diffusion exhibits clear chemical selectivity, competitive inhibition by structural analogs, and saturability at high substrate levels.

(e) Since the driving force is purely the concentration gradient, it does not require cellular energy expenditure, distinguishing it completely from primary or secondary active transport processes.

Final Answer: The transport mechanism is facilitated diffusion.

Answer: (C)

Go Back to Question 4

Q5.

Solution

Concept:

Autonomic pharmacology relies heavily on understanding receptor selectivity and systemic cardiovascular interactions. The classic epinephrine reversal experiment, originally described by Dale, demonstrates how blocking specific receptor types can unmask the opposing physiological effects of a non-selective adrenergic agonist.

Solution:

(a) Drug Z initially causes a marked increase in mean arterial pressure, suggesting activation of alpha-1 adrenergic receptors on vascular smooth muscle, which induces vasoconstriction.

(b) When the non-selective alpha blocker phenoxybenzamine is introduced, the alpha-1 mediated vasoconstriction is completely prevented.

(c) Re-administering Drug Z now causes a net drop in mean arterial pressure below the baseline level, revealing unmasked beta-2 adrenergic receptor activation, which causes vasodilation.

(d) When the beta blocker propranolol is subsequently added, this depressor effect is eliminated, confirming that the vasodilation was mediated through beta receptors.

(e) This unique triphasic response matches epinephrine, which possesses high affinity for both alpha and beta receptors, unlike norepinephrine or phenylephrine, which lack significant beta-2 mediated vasodilatory capacity.

Final Answer: The endogenous substance is Epinephrine.

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept:

The management of open-angle glaucoma focuses on reducing intraocular pressure by altering the dynamics of aqueous humor within the anterior chamber of the eye. This is achieved either by decreasing its production from the ciliary epithelium or by accelerating its drainage through conventional or unconventional outflow pathways.

Solution:

(a) The ciliary body contains adrenergic receptors that modulate the secretion of aqueous humor, where beta-blockers decrease production and alpha agonists modulate both production and drainage.

(b) Brimonidine is a highly selective alpha-2 adrenergic receptor agonist formulated for topical ophthalmic application in glaucoma management.

(c) By stimulating presynaptic alpha-2 receptors, it decreases local norepinephrine release, leading to a down-regulation of cyclic adenosine monophosphate production in the ciliary body and reducing fluid secretion.

(d) Additionally, brimonidine enhances uveoscleral outflow, which is the unconventional drainage pathway of the eye, by inducing prostaglandins that remodel the extracellular matrix.

(e) Timolol only decreases production, pilocarpine increases trabecular outflow via miotic action, and latanoprost primarily enhances uveoscleral drainage without suppressing aqueous production.

Final Answer: The medication is Brimonidine.

Answer: (C)

Go Back to Question 6

Q7.

Solution

Concept:

Organophosphate compounds found in insecticides bind covalently to the active site of the enzyme acetylcholinesterase. This creates a stable phosphorylated complex that stops the breakdown of acetylcholine, leading to an acute cholinergic crisis throughout the central and peripheral nervous systems.

Solution:

(a) The management of organophosphate toxicity involves counteracting excess acetylcholine and attempting to restore normal acetylcholinesterase enzyme functionality.

(b) Atropine is a competitive muscarinic receptor antagonist that blocks the clinical signs of cholinergic excess but does not fix or regenerate the damaged enzyme.

(c) Pralidoxime belongs to the oxime class of medications and acts as a specific chemical antidote by targeting the phosphorylated site of the bound enzyme.

(d) It exerts a nucleophilic attack on the phosphate group, pulling the organophosphate residue away and reactivating the functional acetylcholinesterase enzyme.

(e) This reactivation must happen before enzyme aging occurs, a chemical process where the phosphorylated enzyme loses an alkyl group and becomes permanently irreversible.

Final Answer: The specific antidote is Pralidoxime.

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept:

Smooth muscle tone in both vascular walls and the urinary tract is modulated by adrenergic receptors. Selective antagonism of these receptors can alter systemic blood pressure while simultaneously modifying resistance to urine flow through the prostatic urethra.

Solution:

(a) Post-synaptic alpha-1 adrenergic receptors are located on vascular smooth muscle where they mediate vasoconstriction, and on the smooth muscle of the bladder neck and prostate where they mediate contraction.

(b) Prazosin is a selective, competitive antagonist of post-synaptic alpha-1 receptors that causes systemic vasodilation, making it an effective option for lowering elevated blood pressure.

(c) Simultaneously, by relaxing the smooth muscle capsule of the prostate and bladder neck, it decreases resistance to urinary flow, providing symptomatic relief from benign prostatic hyperplasia.

(d) While tamsulosin is selective for the alpha-1A subtype found mainly in the prostate, it has minimal impact on systemic blood pressure and does not treat hypertension.

(e) Clonidine acts centrally as an agonist, whereas propranolol is a non-selective beta-blocker that lacks direct prostate-relaxing capabilities.

Final Answer: The drug prescribed is Prazosin.

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept:

Treating psychiatric disorders during pregnancy requires evaluating the therapeutic benefits to the mother against potential congenital or neonatal safety risks. Transplacental transfer of medications can alter fetal physiology or cause neonatal adaptation syndromes during late gestation.

Solution:

(a) Selective Serotonin Reuptake Inhibitors (SSRIs) are commonly prescribed for major depressive disorders due to their favorable safety profile and low risk of sedation or cardiac toxicity.

(b) When used during late pregnancy, epidemiological data indicates a small but serious association with persistent pulmonary hypertension of the newborn (PPHN).

(c) This condition happens because elevated fetal serotonin levels may interfere with normal pulmonary vascular remodeling or delay the transition from fetal to neonatal circulation.

(d) Consequently, the newborn experiences high pulmonary vascular resistance, causing severe hypoxemia and right-to-left shunting through fetal shunts.

(e) Despite this risk, SSRIs remain preferred over tricyclic antidepressants and monoamine oxidase inhibitors, which carry higher risks of teratogenicity and maternal hypertensive emergencies.

Final Answer: The class is Selective Serotonin Reuptake Inhibitors.

Answer: (C)

Go Back to Question 9

Q10.

Solution

Concept:

Antipsychotic medications work by blocking dopamine receptors, but long-term use can alter receptor density and sensitivity in the basal ganglia. These secondary neurochemical adaptations can manifest as late-onset extrapyramidal movement disorders.

Solution:

(a) Haloperidol is a high-potency typical antipsychotic that acts as a strong antagonist at dopamine D2 receptors within the central nervous system.

(b) Prolonged, continuous blockade of these D2 receptors in the striatum forces the postsynaptic neurons to compensate for the lack of dopamine signaling.

(c) The brain adapts by upregulating the total number of D2 receptors and increasing their overall baseline sensitivity to any available dopamine.

(d) This state of dopamine receptor supersensitivity causes an exaggerated neurochemical response, manifesting clinically as tardive dyskinesia.

(e) Characterized by involuntary, repetitive movements of the face, mouth, and extremities, this condition can persist even after reducing or stopping the causative antipsychotic medication.

Final Answer: The mechanism is upregulation and supersensitivity of D2 receptors.

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept:

Opioid toxidrome is a life-threatening medical emergency caused by the overstimulation of opioid receptors within the central nervous system. Immediate reversal requires the deployment of a high-affinity antagonist capable of displacing potent agonists from receptor sites to restore baseline respiratory drive.

Solution:

(a) The clinical presentation of severe respiratory depression, objective pinpoint pupils, and bradycardia forms the classic triad of acute opioid overdose.

(b) Naloxone is a pure, competitive opioid receptor antagonist with no intrinsic agonist activity or efficacy of its own.

(c) It exhibits an exceptionally high binding affinity for mu-opioid receptors, allowing it to rapidly displace exogenous opioids from these binding sites.

(d) Upon administration, naloxone reverses opioid-induced respiratory depression and sedation, typically restoring spontaneous ventilation within minutes.

(e) Due to its short duration of action relative to many long-acting opioids, repeated doses or continuous intravenous infusions may be required to prevent recurrent respiratory failure.

(f) Options like methadone and buprenorphine act as agonists or partial agonists used in maintenance therapy, while tramadol is a weak agonist that would worsen toxicity.

Final Answer: The agent that should be administered immediately is Naloxone.

Answer: (C)

Go Back to Question 11

Q12.

Solution

Concept:

Hepatic cytochrome P450 enzymes are responsible for the metabolic clearance of most anti-convulsant drugs. Drug-drug interactions frequently occur when a concurrently administered agent modulates the enzymatic activity of these proteins, causing unexpected alterations in the serum concentration of drugs with narrow therapeutic indices.

Solution:

(a) Phenytoin is an antiepileptic drug characterized by zero-order elimination kinetics at therapeutic concentrations, making its serum levels highly sensitive to minor changes in metabolism.

(b) The primary metabolic pathway for phenytoin involves hydroxylation via the hepatic microsomal enzymes cytochrome P450 2C9 and 2C19.

(c) Cimetidine acts as a potent inhibitor of several cytochrome P450 isoenzymes, including the 2C9 and 2C19 subfamilies responsible for phenytoin clearance.

(d) Co-administration of cimetidine downregulates this enzymatic degradation, resulting in a rapid accumulation of phenytoin in the systemic circulation.

(e) This elevation manifests clinically as phenytoin toxicity, causing classic cerebellar symptoms such as horizontal nystagmus, ataxia, diplopia, and slurred speech.

(f) Conversely, agents like rifampin, carbamazepine, and phenobarbital function as classic enzyme inducers that would lower phenytoin levels rather than cause toxicity.

Final Answer: The co-administered drug that caused toxicity is Cimetidine.

Answer: (C)

Go Back to Question 12

Q13.

Solution

Concept:

Antimicrobial pharmacology categorizes antibacterial agents according to their specific structural targets within a bacterial cell. Understanding whether an antibiotic disrupts peptidoglycan assembly or binds to specific ribosomal subunits during translation is critical for predicting its spectrum of activity and clinical utility.

Solution:

(a) The structural schematic outlines two distinct molecular targets: Site 1, which represents cell wall synthesis inhibitors, and Site 2, which highlights the 30S ribosomal subunit.

(b) Ceftriaxone is a third-generation beta-lactam antibiotic that binds to penicillin-binding proteins, blocking the final transpeptidation step of peptidoglycan synthesis at Site 1.

(c) Gentamicin belongs to the aminoglycoside class, which selectively binds to the 16S rRNA of the 30S bacterial ribosomal subunit at Site 2, causing misreading of mRNA.

(d) Azithromycin and clindamycin bind to the 50S ribosomal subunit, whereas ciprofloxacin inhibits DNA gyrase, meaning they do not fit the 30S paradigm.

(e) Linezolid binds to the 50S subunit to prevent the formation of the 70S initiation complex, excluding it from being a Site 2 inhibitor.

(f) Therefore, the combination of ceftriaxone and gentamicin accurately aligns with the targets highlighted at Site 1 and Site 2 respectively.

Final Answer: The pair corresponding to Site 1 and Site 2 is Ceftriaxone; Gentamicin.

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept:

Bacterial cell wall biosynthesis is a multi-step pathway beginning in the cytoplasm before building blocks are transported across the inner membrane. Disrupting the earliest enzymatic steps of this sequence prevents the formation of key peptidoglycan precursors, leading to bacterial autolysis.

Solution:

(a) The early steps of bacterial cell wall assembly require the conversion of UDP-N-acetylglucosamine into UDP-N-acetylmuramic acid inside the bacterial cytoplasm.

(b) This critical commitment step is catalyzed by the specific enzyme UDP-N-acetylglucosamine enolpyruvyl transferase, frequently designated as MurA.

(c) Fosfomycin is a phosphoenolpyruvate analogue that covalently binds to a cysteine residue in the active site of the MurA enzyme, inactivating it completely.

(d) By blocking this initial cytoplasmic step, fosfomycin halts all subsequent peptidoglycan production, demonstrating broad-spectrum bactericidal activity against common urinary pathogens.

(e) This unique mechanism of action minimizes cross-resistance with other cell wall inhibitors like cephalexin, which act on late-stage transpeptidation outside the membrane.

(f) Nitrofurantoin damages bacterial DNA, while trimethoprim blocks dihydrofolate reductase, making them mechanistically distinct from cytoplasmic cell wall synthesis inhibition.

Final Answer: The antibiotic selected is Fosfomycin.

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept:

The management of Mycobacterium tuberculosis infections utilizes a combination of specialized medications that target different metabolic processes of the bacillus. The clinical utility of these agents is frequently governed by their distinct, class-specific adverse effect profiles.

Solution:

(a) Multi-drug regimens for tuberculosis incorporate several primary agents, including isoniazid, rifampin, ethambutol, and pyrazinamide, each possessing unique toxicity risks.

(b) Ethambutol functions by inhibiting the enzyme arabinosyl transferase, which prevents the polymerization of arabinogalactan in the mycobacterial cell wall.

(c) A well-characterized, dose-dependent adverse effect associated with ethambutol therapy is the development of retrobulbar optic neuritis.

(d) This neurological toxicity presents clinically as a progressive decrease in visual acuity along with an impairment of red-green color discrimination.

(e) Patients receiving long-term ethambutol require routine baseline and periodic ophthalmologic examinations to monitor visual function and detect early toxicity.

(f) Isoniazid is primarily linked to peripheral neuropathy via pyridoxine depletion, rifampin causes orange discoloration of secretions, and pyrazinamide is known for hyperuricemia and hepatotoxicity.

Final Answer: The anti-tubercular medication causing optic neuritis is Ethambutol.

Answer: (C)

Go Back to Question 15

Q16.

Solution

Concept:

Highly active antiretroviral therapy regimens exploit several unique stages of the Human Immunodeficiency Virus replication cycle. Suppressing the integration of viral genetic material into the host genome prevents the establishment of a permanent template for viral transcription.

Solution:

(a) Following reverse transcription of viral RNA into double-stranded DNA, the virus must insert its genetic code into the host cell chromosome.

(b) This critical process is mediated by the viral integrase enzyme, which catalyzes the strand transfer of viral DNA into host genomic sequences.

(c) Dolutegravir belongs to the class of medications known as Integrase Strand Transfer Inhibitors (INSTIs).

(d) It binds selectively to the integrase viral enzyme, blocking the coordination of essential divalent magnesium or manganese ions required for catalytic activity.

(e) By preventing this covalent integration, the drug stalls the viral replication life cycle and stops the production of new viral particles.

(f) Efavirenz acts as a non-nucleoside reverse transcriptase inhibitor, atazanavir blocks the viral protease enzyme, and maraviroc is a CCR5 coreceptor antagonist.

Final Answer: The drug representing this mechanism of action is Dolutegravir.

Answer: (B)

Go Back to Question 16

Q17.

Solution

Concept:

Bacterial resistance to beta-lactam antibiotics is frequently driven by the production of beta-lactamase enzymes that hydrolyze the cyclic amide ring. Combining sensitive antibiotics with beta-lactamase inhibitors restores antimicrobial efficacy by neutralizing these defensive bacterial proteins.

Solution:

(a) Pseudomonas aeruginosa strains often develop resistance by expressing extended-spectrum beta-lactamases that break down extended-spectrum penicillins like piperacillin.

(b) Tazobactam is a structural beta-lactam analogue that functions as a suicide inhibitor, binding irreversibly to the active site of classical beta-lactamases.

(c) This covalent binding shields piperacillin from enzymatic hydrolysis, preserving its ability to inhibit penicillin-binding proteins and destroy the bacterial cell.

(d) While avibactam is a highly effective non-beta-lactam inhibitor, the prompt specifies shielding from an extended-spectrum enzyme where tazobactam is classically paired with piperacillin.

(e) Cilastatin is not a beta-lactamase inhibitor; it inhibits renal dehydropeptidase-1 to prevent the breakdown of imipenem in the kidneys.

(f) Probenecid competes for renal tubular secretion pathways to raise the plasma concentration of penicillins but has no direct inhibitory effect on bacterial enzymes.

Final Answer: The agent combined with the antibiotic is Tazobactam.

Answer: (B)

Go Back to Question 17

Q18.

Solution

Concept:

Neurohormonal modulation in heart failure aims to counter maladaptive remodeling by enhancing beneficial endogenous vasoregulatory mechanisms. Inhibiting the metabolic breakdown of endogenous natriuretic peptides promotes diuresis, natriuresis, and systemic vasodilation.

Solution:

(a) Neprilysin is a zinc-dependent metalloendopeptidase responsible for the degradation of several vasoactive peptides, including atrial and brain natriuretic peptides.

(b) Sacubitril is a prodrug that is metabolized into an active neprilysin inhibitor, preventing the breakdown of these vital natriuretic compounds.

(c) Increased levels of natriuretic peptides stimulate membrane-bound guanylyl cyclase, elevating cyclic GMP concentrations in vascular smooth muscle and renal tubules.

(d) This neurohormonal profile promotes systemic vasodilation, reduces ventricular filling pressures, and enhances sodium and water excretion to lower cardiac preload.

(e) Because neprilysin also breaks down angiotensin II, sacubitril must be paired with an angiotensin receptor blocker like valsartan to prevent dangerous vasoconstriction.

(f) This pharmacological approach does not decrease aldosterone synthesis directly or alter hyperpolarization-activated cyclic nucleotide-gated channels, which are targeted by ivabradine.

Final Answer: The primary physiological benefit is decreasing the degradation of endogenous natriuretic peptides.

Answer: (A)

Go Back to Question 18

Q19.

Solution

Concept:

Renal handling of glucose involves filtering plasma through the glomerulus followed by near-complete reabsorption in the early segments of the nephron. Inhibiting these transport proteins drops the renal threshold for glucose, facilitating calorie clearance and lowering systemic blood pressure.

Solution:

(a) Under normal physiological conditions, more than ninety percent of filtered glucose is reabsorbed in the S1 segment of the proximal convoluted tubule.

(b) This high-capacity transport is driven by Sodium-Glucose Cotransporter 2 (SGLT2) proteins located on the apical membrane of tubular epithelial cells.

(c) SGLT2 inhibitors block this pathway, promoting controlled glucosuria and leading to reductions in hemoglobin A1c, body weight, and systolic blood pressure.

(d) Clinical trial data confirms that this class provides cardiorenal benefits, including a reduced risk of heart failure hospitalizations in patients with cardiovascular disease.

(e) GLP-1 receptor agonists and DPP-4 inhibitors target the incretin pathway to modulate insulin and glucagon secretion without causing direct glucosuria.

(f) Sulfonylureas act directly on pancreatic beta cells to stimulate insulin release by blocking ATP-sensitive potassium channels, which carries a high risk of hypoglycemia.

Final Answer: The drug belongs to the class of SGLT2 inhibitors.

Answer: (C)

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Q20.

Solution

Concept:

Antimetabolite chemotherapy often employs structural analogues that inhibit essential enzymes required for nucleotide synthesis. When high doses are used to clear aggressive malignancies, rescue strategies must be deployed to safeguard healthy host tissues from irreversible toxicity.

Solution:

(a) Methotrexate is an antimetabolite that binds with high affinity to the enzyme dihydrofolate reductase, blocking the conversion of dihydrofolate to active tetrahydrofolate.

(b) This enzymatic block depletes the intracellular pool of folate cofactors needed for purine and thymidylate synthesis, arresting DNA replication in rapidly dividing cells.

(c) Leucovorin, also known as folinic acid, is a fully reduced 5-formyl derivative of tetrahydrofolate that does not require dihydrofolate reductase for activation.

(d) Administered as a rescue agent twenty-four hours after methotrexate, leucovorin enters healthy cells and restores the necessary folate pool for normal nucleic acid synthesis.

(e) This targeted timing protects vulnerable host tissues like the bone marrow and gastrointestinal mucosa from lethal myelosuppression and mucosal ulceration.

(f) Filgrastim stimulates granulocyte production, mesna prevents cyclophosphamide-induced hemorrhagic cystitis, and dexrazoxane mitigates anthracycline-mediated cardiotoxicity.

Final Answer: The rescue agent is Leucovorin (Folinic acid).

Answer: (A)

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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	B
6	C	7	B	8	A	9	C	10	B
11	C	12	C	13	B	14	B	15	C
16	B	17	B	18	A	19	C	20	A