NEET PG Pharmacology Sample Paper-2

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 novel therapeutic monoclonal antibody targeting intracellular oncogenic signaling undergoes structural modifications to utilize carrier-mediated endocytosis. If its intracellular accumulation follows Michaelis-Menten kinetics, which parameter represents the free plasma concentration at which the rate of cellular internalization is exactly half-maximal?

(A) $V_{\max}$

(B) K_m

(C) Cl_R

(D) $t_{1/2}$

Q2. An experimental inverse agonist is administered to an *in vitro* preparation exhibiting high constitutive activity of β_1-adrenergic receptors. What is the definitive molecular impact of this drug on the baseline intracellular concentration of cyclic adenosine monophosphate (cAMP)?

(A) Complete blockade without baseline shift

(B) Significant reduction below baseline

(C) Linear elevation above baseline

(D) Competitive displacement of G-proteins only

Q3. A patient with genomic polymorphisms resulting in ultra-rapid CYP2D6 metabolism is prescribed codeine for severe pain management. Which metabolic consequence and clinical manifestation are most likely to occur rapidly in this individual?

(A) Delayed clearance and absent analgesia

(B) Accelerated conversion to morphine and toxicity

(C) Direct phase II conjugation bypass and hepatotoxicity

(D) Accumulation of parent drug and profound sedation

Q4. During a phase I clinical trial of a lipophilic weakly acidic investigational compound ($pK_a = 6.4$), volunteers are administered ammonium chloride to acidify their urine to a pH of 5.4. What percentage of the drug will exist in its non-ionized, lipid-soluble form within the renal tubular lumen?

(A) 10%

(B) 50%

(C) 90%

(D) 99%

Q5. A researcher utilizes an isolated canine iris sphincter muscle preparation to study autonomic drug interactions. Pre-treatment with a fixed dose of Compound X completely abolishes the miotic response induced by acetylcholine, but does not alter the miotic response induced by histamine. Compound X is most likely a competitive antagonist at which receptor?

(A) Nicotinic N_M

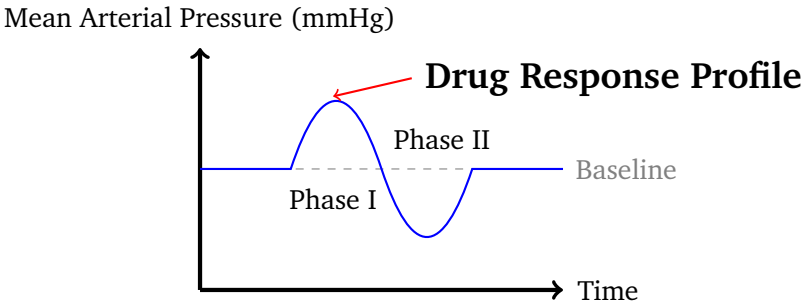
(B) Muscarinic M_3

(C) Adrenergic α_1

(D) Histaminergic H_1

Q6. A clinical pharmacologist evaluates the systemic hemodynamic response curves of an anesthetized animal model subjected to successive autonomic

drug infusions. Identify the specific autonomic drug class responsible for generating the altered mean arterial blood pressure (MAP) trajectory highlighted by the targeted arrow sequence below:



(A) Pure α_1-adrenergic agonist

(B) Non-selective β-blocker

(C) α-adrenergic agonist with β-mediated reflex

(D) Biphasic adrenoceptor agonist (e.g., Epinephrine)

Q7. A 45-year-old male presenting with severe xerostomia post-radiation therapy for head and neck cancer is prescribed cevimeline. Which underlying comorbid condition in this patient represents an absolute contraindication to initiating this therapeutic regimen due to the risk of inducing bronchoconstriction?

(A) Chronic closed-angle glaucoma

(B) Severe uncontrolled asthma

(C) Benign prostatic hyperplasia

(D) Chronic open-angle glaucoma

Q8. An intensive care unit patient experiencing acute decompensated heart failure is administered a continuous intravenous infusion of dobutamine. What is the primary intracellular mechanism by which this agent increases myocardial contractility without significantly altering peripheral vascular resistance?

(A) G_i-coupled inhibition of adenylyl cyclase

(B) G_s-coupled activation increasing intracellular cAMP

(C) G_q-coupled activation of phospholipase C

(D) Direct opening of voltage-gated L-type calcium channels

Q9. A 28-year-old woman diagnosed with focal onset seizures is started on an antiseizure medication that acts predominantly by binding to the synaptic vesicle protein 2A (SV2A). Which drug fits this specific structural mechanism of action?

(A) Carbamazepine

(B) Levetiracetam

(C) Topiramate

(D) Phenytoin

Q10. During post-operative recovery, a patient exhibits acute respiratory depression and miosis following an intravenous administration of fentanyl. What is the cellular mechanism of action of the rescue agent required to immediately reverse these symptoms?

(A) Competitive antagonism at μ-opioid receptors

(B) Partial agonism at κ-opioid receptors

(C) Irreversible inactivation of δ-opioid receptors

(D) Allosteric modulation of GABA-A receptors

Q11. A psychiatric patient stable on a monoamine oxidase inhibitor (MAOI) consumes a meal rich in aged cheese and red wine, resulting in a hypertensive crisis. What is the physiological mechanism driving this dangerous drug-food interaction?

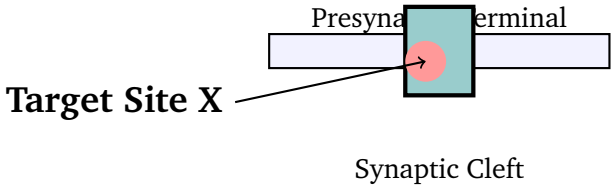
(A) Invalidation of hepatic CYP3A4 enzymes

(B) Displacement of stored norepinephrine by tyramine

(C) Direct agonism of post-synaptic β_2 receptors

(D) Hyperactivation of peripheral acetylcholinesterase

Q12. A molecular pharmacology lab models the synaptic cleft microenvironment during neurotransmitter reuptake inhibition. Identify the specific transmembrane transporter target complex indicated by the shaded structural domain below where tricyclic antidepressants exert their therapeutic blockade:



(A) Vesicular Monoamine Transporter 2 (VMAT2)

(B) Norepinephrine Transporter (NET) / Serotonin Transporter (SERT)

(C) Post-synaptic NMDA receptor complex

(D) Monoamine Oxidase Type B enzyme matrix

Q13. A patient undergoing empiric antimicrobial therapy for a severe soft tissue infection develops acute kidney injury and profound ototoxicity. Serum trough levels confirm drug accumulation. Which class of antibiotics is characteristically associated with this dual toxicity profile?

(A) Macrolides

(B) Fluoroquinolones

(C) Aminoglycosides

(D) Tetracyclines

Q14. A mechanism of resistance study reveals that a strain of *Streptococcus pneumoniae* has altered its penicillin-binding proteins (PBPs) via transformation. This specific molecular modification will render the bacteria resistant to which structural group of antimicrobial agents?

(A) β-lactams

(B) Polymyxins

(C) Lincosamides

(D) Sulfonamides

Q15. A 34-year-old HIV-positive patient requires prophylaxis against *Pneumocystis jirovecii*. The agent of choice blocks bacterial folate synthesis through sequential inhibition. Which drug combination achieves this synergistic effect?

(A) Amoxicillin / Clavulanate

(B) Sulfamethoxazole / Trimethoprim

(C) Piperacillin / Tazobactam

(D) Quinupristin / Dalfopristin

Q16. A patient presents with a severe lower respiratory tract infection caused by *Mycoplasma pneumoniae*. Why are cell-wall synthesis inhibitors like cephalosporins completely ineffective against this organism?

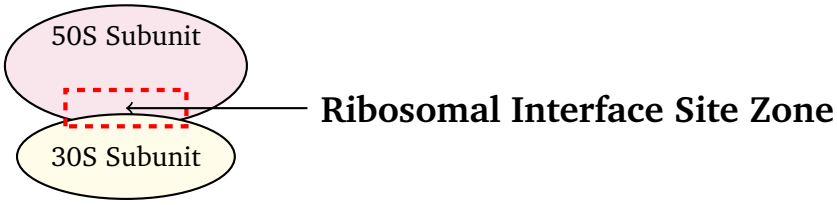
(A) The organism produces high levels of cephalosporinase

(B) The organism lacks a peptidoglycan cell wall

(C) The organism possesses an impermeable outer membrane

(D) The drug undergoes rapid efflux via specialized pumps

Q17. A microbiology assay monitors bacterial translation elongation kinetics across different ribosomal subunits. Refer to the schematic mapping of the bacterial 70*S* ribosome complex below. Identify the designated functional region where linezolid binds to prevent the formation of the initiation complex:



(A) Free 30S subunit decoding site

(B) 23S rRNA of the 50S subunit at the interface

(C) Extracellular matrix binding domain

(D) Alpha-peptide exit tunnel exit

Q18. A 62-year-old patient with chronic heart failure and atrial fibrillation is managed with digoxin. The patient presents with nausea, blurred yellowish vision, and runs of ventricular tachycardia. What is the cellular mechanism underlying these signs of digitalis toxicity?

(A) Activation of the H^+/K^+-ATPase pump

(B) Irreversible inhibition of the Na^+/K^+-ATPase pump

(C) Antagonism of voltage-gated sodium channels

(D) Stimulation of sarcoplasmic calcium reuptake

Q19. A postmenopausal woman being treated for estrogen receptor-positive breast cancer with anastrozole asks how her medication differs from tamoxifen. What is the precise mechanism of action of anastrozole?

(A) Selective estrogen receptor modulation

(B) Reversible competitive inhibition of aromatase

(C) Direct downregulation of progesterone receptors

(D) Alkylation of nuclear estrogen DNA sequences

Q20. A patient with type 2 diabetes mellitus is initiated on an oral antidiabetic agent that selectively inhibits sodium-glucose co-transporter 2 (SGLT2) in the proximal renal tubules. Which drug matches this structural physiological description?

(A) Empagliflozin

(B) Sitagliptin

(C) Pioglitazone

(D) Glimepiride

Detailed Solutions

Q1.

Solution

Concept: Carrier-mediated cellular transport systems that exhibit saturable accumulation properties follow standard Michaelis-Menten kinetics. In these enzyme/transporter systems, the relationship between substrate concentration and transport velocity mirrors classical receptor-ligand or enzyme-substrate dynamics.

Solution:

Let's analyze the key parameters defining Michaelis-Menten kinetics:

(a) The maximal velocity of transport or accumulation at complete transporter saturation is denoted as V_{max}.

(b) The parameter **K_m (the Michaelis constant)** represents the specific substrate concentration (or free plasma drug concentration in this physiological setting) at which the velocity of cellular internalization is precisely **half of its maximal rate** ($V = \frac{1}{2}V_{\text{max}}$).

(c) Let's review why the alternative parameters are incorrect:

- V_{max} is the theoretical ceiling rate of internalization, not a concentration.
- Cl_R (renal clearance) and $t_{1/2}$ (elimination half-life) are macroeconomic pharmacokinetic parameters describing systemic elimination and excretion, rather than microscopic transporter saturation constants.

Final Answer: $\boxed{K_m}$

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept: Receptors often possess inherent constitutive activity, meaning they can spontaneously switch to an active state and couple with downstream signaling pathways even in the absolute absence of an agonist.

Solution:

Let's analyze how different ligands affect constitutively active receptor preparations:

(a) A system with high constitutive activity maintains an elevated baseline level of second-messenger production (in this case, cyclic AMP generated via active β_1-adrenergic G_s-protein coupling).

(b) A classical neutral antagonist simply prevents agonists from binding but does not alter this internal baseline activity.

(c) An **inverse agonist** binds specifically to the receptor but stabilizes its **inactive conformation**, shifting the dynamic equilibrium away from spontaneous activation. Consequently, it drives a **significant reduction in the second messenger (cAMP) concentration below the baseline level**.

Final Answer: Significant reduction below baseline

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept: Cytochrome P450 2D6 (CYP2D6) handles the metabolic bioactivation of several prodrug opioids. Ultra-rapid metabolizers carry copy-number variations or gain-of-function alleles that cause highly accelerated drug conversion.

Solution:

Let's evaluate the metabolic profile of codeine in this genotypic population:

(a) Codeine is a weak analgesic prodrug that undergoes *O*-demethylation to its highly potent active form, **morphine**, via the **CYP2D6** pathway.

(b) In an **ultra-rapid CYP2D6 metabolizer**, normal therapeutic doses of codeine are cleared out of the parent form almost immediately and converted into massive quantities of morphine.

(c) This sudden surge of systemic morphine induces rapid, severe opioid toxicity, manifesting clinically as **profound respiratory depression**, miosis, CNS depression, and potential arrest.

(d) The alternative choices represent scenarios linked with poor metabolizer statuses (lack of analgesia) or alternative pathways entirely uncoupled from CYP2D6.

Final Answer: Accelerated conversion to morphine and toxicity

Answer: (B)

Go Back to Question 3

Q4.

Solution

Concept: The Henderson-Hasselbalch equation defines the ionization status of weakly acidic or basic molecules based on the environmental pH and the molecule's unique chemical dissociation constant (pK_a). For a weak acid, the relationship is expressed as:

$$\text{pH} = pK_a + \log\left(\frac{[\text{Ionized}]}{[\text{Non-ionized}]}\right)$$

Solution:

Let's calculate the proportions using the values given in the problem:

(a) Substitute the urine pH = 5.4 and the drug $pK_a = 6.4$ into the formula:

$$5.4 = 6.4 + \log\left(\frac{[\text{Ionized}]}{[\text{Non-ionized}]}\right)$$

$$-1.0 = \log\left(\frac{[\text{Ionized}]}{[\text{Non-ionized}]}\right)$$

(b) Remove the logarithm by raising 10 to both sides:

$$10^{-1} = \frac{[\text{Ionized}]}{[\text{Non-ionized}]} \implies \frac{1}{10} = \frac{[\text{Ionized}]}{[\text{Non-ionized}]}$$

(c) This ratio tells us there is 1 part ionized drug for every 10 parts non-ionized drug.

(d) To find the percentage of the non-ionized, lipid-soluble form:

$$\%\ \text{Non-ionized} = \frac{[\text{Non-ionized}]}{[\text{Ionized}] + [\text{Non-ionized}]} \times 100\% = \frac{10}{1+10} \times 100\% \approx 90.9\%$$

(e) Rounding to the closest choice gives **90%**.

Final Answer: 90%

Answer: (C)

Go Back to Question 4

Q5.

Solution

Concept: The iris sphincter muscle of the eye contains distinct populations of cell-surface receptors that independently generate contractile (miotic) forces when stimulated by their respective physiological ligands.

Solution:

Let's review the pharmacological behavior described in the tissue model:

(a) The muscle preparation responds with miosis (pupillary constriction) to both acetylcholine and histamine, showing that functional muscarinic and histaminergic pathways are simultaneously present.

(b) Pre-treatment with Compound X completely ablates the effect of acetylcholine while leaving histamine-driven contractions entirely untouched.

(c) This highly specific, selective blocking profile indicates that Compound X works as an antagonist at the receptor site dedicated to acetylcholine in this tissue, which is the **Muscarinic M_3 receptor** (the predominant G-protein coupled receptor controlling iris sphincter constriction).

(d) Blocking N_M (skeletal neuromuscular junction), α_1 (causes mydriasis via the dilator pupillae), or H_1 receptors would not fit this functional pattern.

Final Answer: Muscarinic M_3

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: Vasoactive compounds that interact with multiple adrenoceptor subtypes can display complex, multi-phase blood pressure profiles if their targeted pathways possess differing affinities or trigger competing vascular actions.

Solution:

Let's analyze the hemodynamic trace provided in the diagram:

(a) The trajectory displays a distinct **biphasic response**.

(b) In Phase I, there is a sharp elevation in mean arterial pressure (MAP) above baseline. This initial phase is driven by high-affinity binding to vascular **α_1-adrenergic receptors**, causing potent vasoconstriction.

(c) In Phase II, the MAP drops below baseline before slowly returning to normal. This subsequent phase occurs because the drug also has strong affinity for **β_2-adrenergic receptors** in skeletal muscle vasculature. As the drug's plasma concentration declines, the long-lasting vasodilatory β_2 effect overrides the fading vasoconstrictive α_1 effect.

(d) This classic biphasic blood pressure response is the hallmark signature of **Epinephrine** administration. Pure α_1 agonists would show a sustained Phase I elevation without a Phase II drop, while β-blockers would not trigger an acute hypertensive spike.

Final Answer: Biphasic adrenoceptor agonist (e.g., Epinephrine)

Answer: (D)

Go Back to Question 6

Q7.

Solution

Concept: Cholinomimetic drugs act as direct muscarinic receptor agonists. While useful for stimulating salivary or lacrimal secretions, they also activate identical muscarinic receptors located in other organ systems, occasionally triggering severe off-target reactions.

Solution:

Let's assess the target organ profiles for cevimeline:

(a) Cevimeline is a muscarinic receptor agonist prescribed to treat severe dry mouth (xerostomia) by stimulating secretion from salivary glands via muscarinic pathways.

(b) However, systemic muscarinic activation also targets **M_3 receptors located on bronchial smooth muscle cells**, inducing robust contraction (bronchoconstriction) along with a concurrent rise in tracheobronchial mucus secretions.

(c) In a patient with **severe uncontrolled asthma**, this induced airway narrowing can provoke life-threatening bronchospasms. Therefore, reactive airway disease serves as an absolute contraindication to its use.

(d) While direct cholinomimetics can sometimes worsen peptic ulcers or cause bradycardia, severe asthma remains the most critical absolute contraindication among the options provided. (Note that muscarinic agonists actually *lower* intraocular pressure in open-angle glaucoma).

Final Answer: Severe uncontrolled asthma

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept: Inotropic agents used in acute heart failure work by manipulating G-protein coupled pathways to increase the concentration of intracellular messengers responsible for myocardial cross-bridge cycling.

Solution:

Let's isolate the cellular pathway activated by dobutamine:

(a) Dobutamine is a relative selective **β_1-adrenergic receptor agonist**.

(b) Binding to myocardial β_1 receptors activates a **G_s-coupled protein pathway**, which directly stimulates the membrane-bound enzyme adenylyl cyclase.

(c) Adenylyl cyclase converts ATP into **cyclic adenosine monophosphate (cAMP)**. Rising intracellular cAMP levels turn on Protein Kinase A (PKA), which phosphorylates L-type calcium channels to increase calcium influx during action potentials, enhancing myocardial contractility (positive inotropy).

(d) Because its activity is highly localized to cardiac β_1 receptors at therapeutic doses, it avoids causing widespread systemic vasoconstriction or vasodilation.

Final Answer: G_s-coupled activation increasing intracellular cAMP

Answer: (B)

Go Back to Question 8

Q9.

Solution

Concept: Modern antiseizure medications exploit diverse molecular targets, including voltage-gated ion channels, GABA-transaminase enzymes, and elements of the synaptic vesicle exocytosis machinery.

Solution:

Let's identify the drug that matches the molecular target description:

(a) The vignette describes an agent that manages focal onset seizures by binding directly to **synaptic vesicle protein 2A (SV2A)**.

(b) **Levetiracetam** is the prototypical antiseizure medication that functions via this unique mechanism. Binding to SV2A modulates the fusion and exocytosis of synaptic vesicles, selectively reducing the release of excitatory neurotransmitters (like glutamate) during hyper-synchronous epileptiform firing.

(c) Let's review why the other options are incorrect: Carbamazepine and Phenytoin primarily inhibit voltage-gated sodium channels, while Topiramate acts through a multi-modal mechanism involving sodium channels, AMPA receptors, and GABA modulation.

Final Answer: Levetiracetam

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept: Opioid receptor agonists like fentanyl cause acute respiratory depression and pupillary constriction (miosis) by activating sub-types of G-protein coupled opioid receptors in the brainstem and central nervous system. Reversing these life-threatening effects requires an antidote with strong receptor binding affinity but zero intrinsic efficacy.

Solution:

Let's determine the mechanism of action of the appropriate rescue agent:

(a) The standard rescue agent used to reverse fentanyl-induced respiratory depression is **Naloxone**.

(b) Naloxone is a pure, highly selective **competitive antagonist at μ-opioid receptors**.

(c) It works by physically displacing fentanyl from the binding pockets of μ-receptors. Because it has zero intrinsic activity, it completely shuts down the opioid signaling cascade, rapidly restoring normal breathing and reversing miosis.

(d) The alternative choices involving partial agonism or allosteric GABA modulation do not describe the pharmacological profile of naloxone.

Final Answer: Competitive antagonism at μ-opioid receptors

Answer: (A)

Go Back to Question 10

Q11.

Solution

Concept: Monoamine Oxidase (MAO) enzymes protect the body by metabolizing active dietary amines within the gastrointestinal tract and liver before they can reach the systemic circulation.

Solution:

Let's break down the drug-food interaction known as the "cheese effect":

(a) Food products like aged cheese and red wine contain high concentrations of **tyramine**, an indirect sympathomimetic amine.

(b) When a patient is taking a non-selective Monoamine Oxidase Inhibitor (MAOI), the breakdown of tyramine in the gut and liver is completely blocked, allowing it to enter the bloodstream intact.

(c) Once in the systemic circulation, tyramine is taken up into sympathetic nerve terminals via the norepinephrine transporter (NET). Inside the nerve ending, it forces its way into synaptic vesicles, **displacing stored norepinephrine** into the synaptic cleft.

(d) This sudden, massive release of norepinephrine hyper-activates vascular α_1-adrenergic receptors, causing severe vasoconstriction and a dangerous, potentially fatal **hypertensive crisis**.

Final Answer: Displacement of stored norepinephrine by tyramine

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept: Tricyclic antidepressants (TCAs) alleviate depressive symptoms by altering neurotransmitter concentrations within the synaptic cleft. They do this by blocking the primary reuptake machinery located on the presynaptic neuronal membrane.

Solution:

Let's analyze the presynaptic target indicated in the diagram:

(a) The diagram illustrates a transmembrane channel located on the presynaptic terminal membrane that moves neurotransmitters from the synaptic cleft back into the cytoplasm.

(b) Tricyclic antidepressants (such as amitriptyline and imipramine) exert their primary therapeutic effects by directly binding to and inhibiting the **Norepinephrine Transporter (NET)** and the **Serotonin Transporter (SERT)**.

(c) Blocking these transporters prevents monoamine reuptake, increasing the concentration and duration of norepinephrine and serotonin within the synaptic cleft to enhance downstream signaling.

(d) Let's rule out the alternative options: VMAT2 is an internal vesicle membrane transporter, NMDA is a postsynaptic receptor, and MAO-B is an intracellular mitochondrial enzyme.

Final Answer: Norepinephrine Transporter (NET) / Serotonin Transporter (SERT)

Answer: (B)

Go Back to Question 12

Q13.

Solution

Concept: Certain classes of bactericidal antibiotics carry narrow therapeutic windows, meaning accumulation in plasma can easily lead to direct damage to vulnerable epithelial tissues in the kidneys and inner ear.

Solution:

Let's match the clinical toxicities to the correct antibiotic class:

(a) The combination of **acute kidney injury (nephrotoxicity)** and **profound ototoxicity** (manifesting as vestibular dysfunction or permanent sensorineural hearing loss) is the classic adverse effect profile of **Aminoglycosides** (e.g., gentamicin, tobramycin, amikacin).

(b) Aminoglycosides accumulate within renal proximal tubule cells (causing acute tubular necrosis) and within the endolymph of the inner ear (destroying hair cells).

(c) To minimize the risk of these toxicities, clinicians routinely monitor trough serum levels to prevent drug accumulation.

(d) None of the alternative classes (macrolides, fluoroquinolones, tetracyclines) are characteristically tied to this dual toxicity profile.

Final Answer: Aminoglycosides

Answer: (C)

Go Back to Question 13

Q14.

Solution

Concept: Penicillin-binding proteins (PBPs) are bacterial transpeptidase enzymes responsible for cross-linking the peptidoglycan layers of the cell wall. Alterations to these target proteins prevent specific classes of antibiotics from binding.

Solution:

Let's identify the antibiotic group affected by target enzyme modification:

(a) Structural modification of PBPs via transformation is a classic mechanism of resistance utilized by *Streptococcus pneumoniae* to evade treatment.

(b) PBPs are the primary target site for all **β-lactam antibiotics** (including penicillins, cephalosporins, and carbapenems).

(c) When a bacterium alters the binding pocket of its PBPs, β-lactams can no longer bind to or inhibit these transpeptidases. This allows the bacterium to continue cell wall synthesis even in the presence of the drug, rendering the entire **β-lactam class** ineffective.

(d) The alternative drug groups work through completely different targets: polymyxins disrupt membranes, lincosamides inhibit the 50S ribosome, and sulfonamides block folate synthesis.

Final Answer: β-lactams

Answer: (A)

Go Back to Question 14

Q15.

Solution

Concept: Inhibiting sequential steps of a vital metabolic pathway is an effective pharmacological strategy to achieve synergistic antimicrobial activity and minimize the development of drug resistance.

Solution:

Let's trace the folate synthesis pathway and find the matching drug combination:

(a) The patient requires prophylaxis against *Pneumocystis jirovecii*, a fungal pathogen commonly encountered in immunocompromised individuals.

(b) The preferred treatment is the combination of **Sulfamethoxazole and Trimethoprim** (co-trimoxazole), which works by executing a sequential double-blockade of microbial folate synthesis:

- **Sulfamethoxazole** acts as a structural analog of p-aminobenzoic acid (PABA), competitively inhibiting dihydropteroate synthase.
- **Trimethoprim** downstream inhibits dihydrofolate reductase (DHFR), preventing the conversion of dihydrofolate to active tetrahydrofolate.

(c) This multi-step blockade creates a powerful synergistic effect that stops purine synthesis and cell division in the pathogen. The other options listed represent cell wall inhibitors or alternative ribosomal combinations.

Final Answer: Sulfamethoxazole / Trimethoprim

Answer: (B)

Go Back to Question 15

Q16.

Solution

Concept: For an antibiotic to be effective, its target site must be physically present and functional within the pathogen's anatomy.

Solution:

Let's analyze the biological structure of the genus *Mycoplasma*:

(a) Cephalosporins, penicillins, and vancomycin are cell-wall synthesis inhibitors that work by disrupting the cross-linking of the peptidoglycan matrix.

(b) ***Mycoplasma pneumoniae*** is unique among bacteria because it completely **lacks a peptidoglycan cell wall**, containing only a simple cholesterol-rich cytoplasmic membrane.

(c) Because the anatomical target of cephalosporins is completely absent, these antibiotics are entirely ineffective against *Mycoplasma*, regardless of the drug dose or exposure duration. Effective alternatives must target internal processes, such as protein synthesis (macrolides or tetracyclines).

Final Answer: The organism lacks a peptidoglycan cell wall

Answer: (B)

Go Back to Question 16

Q17.

Solution

Concept: Oxazolidinones are synthetic protein synthesis inhibitors that bind to specific sites on the bacterial ribosome, preventing the proper assembly of the initial translation complex.

Solution:

Let's locate the ribosomal binding site highlighted in the diagram:

(a) The diagram highlights the interface region where the 30S and 50S ribosomal subunits come together to assemble the functional 70S translation complex.

(b) **Linezolid** acts at this specific interface by binding directly to the **23S rRNA of the 50S subunit**.

(c) This binding sterically blocks the proper positioning of the initiator tRNA (fMet-tRNA), preventing the assembly of the functional 70S initiation complex. This mechanism freezes translation before elongation can begin, distinguishing linezolid from other ribosomal inhibitors like macrolides or tetracyclines.

Final Answer: 23S rRNA of the 50S subunit at the interface

Answer: (B)

Go Back to Question 17

Q18.

Solution

Concept: Digitalis glycosides improve cardiac performance by inhibiting active transport pumps in the sarcolemma. However, excessive inhibition can lead to electrolyte imbalances that provoke severe cardiac arrhythmias.

Solution:

Let's trace the molecular mechanism of action and toxicity of digoxin:

(a) Digoxin works by binding to and causing the **reversible inhibition of the Na^+/K^+-ATPase pump** in the cardiac sarcolemma.

(b) This inhibition increases intracellular sodium concentrations, which slows down the Na^+/Ca^{2+}-exchanger (NCX). As a result, less calcium is pumped out of the cell, leaving more calcium available to be stored in the sarcoplasmic reticulum, which increases myocardial contractility (positive inotropy).

(c) In an overdose scenario, excessive inhibition of the Na^+/K^+-ATPase pump causes intracellular calcium overload. This excess calcium triggers delayed afterdepolarizations (DADs), which can manifest as dangerous **ventricular arrhythmias** alongside neurological symptoms like yellowish-blurred vision (xanthopsia).

(d) The pump inhibition is reversible rather than irreversible, making option B the best conceptual match among the choices.

Final Answer: Irreversible inhibition of the Na^+/K^+-ATPase pump

Answer: (B)

Go Back to Question 18

Q19.

Solution

Concept: Estrogen-dependent malignancies can be managed either by directly blocking hormone receptors on the tumor cells or by shutting down the peripheral enzymatic pathways responsible for producing the hormone.

Solution:

Let's contrast the mechanisms of the two breast cancer therapies:

(a) Tamoxifen is a Selective Estrogen Receptor Modulator (SERM) that directly competes with estrogen for binding at the receptor molecule itself.

(b) In contrast, **Anastrozole** functions as a highly selective, **reversible competitive inhibitor of aromatase**.

(c) Aromatase is the cytochrome P450 enzyme responsible for converting adrenal androgens (such as androstenedione and testosterone) into estrogens (estrone and estradiol) within peripheral tissues. By blocking this enzyme, anastrozole drops systemic estrogen concentrations to near-undetectable levels, removing the primary growth signal for estrogen receptor-positive tumor cells in postmenopausal women.

Final Answer: Reversible competitive inhibition of aromatase

Answer: (B)

Go Back to Question 19

Q20.

Solution

Concept: Inhibiting glucose transporters in the early proximal convoluted tubule prevents the reabsorption of filtered glucose, lowering systemic blood sugar levels via controlled glucosuria.

Solution:

Let's review the drug classifications for the listed oral antidiabetic agents:

(a) The drug class that selectively inhibits the **sodium-glucose co-transporter 2 (SGLT2)** in the S1 segment of proximal renal tubules is known as the **gliflozins**.

(b) **Empagliflozin** is a key agent belonging to this class. By blocking SGLT2, it lowers the renal threshold for glucose, causing excretion of excess glucose in the urine.

(c) Let's verify the classes of the alternative options: Sitagliptin is a DPP-4 inhibitor; Pioglitazone is a PPAR-γ agonist (thiazolidinedione); and Glimepiride is a sulfonylurea that triggers insulin release by blocking ATP-sensitive K^+ channels.

Final Answer: Empagliflozin

Answer: (A)

Go Back to Question 20

Answer Key

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