USMLE Step 3 Drug MOA Practice Test (Sample)

Study Guide

BY EXAMZIFY

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Questions

1. **Which ribosomal subunit does clindamycin act on and how does it affect protein synthesis?**

   A. 50S ribosome, inhibits translocation

   B. 30S ribosome, blocks tRNA-aa from binding

   C. 50S ribosome, inhibits peptidyl transferase

   D. 30S ribosome, induces premature termination

2. **What is the primary effect of statins on cholesterol levels?**

   A. Increases hepatic cholesterol synthesis

   B. Decreases hepatic cholesterol synthesis

   C. Inhibits penicillin-binding proteins

   D. Activates autolytic enzymes

3. **How is acyclovir activated in the body?**

   A. By liver enzymes for metabolism

   B. By viral thymidine kinase

   C. By human cellular kinases

   D. By direct absorption in the gastrointestinal tract

4. **Infliximab and adalimumab inhibit which cytokine to treat autoimmune conditions?**

   A. IL-6

   B. TNF-α

   C. IL-1

   D. IL-10

5. **How do antimalarial drugs like chloroquine function against malaria parasites?**

   A. They inhibit folate synthesis in the parasites

   B. They inhibit heme polymerization, causing cell death

   C. They enhance the immune response to infection

   D. They block protein synthesis in parasites

6. Which drug class decreases the synthesis of thymidylate and purine nucleotides?

A. Antifolates like trimethoprim

B. Beta-lactam antibiotics

C. HMG-CoA reductase inhibitors

D. Non-selective beta antagonists

7. What is the primary action of warfarin in blood coagulation?

A. Increases intracellular calcium levels

B. Inhibits ADP receptors

C. Blocks vitamin K dependent carboxylation of clotting factors

D. Activates guanylate cyclase

8. How does fluorouracil work as a chemotherapy agent?

A. It induces apoptosis in tumor cells

B. It inhibits thymidylate synthase, blocking DNA synthesis

C. It enhances tumor immunogenicity

D. It increases metabolic activity in cancer cells

9. What is the main therapeutic action of phenylephrine?

A. β2 agonist for bronchodilation

B. α1 and α2 agonist for vasoconstriction

C. Inhibition of Na/K ATPase

D. Calcium channel blockade

10. How does the mechanism of action of benzodiazepines contribute to their anxiolytic effects?

A. It selectively blocks dopamine receptors

B. It enhances GABA’s inhibitory neurotransmission

C. It inhibits norepinephrine's action

D. It activates serotonin receptors

Answers

1. **C**
2. **B**
3. **B**
4. **B**
5. **B**
6. **A**
7. **C**
8. **B**
9. **B**
10. **B**

Explanations

1. Which ribosomal subunit does clindamycin act on and how does it affect protein synthesis?

A. 50S ribosome, inhibits translocation

B. 30S ribosome, blocks tRNA-aa from binding

C. 50S ribosome, inhibits peptidyl transferase

D. 30S ribosome, induces premature termination

Clindamycin acts on the 50S ribosomal subunit and inhibits peptidyl transferase activity. By binding to this subunit, clindamycin disrupts the normal function of the ribosome during the protein synthesis process. Specifically, it prevents the formation of peptide bonds between amino acids, which is a crucial step in building polypeptide chains. This inhibition effectively stops the elongation of the nascent protein, leading to a reduction in protein synthesis. The choice indicating that clindamycin inhibits translocation is close but not entirely accurate since clindamycin's primary action is on the peptidyl transferase aspect of protein synthesis. Understanding the specific mechanisms involved is important in recognizing how antibiotics target bacterial ribosomes, as well as how they differ in their actions from other classes of antibiotics that may affect other subunits or processes in the protein synthesis pathway.

2. What is the primary effect of statins on cholesterol levels?

A. Increases hepatic cholesterol synthesis

B. Decreases hepatic cholesterol synthesis

C. Inhibits penicillin-binding proteins

D. Activates autolytic enzymes

Statins primarily function by decreasing hepatic cholesterol synthesis. They achieve this by inhibiting the enzyme HMG-CoA reductase, which is a crucial component in the cholesterol biosynthesis pathway. When HMG-CoA reductase is inhibited, the liver reduces its production of cholesterol. This, in turn, lowers the levels of low-density lipoprotein (LDL) cholesterol in the bloodstream, as the liver responds to the decreased intracellular cholesterol by upregulating LDL receptors to clear more cholesterol from the blood. The decrease in hepatic cholesterol synthesis not only contributes to lower LDL levels but also has several beneficial effects on cardiovascular health, including the stabilization of atherosclerotic plaques and a reduction in inflammation. Thus, statins are widely utilized in the management of hyperlipidemia and prevention of cardiovascular diseases.

3. How is acyclovir activated in the body?

- **A. By liver enzymes for metabolism**
- **B. By viral thymidine kinase**
- **C. By human cellular kinases**
- **D. By direct absorption in the gastrointestinal tract**

Acyclovir is a nucleoside analog that exhibits its antiviral properties primarily through its conversion to its active form by viral thymidine kinase. This enzyme is produced by certain herpes viruses, including HSV (Herpes Simplex Virus) and VZV (Varicella-Zoster Virus). When acyclovir enters infected cells, it is phosphorylated by viral thymidine kinase to form acyclovir monophosphate. Further phosphorylation occurs with the help of human cellular kinases, resulting in the formation of acyclovir triphosphate, which is the active metabolite. This active form inhibits viral DNA polymerase, thereby terminating viral DNA synthesis and replication, which leads to the antiviral effects observed in the treatment of infections caused by these viruses. The activation of acyclovir is significant because it selectively affects virus-infected cells due to the dependency on the viral thymidine kinase for its initial phosphorylation step, resulting in a more targeted action compared to other antiviral medications. This selective activation contributes to the low toxicity of acyclovir to uninfected cells.

4. Infliximab and adalimumab inhibit which cytokine to treat autoimmune conditions?

- **A. IL-6**
- **B. TNF-α**
- **C. IL-1**
- **D. IL-10**

Infliximab and adalimumab are monoclonal antibodies that target and inhibit tumor necrosis factor-alpha (TNF-α), a pro-inflammatory cytokine involved in systemic inflammation. By neutralizing TNF-α, these medications help reduce inflammation and have proven effective in treating various autoimmune conditions, such as rheumatoid arthritis, Crohn's disease, and psoriasis. This inhibition leads to decreased inflammatory responses that can contribute to the symptoms and progression of these diseases, making TNF-α a crucial target for therapeutic intervention in autoimmune disorders. Other cytokines listed, such as IL-6, IL-1, and IL-10, are involved in different aspects of immune function and inflammation but are not the primary targets of infliximab and adalimumab.

5. How do antimalarial drugs like chloroquine function against malaria parasites?

A. They inhibit folate synthesis in the parasites

B. They inhibit heme polymerization, causing cell death

C. They enhance the immune response to infection

D. They block protein synthesis in parasites

Antimalarial drugs like chloroquine function primarily by inhibiting heme polymerization in malaria parasites. Chloroquine acts on the food vacuole of the Plasmodium species, the causal agent of malaria. During their life cycle, malaria parasites digest hemoglobin from host red blood cells to obtain amino acids for their growth. This digestion releases heme, which is toxic to the parasites. To counteract this toxicity, the parasites polymerize free heme into non-toxic hemozoin. Chloroquine interferes with this polymerization process, leading to the accumulation of toxic free heme within the parasite. This accumulation ultimately results in cell death, thereby effectively combating the malaria infection. The other options describe mechanisms that are not applicable to chloroquine's action in treating malaria. For example, inhibiting folate synthesis pertains to drugs like sulfonamides and pyrimethamine, while enhancing the immune response relates to immunomodulatory therapies that are not specific to chloroquine. Blocking protein synthesis is typically the mechanism of action for antibiotics like tetracyclines, which target bacterial infections rather than malaria.

6. Which drug class decreases the synthesis of thymidylate and purine nucleotides?

A. Antifolates like trimethoprim

B. Beta-lactam antibiotics

C. HMG-CoA reductase inhibitors

D. Non-selective beta antagonists

Antifolates, such as trimethoprim, act by inhibiting enzymes involved in the folate metabolism pathway, which is essential for nucleotide synthesis. Specifically, trimethoprim inhibits dihydrofolate reductase, an enzyme that catalyzes the reduction of dihydrofolate to tetrahydrofolate. Tetrahydrofolate is a critical cofactor required for the synthesis of thymidylate (a pyrimidine nucleotide) and purines. Therefore, by impeding the production of tetrahydrofolate, antifolates effectively decrease the synthesis of these nucleotides, which are vital for DNA replication and cell division. Other drug classes listed do not exert this effect. Beta-lactam antibiotics primarily act by inhibiting bacterial cell wall synthesis. HMG-CoA reductase inhibitors, commonly known as statins, reduce cholesterol synthesis and do not influence nucleotide synthesis. Non-selective beta antagonists are primarily used to manage cardiovascular conditions and do not have a mechanism related to nucleic acid metabolism. Thus, antifolates like trimethoprim are the correct choice for decreasing the synthesis of thymidylate and purine nucleotides.

7. What is the primary action of warfarin in blood coagulation?

A. Increases intracellular calcium levels

B. Inhibits ADP receptors

C. Blocks vitamin K dependent carboxylation of clotting factors

D. Activates guanylate cyclase

Warfarin's primary action in blood coagulation is to block the vitamin K-dependent carboxylation of clotting factors. This mechanism is fundamental to its anticoagulant effect. Warfarin inhibits the enzyme vitamin K epoxide reductase, which is necessary for the regeneration of reduced vitamin K. Reduced vitamin K is essential for the carboxylation (and subsequent activation) of specific clotting factors—namely factors II (prothrombin), VII, IX, and X—as well as proteins C and S. When these factors are not carboxylated, they cannot bind calcium ions, which is crucial for their proper function and interaction within the coagulation cascade. Consequently, warfarin leads to a decrease in the synthesis of functional coagulation factors, effectively reducing blood clotting and helping to prevent thromboembolic events. This understanding is key in clinical settings where the management of coagulation is necessary, such as in patients with a history of deep vein thrombosis or atrial fibrillation.

8. How does fluorouracil work as a chemotherapy agent?

A. It induces apoptosis in tumor cells

B. It inhibits thymidylate synthase, blocking DNA synthesis

C. It enhances tumor immunogenicity

D. It increases metabolic activity in cancer cells

Fluorouracil is primarily recognized for its mechanism of action as an antineoplastic agent, specifically through the inhibition of thymidylate synthase. Thymidylate synthase is an enzyme crucial for the synthesis of thymidine monophosphate (dTMP), which is a necessary precursor for DNA synthesis. By inhibiting this enzyme, fluorouracil effectively reduces the availability of dTMP, leading to a disruption in DNA synthesis and ultimately impairing the ability of rapidly dividing tumor cells to proliferate. This mechanism of action is particularly significant because it specifically targets tumor cells that are highly reliant on rapid DNA synthesis for growth and division. Fluorouracil's effectiveness in blocking DNA synthesis is one of the reasons it is utilized in the treatment of various cancers, including colorectal and breast cancer. In the context of the other options, apoptosis induction is a separate mechanism that can occur through various agents, but it is not the primary action of fluorouracil. Enhancing tumor immunogenicity refers to assuring that the immune system can better recognize and attack the tumor, which is not directly relevant to fluorouracil's mechanism. Additionally, increasing metabolic activity in cancer cells would typically suggest promoting growth rather than inhibiting

9. What is the main therapeutic action of phenylephrine?

A. β2 agonist for bronchodilation

B. α1 and α2 agonist for vasoconstriction

C. Inhibition of Na/K ATPase

D. Calcium channel blockade

Phenylephrine primarily acts as an alpha-1 adrenergic agonist, leading to vasoconstriction of blood vessels. This mechanism occurs through the stimulation of alpha-1 receptors located on vascular smooth muscle. When phenylephrine binds to these receptors, it causes an increase in intracellular calcium concentration, which results in smooth muscle contraction and subsequent vasoconstriction. Phenylephrine is commonly used as a decongestant to alleviate nasal congestion and as a treatment for hypotension during surgical procedures due to its ability to raise blood pressure by increasing systemic vascular resistance. While it does have some weak activity on alpha-2 receptors, its predominant action is through alpha-1 stimulation, making it effective in conditions where increased vascular tone is desired. The other choices pertain to mechanisms not associated with phenylephrine; for instance, bronchodilation is related to beta-2 adrenergic agonists, and inhibition of Na/K ATPase is characteristic of cardiac glycosides like digoxin, while calcium channel blockade relates to medications such as calcium channel blockers that lower blood pressure through different actions.

10. How does the mechanism of action of benzodiazepines contribute to their anxiolytic effects?

A. It selectively blocks dopamine receptors

B. It enhances GABA’s inhibitory neurotransmission

C. It inhibits norepinephrine's action

D. It activates serotonin receptors

The anxiolytic effects of benzodiazepines are primarily due to their mechanism of action of enhancing GABA (gamma-aminobutyric acid) neurotransmission. Benzodiazepines bind to a specific site on the GABA-A receptor, a type of ligand-gated ion channel that mediates fast inhibitory synaptic transmission in the central nervous system. When benzodiazepines bind to their site on this receptor, they increase the frequency of chloride ion channel opening in the presence of GABA. This leads to an influx of chloride ions into the neuron, making it more hyperpolarized and less likely to fire. By enhancing GABA's inhibitory effects, benzodiazepines produce a calming effect on the brain, reducing anxiety, promoting sedation, and facilitating muscle relaxation. This mechanism is central to their effectiveness in treating anxiety disorders, insomnia, and certain seizure disorders, differentiating them from other drug classes that might target different pathways, such as dopamine or serotonin systems, without enhancing GABAergic tone directly.