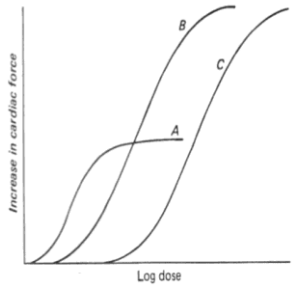
اسئلة الوزاري

1. **The process by which the effect of a ligand is reversed and the activity of receptors is decreased below baseline is characteristic of:**
   a) Full agonists
   b) Competitive antagonists
   c) Allosteric modulators
   d) Partial agonist
   e) Inverse agonist
2. **Which describes the response when a ligand binds to a Gi-coupled GPCR receptor?**
   a) Increased intracellular Ca^{2+}
   b) A channel opens and positive ions flow into the cell
   c) Increased intracellular cAMP
   d) Decreased intracellular cAMP
   e) Translocation of the receptor into the nucleus
3. **A drug with high intrinsic activity means:**
   a) It has high selectivity for its target receptor
   b) It has high efficacy
   c) It has high potency
   d) It has a high therapeutic index
   e) It has high bioavailability
4. **In the context of a quantal dose-response relationship, which of the following best describes the ED50?**
   a) The dose at which 50% of individuals exhibit the maximum response to the drug
   b) The dose at which 50% of the population shows the therapeutic effect of a drug
   c) The dose that causes side effects in 50% of the population
   d) The dose required to cause death in 50% of the population
   e) The dose at which 50% of the drug is metabolized by the liver
5. **A drug with a high therapeutic index is considered:**
   a) Less safe because it has a narrow margin of safety
   b) More effective regardless of its safety
   c) Highly toxic due to high potency
   d) Safer because it has a wide margin between therapeutic and toxic doses
   e) Fast-acting with a short half-life

6. **In the context of adrenergic agonists, what does the term (tachyphylaxis) refer to?**
   a) The rapid development of tolerance to a drug's effects
   b) An unexpectedly rapid onset of drug action
   c) The development of withdrawal symptoms upon cessation
   d) An increase in drug effectiveness with repeated dosing
   e) None of the above

7. **Prior to clinical trials in patients with heart failure, an animal study was carried out to compare two new positive inotropic drugs (A and B) to a current standard agent (C). The results of cardiac output measurements are shown in the graph below. Which of the following statements is correct?**
   a) Drug A is most effective
   b) Drug B is least potent
   c) Drug C is most potent
   d) Drug B is more potent than drug C and more effective than drug A
   e) Drug A is more potent than drug B and more effective than drug C



34. Which of the following cytochrome P450 isoenzymes is involved in the metabolism of the largest number of drugs in human beings and has been implicated in some dangerous drug interactions?
a) CYP 3A4
b) CYP 206
c) CYP 201
d) CYP 1A2
e) CYP 2C19

39. Regarding secondary messengers, the increase of second messengers (cAMP, cGMP, Ca+2, etc.) concentration leads to:
a) Inhibition of intracellular protein kinases and protein phosphorylation
b) Inhibition of protein phosphorylation only
c) Protein kinases activation and protein phosphorylation
d) Blocking of interaction between a receptor and an effector
e) Antagonism with endogenous ligands

40. Regarding the DOSE-RESPONSE RELATIONSHIPS, full Agonists have:
a) Affinity to receptors only
b) Affinity and maximal intrinsic activity
c) Affinity but no intrinsic activity
d) Affinity and submaximal intrinsic activity
e) Affinity without potency

46. Therapeutic index, all true except:
a) Larger value indicates the drug is toxic.
b) Therapeutic index = TD50/ED50
c) As the toxic dose of a drug is large, the drug has a large therapeutic index
d) Penicillin is an example of a drug with a large therapeutic index.
e) Warfarin is an example of a drug with a low therapeutic index.

55. Cells have many different types of receptors. All the following agents act by intracellular receptors EXCEPT:
a) Thyroid hormone
b) Vitamin D
c) Insulin
d) Cortisol
e) Steroids

56. Graded dose-response relationship is used to determine the following:
a) Potency only
b) Efficacy only
c) Safety only
d) Potency and efficacy
e) Potency, efficacy, safety

63. Regarding allosteric antagonists, choose the most appropriate answer:
a) Reduce agonist potency
b) Reduce agonist efficacy
c) Causes downward shift of the EC50 with no change in the Emax value of an agonist
d) Reduce agonist efficacy
e) Decrease EC50

اسئلة تجميع للجابتر الثاني من مختلف الجامعات (امتحان المد)

- **Which type of drugs interact with receptors to exert their effects?**

A) Antacids
B) Antibiotics
C) Antihistamines
D) Analgesics
E) Agonists

- **Receptors exist in which of the following states?**

A) Inactive (R)
B) Active (R*)
C) Both A and B
D) Neither A nor B
E) None of the above

- **Which of the following drugs stabilizes the fraction of inactive receptor states?**

A) Agonists
B) Antagonists
C) Partial agonists
D) Second messengers
E) Ligands

- **Which receptor mediates sodium influx and potassium outflux upon stimulation by acetylcholine?**

A) Nicotinic receptor
B) Muscarinic receptor
C) GABA receptor
D) Dopamine receptor
E) Serotonin receptor

- **G protein-coupled receptors interact with which intracellular components upon activation?**

A) DNA
B) RNA
C) Protein kinases
D) Structural proteins
E) None of the above

- **What is a common effector activated by Gs and inhibited by Gi?**

A) cAMP
B) IP3
C) DAG

D) GTP
E) ATP

- **Which receptors possess tyrosine kinase activity?**

A) Ligand-gated ion channels
B) G protein-coupled receptors
C) Enzyme-linked receptors
D) Intracellular receptors
E) None of the above

- **The time course of activation and response for intracellular receptors is typically:**

A) Milliseconds
B) Seconds to minutes
C) Minutes to hours
D) Hours to days
E) None of the above

- **What is a characteristic of signal transduction?**

A) Inability to amplify small signals
B) Mechanisms to stimulate excessive stimulation
C) Ability to amplify small signals
D) Short duration of G protein activation
E) None of the above

- **What is responsible for further actions within the cell upon dissociation from the α-GTP complex?**

A) G protein
B) βγ complex
C) Adenylyl cyclase
D) GTP
E) Effector molecule

- **Which receptors are responsible for generating two second messengers: IP3 and DAG?**

A) Ligand-gated ion channels
B) G protein-coupled receptors
C) Enzyme-linked receptors
D) Intracellular receptors
E) None of the above

- **What is the primary target of intracellular receptors?**

A) Enzymes
B) DNA
C) Structural proteins
D) Transcription factors
E) RNA

- **Which receptors undergo conformational changes upon activation?**

A) Ligand-gated ion channels
B) G protein-coupled receptors
C) Enzyme-linked receptors
D) Intracellular receptors
E) All of the above

- **Which of the following is NOT a second messenger?**

A) cAMP
B) IP3
C) DAG
D) GTP
E) None of the above

- **What is responsible for the phosphorylation of tyrosine residues upon activation?**

A) G protein
B) Ligand
C) Adenylyl cyclase
D) Tyrosine kinase
E) None of the above

- **Which of the following receptors is entirely intracellular?**

A) Ligand-gated ion channels
B) G protein-coupled receptors
C) Enzyme-linked receptors
D) Intracellular receptors
E) None of the above

- **What protects the cell from excessive stimulation during signal transduction?**

A) Signal amplification
B) Inactivation of G proteins
C) Mechanisms to downregulate receptors
D) Short duration of signal cascades
E) None of the above

- **What describes the actions of a drug on the body and the influence of drug concentrations on the magnitude of the response?**

A) Pharmacokinetics
B) Pharmacodynamics
C) Pharmacogenomics
D) Pharmacoeconomics
E) Pharmacognosy

- **What is NOT a major receptor family?**

A) Ligand-gated ion channels
B) G protein-coupled receptors

C) Enzyme-inhibited receptors
D) Intracellular receptors
E) None of the above

- **Ligand-gated ion channels regulate the flow of ions across cell membranes by:**

A) Keeping the channel open indefinitely
B) Closing the channel in the presence of agonists
C) Opening the channel briefly upon agonist activation
D) Preventing ions from crossing the membrane
E) None of the above

- The action of the drug on the body is known as ……

  - a) Pharmacokinetic
  - b) Pharmacodynamic
  - c) Pharmacology
  - d) Pharmacotherapeutics

- Which of the following statement about competitive antagonism is true?

  - a) Can cause a parallel shift to the right in a drug-response curve for agonist
  - b) Can be reversed by increasing dose of agonist
  - c) Appear to decrease the potency of the agonist
  - d) All of the above
  - e) None of the above

- Antagonists have:

  - a) Affinity to receptors.
  - b) Affinity and maximal intrinsic activity.
  - c) Affinity but no intrinsic activity.
  - d) Affinity and submaximal intrinsic activity.
  - e) Inverse the intrinsic activity.

- The following log dose-response curve show that:

  - a) Drug A and B have equal potency
  - b) Drug B and C have equal efficacy
  - c) Drug A and B have equal efficacy
  - d) Drug A and C have the same affinity and efficacy
  - e) Drug C more potent than drug B

- A drug that binds to a receptor and produces a biological response that mimics the response to the endogenous ligand is known as:

  - a) Agonist

- b) Antagonist
- c) Functional antagonist
- d) Partial agonist
- e) Partial antagonist

- When EMax of drug A is less than EMax of drug B, this means: drug B:

  - a) More potent.
  - b) Less potent.
  - c) Equal potency.
  - d) Less efficacy.
  - e) More efficacy.

- All the following are cell membrane receptors except:

  - a) Ligand-gated ion channels
  - b) G protein-coupled receptors.
  - c) Enzyme-linked receptors.
  - d) Steroid hormone receptors.
  - e) (GABA) receptors.

- When EC50 of drug A is less than EC50 of drug B, this means: drug B:

  - a) More potent.
  - b) Less potent.
  - c) Equal potency.
  - d) Less efficacy.
  - e) More efficacy.

- Antagonism by Atropine to acetylcholine is called:

  - a) Competitive antagonism.
  - b) Noncompetitive antagonism.
  - c) Allosteric antagonism.
  - d) Functional antagonism.
  - e) Reversible antagonism.

- **Which of the following up-regulates postsynaptic α1-adrenergic receptors?**

A. Daily use of amphetamine that causes release of norepinephrine
B. A disease that causes an increase in the activity of norepinephrine neurons
C. Daily use of phenylephrine, an α1 receptor agonist
D. Daily use of prazosin, an α1 receptor antagonist
E. Number of receptors cannot be changed in any circumstances.

- **A drug may act by all the following mechanisms EXCEPT:**

A. Interaction with cell membrane phospholipids

B. Interaction with protein macromolecules embedded in the cell membranes
C. Interaction with cell membrane ion channels
D. Interaction with intracellular enzymes
E. Interaction with gene functions

- **Ion-channel-linked receptors (direct ligand-gated ion channels) are characterized by:**

A. They are the type of receptors principally present in autonomic ganglia and skeletal muscles' motor end plate
B. They are the type of receptors principally present in vascular endothelium
C. They are rosette-shaped structures consisting of 7 membrane subunits
D. Their response is slower than other receptors
E. Activation of these receptors leads to activation of a second messenger

- **The following statements are true for the quantal dose-response relationship EXCEPT:**

A. It is the response to anticonvulsant and antiarrhythmic drugs
B. The response to the drug is not directly proportional to drug concentration (all-or-none)
C. It could be tested in one animal
D. It helps in the calculation of the ED50 and LD50 of drugs
E. It helps in estimation of the degree of drug safety

- **The following is true for competitive antagonism:**

A. It never occurs with enzymes
B. Is the same as physiological antagonism
C. The agonist can never abolish the effect of the antagonist
D. Is best exemplified by the use of neostigmine to treat curare toxicity
E. Best described as a non-surmountable process

- **Which of the following best describes what the term “tachyphylaxis” means?**

A. An increase in the rate of the response, for example, an increase in the rate of muscle contraction
B. Immediate hypersensitivity reactions (i.e., anaphylaxis)
C. Prompt conformational changes of the receptor such that agonists, but not antagonists, are able to bind and cause a response
D. Quick and progressive rises in the intensity of the drug response, with repeated administration, even when the doses are unchanged
E. Rapid development of tolerance to the drug’s effects

- **Drugs X and Y have the same mechanism of diuretic action. Drug X in a dose of 5 mg produces the same magnitude of diuresis as 500 mg of drug Y. This suggests that:**

A. Drug Y is less efficacious than drug X
B. Drug X is more potent than drug Y
C. Toxicity of drug X is less than that of drug Y
D. Drug X is a safer drug than drug Y
E. Drug X will have a shorter duration of action than drug Y because less of drug X is present for a given effect

- **Which of the following can produce a therapeutic response? A drug that is:**

A. Bound to plasma albumin
B. Unbound to plasma proteins
C. Concentrated in the bile
D. Concentrated in the urine
E. Not absorbed from the GI tract

- **For intravenous (IV) dosages, what is the bioavailability assumed to be?**

A. 0%
B. 25%
C. 50%
D. 75%
E. 100%

- **A pro-drug is:**

A. The prototype member of a class of drugs
B. The oldest member of a class of drugs
C. An inactive drug that is transformed in the body to an active metabolite
D. A drug that is stored in the body tissues and is then gradually released in the circulation
E. An ionized drug trapped in breast milk

- **Which is correct concerning the safety/therapeutic index of using warfarin versus penicillin?**

A. Warfarin is a safer drug because it has a low therapeutic index.
B. Warfarin treatment has a high chance of resulting in dangerous adverse effects if bioavailability is altered.
C. The high therapeutic index makes penicillin a safe drug for all patients.
D. Penicillin treatment has a high chance of causing dangerous adverse effects if bioavailability is altered.
E. Both drugs are considered dangerous drugs if bioavailability is altered.

- **In relation to clearance, one is false:**

  - a) The clearance is increased in patients with heart failure.
  - b) Clearance is used to determine the maintenance dose.
  - c) The clearance is increased in patients with hypoalbuminemia.
  - d) Children have high clearance.

- **In relation to half-life, one is false:**

  - a) It is important in determining the dose interval.
  - b) It is correlated with clearance.
  - c) Drugs with high volume of distribution (Vd) have a long half-life.
  - d) None of the above.

- **In relation to the Michaelis-Menten equation, one is false:**
    - a) It is used for drugs that follow zero-order kinetics.
    - b) The rate of metabolism is not affected by drug concentration.
    - c) Because of saturable mechanism, the rate of metabolism is constant.
    - d) None of the above.

Study Questions

Choose the ONE best answer.

2.1 Which of the following best describes how a drug that acts as an agonist at the A subtype of GABA receptors affects signal transduction in a neuron?

A. Activation of this receptor subtype alters transcription of DNA in the nucleus of the neuron.
B. Activation of this receptor subtype opens ion channels that allow sodium to enter cells and increases the chance of generating an action potential.
C. Activation of this receptor subtype opens ion channels that allow chloride to enter cells and decreases the chance of generating an action potential.
D. Activation of this receptor subtype results in G protein activation and increased intracellular second messenger levels.

Correct answer = C. The GABA-A receptor is a ligand-gated ion channel selective for chloride. Agonists for the GABA-A receptor increase opening of channels, resulting in chloride entry into the neuron, hyperpolarization, and decreased action potential events.

2.2 If 1 mg of lorazepam produces the same anxiolytic response as 10 mg of diazepam, which is correct?

A. Lorazepam is more potent than is diazepam.
B. Lorazepam is more efficacious than is diazepam.
C. Lorazepam is a full agonist, and diazepam is a partial agonist.
D. Lorazepam is a better drug to take for anxiety than is diazepam.

Correct answer = A. A drug that causes the same effect at a lower dose is more potent. B and C are incorrect because without information about the maximal effect of these drugs, no conclusions can be made about efficacy or intrinsic activity. D is incorrect because the maximal response obtained is often more important than the amount of drug needed to achieve it.

2.3 If 10 mg of oxycodone produces a greater analgesic response than does aspirin at any dose, which is correct?

A. Oxycodone is more efficacious than is aspirin.
B. Oxycodone is less potent than is aspirin.
C. Aspirin is a full agonist, and oxycodone is a partial agonist.
D. Oxycodone and aspirin act on the same drug target.

Correct answer = A. Drugs with greater response at maximally effective concentrations are more efficacious than drugs with a lower maximal response. Choice B is incorrect since no information is given about the half maximal concentrations of either drug. Choices C and D are incorrect since it is not known if both drugs bind to the same receptor population.

2.4 In the presence of propranolol, a higher concentration of epinephrine is required to elicit full antiasthmatic activity. Propranolol has no effect on asthma symptoms. Which is correct regarding these medications?

A. Epinephrine is less efficacious than is propranolol.
B. Epinephrine is a full agonist, and propranolol is a partial agonist.
C. Epinephrine is an agonist, and propranolol is a competitive antagonist.
D. Epinephrine is an agonist, and propranolol is a noncompetitive antagonist.

Correct answer = C. Since propranolol decreases the effect of epinephrine but the inhibition can be overcome by giving a higher dose of epinephrine, propranolol must be a competitive antagonist. If D were correct, even very high concentrations of epinephrine would not be able to elicit a maximal effect in the presence of propranolol. Since propranolol has no effect by itself, A and B are incorrect.

2.5 In the presence of picrotoxin, diazepam is less efficacious at causing sedation, regardless of the dose. Picrotoxin has no sedative effect, even at the highest dose. Which of the following is correct regarding these agents?

A. Picrotoxin is a competitive antagonist.
B. Picrotoxin is a noncompetitive antagonist.
C. Diazepam is less efficacious than is picrotoxin.
D. Diazepam is less potent than is picrotoxin.

Correct answer = B. Since picrotoxin decreases the maximal effect of diazepam regardless of the diazepam dose, it is a noncompetitive antagonist. Picrotoxin has no efficacy alone, so C is incorrect. No information is provided about potency of either drug.

2.6 Haloperidol, chlorpromazine, and clozapine are antipsychotic medications that bind to the D2 subtype of dopamine receptors, with a binding affinity of haloperidol > chlorpromazine > clozapine. Which statement would have to be correct to conclude that the mechanism of antipsychotic effects for these drugs is via binding to D2 receptors?

A. Haloperidol should have the lowest potency of the three antipsychotic drugs.
B. D2 receptor binding should also be related to the potency of these drugs in causing Parkinson's-like adverse effects.
C. A positive correlation should exist between the affinity of these drugs to bind to D2 receptors and their potency for antipsychotic actions.
D. Clozapine would have to be more potent than chlorpromazine for decreasing psychosis.

Correct answer = C. To conclude that the mechanism of antipsychotic effect for these drugs is via binding to D2 receptors, there should be a positive correlation between the affinity of the drugs for D2 receptors and their potency for antipsychotic actions. Haloperidol should have the highest antipsychotic potency and clozapine the lowest. There is no guarantee the therapeutic effects and adverse effects are mediated by the same receptor population; therefore, a different correlation may exist for the adverse effects and D2 receptor affinity.

2.7 If there were spare β_1-adrenergic receptors on cardiac muscle cells, which statement would be correct?

A. The number of spare β_1-adrenergic receptors determines the size of the maximum effect of the agonist epinephrine.
B. Spare β_1 adrenergic receptors make the cardiac tissue less sensitive to epinephrine.
C. A maximal effect of epinephrine is seen when only a portion of β_1 adrenergic receptors are occupied.
D. Spare receptors are active even in the absence of epinephrine.

Correct answer = C. Only a fraction of the total receptors need to be bound to elicit a maximum cellular response when spare receptors are present. The other choices do not accurately describe the effects of having spare receptors.

2.8 Which of the following up-regulates postsynaptic α_1-adrenergic receptors?

A. Daily use of amphetamine that causes release of norepinephrine
B. A disease that causes an increase in the activity of norepinephrine neurons
C. Daily use of phenylephrine, an α_1 receptor agonist
D. Daily use of prazosin, an α_1 receptor antagonist

Correct answer = D. Up-regulation of receptors occurs when receptor activation is lower than normal, such as when the receptor is continuously exposed to an antagonist for that receptor. Down-regulation of receptors occurs when receptor activation is greater than normal because of continuous exposure to an agonist, as described in A, B, and C.

2.9 Methylphenidate helps patients with attention deficit hyperactivity disorder (ADHD) maintain attention and perform better at school or work, with an ED_{50} of 10 mg. However, methylphenidate can also cause significant nausea at higher doses (TD_{50} = 30 mg). Which is correct regarding methylphenidate?

A. The therapeutic index of methylphenidate is 3.
B. The therapeutic index of methylphenidate is 0.3.
C. Methylphenidate is more potent at causing nausea than treating ADHD.
D. Methylphenidate is more efficacious at causing nausea than treating ADHD.

Correct answer = A. Therapeutic index is calculated by dividing TD_{50} by ED_{50} (30/10), making B incorrect. C is incorrect because methylphenidate is more potent at treating ADHD (it takes a lower dose) than causing nausea. D. No information about efficacy is provided.

2.10 Which is correct concerning the safety of using warfarin (with a small therapeutic index) versus penicillin (with a large therapeutic index)?

A. Warfarin is a safer drug because it has a low therapeutic index.
B. Warfarin treatment has a high chance of resulting in dangerous adverse effects if bioavailability is altered.
C. The high therapeutic index makes penicillin a safe drug for all patients.
D. Penicillin treatment has a high chance of causing dangerous adverse effects if bioavailability is altered.

Correct answer = B. Agents with a low TI (that is, drugs for which dose is critically important) are those drugs for which bioavailability critically alters the therapeutic and adverse effects. A is incorrect, because a drug with a low TI is not generally considered to be safe. C is incorrect because a high TI does not ensure safety across the entire patient population. D is incorrect because the high TI makes it unlikely that bioavailability alters the incidence of therapeutic or adverse effects.

MCQ Pharmacology CHAPTER ٢ Drug-Receptor Interactions and Pharmacodynamics Dr. Omar 2025

1. which of the following best describes the role of an agonist in receptor signaling?
A) it binds to a receptor and inhibits its activity
B) it binds to a receptor and activates it, leading to a cellular response
C) it degrades the receptor to prevent further signaling
D it competes with second messengers to reduce signal transduction
E) it prevents ligand binding without affecting receptor function

2. what is the role of second messenger molecules in drug-receptor interactions?
A) they bind directly to the receptor to initiate a response
B) they are responsible for translating agonist binding into a cellular response
C) they prevent the drug from binding to the receptor
D) they act as antagonists to block receptor activation
E) they degrade the drug after receptor binding

3.Which of the following statements best describes the interaction between β-adrenergic and muscarinic receptors in cardiac cells?
A) They both bind the same agonist to produce identical effects.
B) They compete for the same binding site on the receptor.
C) They bind different agonists and regulate heart function dynamically.
D) They prevent the binding of neurotransmitters to cardiac receptors.
E) They are responsible for the degradation of neurotransmitters.

4. What determines the magnitude of a cellular response to an agonist?
A) The speed at which the agonist binds to the receptor
B) The number of drug-receptor complexes formed
C) The metabolism of the drug in the liver
D) The duration of drug-receptor interaction
E) The presence of antagonists in the system

5. How is drug-receptor binding similar to enzyme-substrate interactions?
A) Both involve the breakdown of the receptor or enzyme.
B) Both are non-specific and can bind to multiple targets.
C) Both involve specificity for a particular ligand or substrate.
D) Both result in the permanent activation of the receptor or enzyme.
E) Both require second messengers to function.

6. Which of the following is an example of a drug that does not act via receptor interaction?
A) Epinephrine
B) Acetylcholine
C) Propranolol
D) Antacids
E) Dopamine

7. Which of the following best describes the action of an agonist on receptor states?
A) It shifts the equilibrium from R* to R.
B) It stabilizes the receptor in the inactive state (R).
C) It shifts the equilibrium from R to R*, increasing the fraction of R*.
D) It prevents receptor activation by blocking ligand binding.
E) It has no effect on the receptor equilibrium.

8. What distinguishes a partial agonist from a full agonist?
A) A partial agonist stabilizes the receptor in the inactive state (R).
B) A partial agonist shifts R to R*, but to a lesser extent than a full agonist.
C) A partial agonist completely prevents receptor activation.
D) A partial agonist increases the fraction of R* more than a full agonist.
E) A partial agonist binds irreversibly to the receptor.

9. How does an antagonist affect receptor activation? A) It shifts the equilibrium toward R*, leading to activation.
B) It prevents agonist binding and stabilizes the receptor in the inactive state (R).
C) It enhances receptor activation by increasing the fraction of R*.
D) It increases the biological effect of the agonist.
E) It completely deactivates the receptor permanently.

10. Which of the following best describes the effect of an antagonist on receptor states?
A) It shifts the equilibrium toward the active state (R*).
B) It prevents the agonist from binding while increasing R*.
C) It stabilizes the receptor in the inactive state (R).
D) It converts all receptors to the active state (R*).
E) It increases the fraction of R* but less than a full agonist.

11. How does a partial agonist differ from a full agonist?
A) A partial agonist shifts R to R*, but to a lesser extent than a full agonist.
B) A partial agonist completely stabilizes the receptor in the inactive state (R).
C) A partial agonist irreversibly binds to the receptor, preventing activation.
D) A partial agonist increases R* to a greater extent than a full agonist.
E) A partial agonist functions identically to an antagonist.

12. Which of the following is the richest source of drug receptors?
A) Nucleic acids
B) Enzymes
C) Membrane-bound proteins
D) Structural proteins
E) Cytoplasmic organelles

13.Which type of receptor is most likely to be activated by a hydrophobic ligand?
A) Ligand-gated ion channels
B) G protein-coupled receptors
C) Enzyme-linked receptors

D) Intracellular receptors
E) Membrane-bound receptors

14. Which of the following receptor families is responsible for transducing extracellular signals into intracellular responses?
A) G protein-coupled receptors
B) Nucleic acids
C) Structural proteins
D) Phospholipids
E) Cytoplasmic enzymes

15. What is the primary function of the extracellular portion of ligand-gated ion channels?
A) Ion conduction across the membrane
B) ATP synthesis
C) Ligand binding and channel regulation
D) Protein phosphorylation
E) Endocytosis

16. Which of the following best describes the function of nicotinic acetylcholine receptors?
A) They increase chloride influx, leading to neuronal hyperpolarization
B) They allow sodium influx and potassium efflux, leading to action potential generation
C) They act as secondary messengers in G-protein coupled receptor pathways
D) They inhibit sodium influx, reducing neuronal excitability
E) They activate cyclic AMP (cAMP) pathways

17. How does activation of the GABA receptor influence neuronal activity?
A) It depolarizes the neuron, making it more excitable
B) It inhibits chloride ion flow, preventing hyperpolarization
C) It increases chloride influx, leading to hyperpolarization
D) It promotes calcium influx, causing neurotransmitter release
E) It activates sodium influx, leading to muscle contraction

18. Which statement is TRUE regarding voltage-gated ion channels?
A) They only open in response to neurotransmitter binding
B) They do not contain any ligand-binding sites
C) Local anesthetics block voltage-gated sodium channels, reducing neuronal conduction
D) They remain open continuously to allow ion movement
E) They are exclusively found in skeletal muscle

19. What is the primary function of ligand-gated ion channels in neurotransmission?
A) They directly produce action potentials in all neurons
B) They convert chemical signals into electrical signals by regulating ion flow
C) They function independently of agonist binding
D) They inhibit all excitatory signals in the nervous system
E) They primarily store neurotransmitters for release

20. What happens when an agonist binds to a G protein–coupled receptor (GPCR)?
A) The receptor directly phosphorylates intracellular proteins
B) The α subunit binds GTP and dissociates from the βγ complex
C) The receptor allows ions to pass through it
D) The βγ complex binds to GTP and activates adenylyl cyclase
E) The receptor undergoes endocytosis immediately

21. Which of the following G proteins activates phospholipase C?
A) Gs
B) Gi
C) Gq
D) Gt
E) G12

22. What is the primary second messenger produced by the activation of adenylyl cyclase?
A) Diacylglycerol (DAG)
B) Inositol 1,4,5-trisphosphate (IP3)
C) Guanosine triphosphate (GTP)
D) Cyclic adenosine monophosphate (cAMP)
E) Phosphatidylinositol 4,5-bisphosphate (PIP2)

23. Which of the following statements about G proteins is TRUE?
A) The βγ complex has no role after α subunit activation
B) The α subunit binds to GDP when the receptor is activated
C) Gi inhibits adenylyl cyclase, decreasing cAMP levels
D) Gq inhibits phospholipase C activity
E) G proteins function independently of membrane receptors

24. How does IP3 affect intracellular signaling? A) It decreases calcium levels inside the cell
B) It activates protein kinase A (PKA)
C) It activates protein kinase C (PKC) by binding directly to it
D) It increases intracellular calcium concentrations
E) It deactivates adenylyl cyclase

25. What is the primary function of enzyme-linked receptors?
A) To directly activate G proteins
B) To regulate ion channels by changing membrane potential
C) To undergo conformational changes and activate cytosolic enzymes
D) To transport molecules across the plasma membrane
E) To act as transcription factors

26. Which of the following enzyme activities is most commonly associated with enzyme-linked receptors?
A) Serine kinase activity
B) Tyrosine kinase activity
C) Phosphatase activity
D) Guanylyl cyclase activity
E) Lipase activity

27. How does insulin signaling occur through its receptor?
A) The receptor activates adenylyl cyclase, increasing cAMP
B) The receptor undergoes autophosphorylation, activating downstream proteins
C) The receptor acts as an ion channel, allowing glucose entry
D) The receptor directly binds to DNA and regulates gene expression
E) The receptor degrades glucose directly

28. What happens after phosphorylation of tyrosine residues on enzyme-linked receptors?
A) The receptor degrades itself and terminates signaling
B) The receptor internalizes and prevents further activation
C) The receptor recruits and activates specific signaling proteins
D) The receptor releases calcium ions into the cytoplasm
E) The receptor transports sodium ions across the membrane

29. Which of the following is NOT an example of an enzyme-linked receptor?
A) Epidermal growth factor receptor (EGFR)
B) Platelet-derived growth factor receptor (PDGFR)
C) Atrial natriuretic peptide receptor (ANPR)
D) Nicotinic acetylcholine receptor
E) Insulin receptor

30. How do intracellular receptors differ from other receptor families?
A) They are membrane-bound and activate second messengers
B) They require ligand binding to trigger ion channel opening
C) They are entirely located within the cell and require lipid-soluble ligands
D) They activate G proteins to initiate signaling cascades
E) They function through direct phosphorylation of proteins

31. Which characteristic must a ligand have to activate intracellular receptors?
A) High water solubility
B) High molecular weight
C) The ability to bind to cell surface receptors
D) Sufficient lipid solubility to cross the cell membrane
E) The ability to activate ion channels

32. What is the primary target of intracellular receptor-ligand complexes?
A) Ion channels
B) G proteins
C) Membrane phospholipids
D) Transcription factors in the nucleus
E) Extracellular matrix proteins

33. What typically happens to intracellular receptors upon ligand binding?
A) They open ion channels
B) They dissociate from binding proteins and translocate to the nucleus
C) They phosphorylate themselves and other proteins
D) They trigger immediate cellular responses within seconds
E) They activate membrane-bound enzymes

34. What is the usual time course for the effects of intracellular receptor activation?
A) Milliseconds
B) Seconds
C) Minutes
D) Hours to days
E) Immediate

35. Which of the following hormones acts via intracellular receptors?
A) Insulin
B) Epinephrine
C) Cortisol
D) Glucagon
E) Dopamine

36. Which of the following is NOT a target of intracellular ligands?
A) Tubulin
B) Dihydrofolate reductase
C) 50S ribosomal subunit
D) G protein-coupled receptors
E) Transcription factors

37. Which of the following drugs targets the enzyme dihydrofolate reductase?
A) Paclitaxel
B) Trimethoprim
C) Erythromycin
D) Ciprofloxacin
E) Aspirin

38. How do steroid hormones exert their effects on target cells?
A) By binding to cell surface receptors
B) By activating intracellular receptors that regulate gene expression
C) By directly increasing ATP production
D) By stimulating rapid neurotransmitter release
E) By blocking ion channels
39. What is the target of macrolide antibiotics like erythromycin? A) DNA polymerase
B) Peptidoglycan synthesis enzymes
C) The 50S ribosomal subunit of bacteria
D) RNA polymerase
E) The 30S ribosomal subunit of bacteria

40. What is a key feature of signal transduction?
A) It allows direct ligand entry into the nucleus
B) It amplifies small signals and prevents excessive stimulation
C) It only occurs in enzyme-linked receptors
D) It requires 100% receptor occupancy for activation
E) It blocks all downstream signaling pathways

41. How do G protein-linked and enzyme-linked receptors amplify signals?
A) By increasing ligand concentration at the receptor site
B) Through direct activation of gene transcription
C) By triggering a signal cascade that prolongs and enhances the response
D) By rapidly degrading second messengers
E) By immediately shutting down receptor activity after ligand binding

42. What is the concept of "spare receptors"?
A) Receptors that are permanently inactive
B) Receptors that exist in excess and are not necessary for a maximal response
C) Receptors that are only found in neurons
D) Receptors that require 100% occupancy for activation
E) Receptors that are stored in vesicles and never reach the membrane
43. What percentage of insulin receptors are considered "spare"?
A) 5%
B) 10%
C) 50%
D) 75%
E) 99%

44. Why does heart failure result in reduced functional reserve of B3-adrenoceptors?
A) Most receptors must be occupied to obtain maximum contractility
B) The receptors are permanently blocked by inhibitory proteins
C) There are no receptors present in the failing heart
D) The receptors are located inside the nucleus instead of the membrane
E) The receptors undergo permanent genetic mutations

45. What is tachyphylaxis in the context of receptor desensitization?
A) An increased response to an agonist after repeated exposure
B) A prolonged receptor activation without any response
C) A diminished response due to too much agonist stimulation
D) The process of receptor internalization within the cell
E) The development of receptor resistance to antagonists

46. What is one common mechanism that leads to receptor desensitization?
A) Phosphorylation that renders receptors unresponsive to the agonist
B) Activation of ion channels
C) Degradation of receptor mRNA
D) Increased production of second messengers
E) Enhanced receptor internalization without deactivation

47. What is down-regulation of receptors?
A) Receptors are continuously activated, leading to a permanent increase in sensitivity
B) Receptors are internalized and made unavailable for further agonist interaction
C) Receptors are phosphorylated to enhance agonist binding
D) Receptors are activated faster with repeated exposure to an agonist
E) Receptors are up-regulated in response to continuous stimulation

48. What happens to receptors during the "refractory" phase?
A) They become hyperactive and can be continuously activated
B) They require a recovery period before they can be activated again
C) They are down-regulated and removed from the cell membrane
D) They are phosphorylated to become more sensitive to stimuli
E) They increase their affinity for antagonists

49. What occurs as a result of repeated exposure to an antagonist?
A) Receptors undergo desensitization, reducing their response to further stimulation
B) Receptors are degraded in the cell and become nonfunctional
C) Receptor numbers decrease, leading to less sensitivity to the antagonist
D) Receptors are up-regulated, increasing sensitivity to agonists and/or resistance to antagonists
E) Receptors are blocked, preventing agonist binding

50. What determines the magnitude of the effect of an agonist drug?
A) Only the pharmacokinetic profile of the drug
B) The receptor's affinity for the drug and the drug concentration at the receptor site
C) The drug's metabolism rate
D) The duration of the drug’s effect
E) The route of administration of the drug

51. What is an example of an agonist drug mimicking the action of an endogenous ligand?
A) Isoproterenol mimicking norepinephrine on β1 receptors of the heart
B) Propranolol blocking norepinephrine on β1 receptors of the heart
C) Atropine inhibiting acetylcholine at muscarinic receptors
D) Captopril blocking the conversion of angiotensin I to angiotensin II
E) Naloxone blocking the effects of opioids at their receptors

52. What does a graded dose-response curve plot?
A) The drug's metabolism against its concentration
B) The drug's therapeutic effect against its side effects
C) The magnitude of response against increasing doses of a drug
D) The drug's absorption rate against its efficacy
E) The drug's duration of action against its concentration

53. What does potency refer to in pharmacology?
A) The maximum effect a drug can produce
B) The duration of action of a drug
C) The amount of drug necessary to produce an effect
D) The drug's ability to bind to its receptor
E) The drug's ability to be metabolized by the liver

54. How is potency typically determined?
A) By the maximal effect produced by the drug
B) By the time it takes for the drug to reach its peak effect
C) By the EC50 value, which is the concentration producing 50% of the maximum effect
D) By the dose required to produce a minimal effect
E) By the number of receptors the drug occupies

55. Which drug is more potent based on EC50 values?
A) Drug A with a higher EC50 than Drug B
B) Drug A with a lower EC50 than Drug B
C) Drug A and Drug B have the same potency
D) Potency cannot be determined by EC50
E) Potency depends on drug metabolism, not EC50

56. What is an example of a therapeutic preparation based on drug potency?
A) The dose of irbesartan compared to candesartan
B) The dose of aspirin compared to ibuprofen
C) The dose of penicillin compared to amoxicillin
D) The dose of acetamide compared to paracetamol
E) The dose of propranolol compared to metoprolol

57. What is a characteristic of drugs with higher efficacy?
A) They require higher doses to be effective
B) They produce a greater response even at lower concentrations
C) They have a higher EC50 value
D) They have a longer duration of action
E) They bind less efficiently to receptors

58. What does efficacy reflect in a drug?
A) The drug's ability to bind to a receptor
B) The number of receptors occupied by the drug
C) The maximal response the drug produces when it interacts with a receptor
D) The drug's side effects at high concentrations
E) The drug's interaction with other drugs

59. What is the maximal efficacy of a drug (Emax)?
A) The drug's ability to bind to its receptor
B) The concentration of the drug at the receptor site
C) The point at which all receptors are occupied, and no further increase in response occurs
D) The time it takes for the drug to reach maximum effect
E) The dose at which a drug is most potent

60. What happens when a drug reaches its maximal efficacy (Emax)?
A) The drug's effect increases with higher doses
B) The drug's effect plateaus, and no further increase in response occurs
C) The drug's potency increases
D) The drug’s receptors become desensitized
E) The drug is eliminated from the body

61. How does the efficacy of a full agonist differ from that of a partial agonist?
A) A full agonist produces a lower maximal response compared to a partial agonist
B) A full agonist and partial agonist have identical maximal responses
C) A full agonist has a higher maximal efficacy than a partial agonist
D) A partial agonist produces no response at all
E) A full agonist only activates a subset of receptors

62. What is a feature of antagonists regarding efficacy?
A) They produce a maximal response when bound to receptors
B) They do not produce a response even if they occupy all receptors
C) They increase the potency of other drugs
D) They enhance the intrinsic activity of the receptor
E) They have high efficacy in triggering cellular responses

63. What is the significance of a drug's potency in clinical practice?
A) Potency determines the drug's ability to cause side effects
B) Potency indicates the drug's speed of action
C) Potency helps in determining the correct dose needed for therapeutic effect
D) Potency reflects the drug's ability to bind to DNA
E) Potency is unrelated to the clinical effectiveness of a drug

64. What is the relationship between potency and EC50 value?
A) The higher the EC50 value, the more potent the drug
B) The lower the EC50 value, the more potent the drug
C) Potency is unrelated to EC50
D) EC50 values are only relevant for antagonists
E) EC50 values increase as potency decreases

65. What does a semilogarithmic plot of drug concentration versus response help determine?
A) The drug's side effects
B) The duration of action of a drug
C) The range of drug concentrations that cause a maximal response
D) The specific receptors involved in drug action
E) The number of receptors available for binding

66. Why is efficacy considered a more clinically useful characteristic than potency?
A) Efficacy determines how long a drug lasts in the body
B) Efficacy is more important for ensuring a therapeutic response
C) Efficacy reflects how easily the drug is metabolized
D) Efficacy determines the drug's cost-effectiveness
E) Efficacy ensures the drug has fewer side effects

67. What determines the ability of an agonist to fully or partially activate a receptor?
A) The drug's concentration alone
B) The drug's intrinsic activity
C) The receptor's affinity for the drug
D) The rate of metabolism of the drug
E) The drug's pharmacokinetic profile

68. How do drugs with different intrinsic activity levels affect Emax values?
A) Drugs with higher intrinsic activity produce lower Emax values
B) Drugs with lower intrinsic activity produce higher Emax values
C) Full agonists have a higher Emax value than partial agonists
D) Drugs with intrinsic activity do not influence Emax
E) Partial agonists always reach the same Emax as full agonists

69. What defines a full agonist in pharmacology?
A) It binds to a receptor but does not produce a biological response
B) It produces a partial response compared to the endogenous ligand
C) It binds to a receptor and produces a maximal biological response mimicking the endogenous ligand
D) It blocks the receptor, preventing any response
E) It binds to a receptor but stabilizes it in an inactive state

70. What is the intrinsic activity of a full agonist?
A) 0
B) 0.5
C) 1
D) 2
E) It varies depending on the drug

71. Which of the following is an example of a full agonist at α1-adrenoceptors?
A) Phenylephrine
B) Propranolol
C) Atropine
D) Naloxone
E) Fentanyl

72. What happens when phenylephrine binds to α1-adrenoceptors on vascular smooth muscle?
A) It decreases intracellular Ca2+ levels
B) It increases blood vessel diameter
C) It stabilizes the receptor in its active state, causing muscle contraction
D) It inhibits actin and myosin interaction
E) It causes relaxation of the smooth muscle

73. How does a full agonist produce the same maximal effect (Emax) as the endogenous ligand?
A) By occupying only a few receptors at high concentrations
B) By binding to the receptor and preventing any response
C) By stabilizing the receptor in its active state, leading to the maximal biological response
D) By inhibiting receptor activation completely
E) By reducing the number of receptors available for binding

74. What defines a partial agonist in pharmacology?
A) It has an intrinsic activity of one
B) It produces a maximal response even when only a few receptors are occupied
C) It has an intrinsic activity greater than zero but less than one
D) It prevents any response at the receptor site
E) It blocks the receptor and inhibits receptor activation

75. What happens when a partial agonist and a full agonist are present at the same receptor?

A) The full agonist always produces a greater response than the partial agonist
B) The partial agonist increases the Emax of the full agonist
C) The partial agonist may act as an antagonist, decreasing the Emax of the full agonist
D) The partial agonist has no effect on the full agonist
E) The partial agonist increases receptor sensitivity to the full agonist

76. In the presence of increasing concentrations of a partial agonist, what happens to the Emax of a receptor saturated with a full agonist?

A) It remains unchanged
B) It increases to the maximum response of the full agonist
C) It decreases until it reaches the Emax of the partial agonist
D) It causes an irreversible decrease in the receptor's ability to respond
E) It causes the full agonist to become ineffective

77. What is one therapeutic use of a partial agonist like aripiprazole?

A) It acts as a full agonist to fully activate dopamine receptors
B) It inhibits dopamine receptors completely
C) It stimulates underactive dopaminergic pathways and inhibits overactive pathways
D) It produces an intense dopaminergic response
E) It prevents dopamine receptor activation in all pathways

78. Why might aripiprazole cause a small risk of extrapyramidal adverse effects?

A) Because it is a full agonist at all dopamine receptors
B) Because it has a high affinity for dopamine receptors
C) Because it is a partial agonist that can both stimulate and inhibit dopaminergic pathways
D) Because it completely blocks dopamine receptors
E) Because it has no effect on dopaminergic pathways

79. What is the primary effect of an inverse agonist on a receptor?

A) It stabilizes the active form of the receptor
B) It increases the number of activated receptors above normal levels
C) It stabilizes the inactive form of the receptor, reducing the number of activated receptors
D) It has no effect on the receptor's activity
E) It prevents the receptor from binding to its endogenous ligand

80. How do inverse agonists differ from full agonists?

A) Inverse agonists stabilize the receptor in its active form, while full agonists stabilize the inactive form
B) Inverse agonists have intrinsic activity greater than one, while full agonists have intrinsic activity of zero
C) Full agonists increase receptor activity, while inverse agonists decrease receptor activity below the basal level
D) Full agonists prevent receptors from becoming activated, whereas inverse agonists promote receptor activation

E) Full agonists have no effect on receptor conformation, while inverse agonists change the receptor conformation to inactive

81. Inverse agonists exert the opposite pharmacological effect of which of the following?
A) Full agonists
B) Antagonists
C) Partial agonists
D) Allosteric modulators
E) Receptor blockers

82. What does the intrinsic activity of an inverse agonist typically indicate?
A) Intrinsic activity of zero
B) Intrinsic activity of one
C) Intrinsic activity greater than one
D) Intrinsic activity less than zero
E) No intrinsic activity

83. What characteristic defines a competitive antagonist?
A) It binds to the same site on the receptor as the agonist and has intrinsic activity
B) It binds to a different site on the receptor than the agonist and enhances the agonist's effect
C) It binds to the same site on the receptor as the agonist but does not activate the receptor
D) It binds to the receptor with low affinity but has a strong activating effect
E) It prevents the agonist from binding to its receptor by causing receptor activation

84. What happens to the dose-response curve in the presence of a competitive antagonist?
A) The Emax is decreased, and the EC50 remains unchanged
B) The EC50 decreases, and the Emax remains unchanged
C) The dose-response curve shifts to the right, and the Emax is unaffected
D) The dose-response curve shifts downward, and the EC50 increases
E) The Emax is decreased, and the curve shifts to the left

85. Which of the following is an example of a competitive antagonist?
A) Terazosin at α1-adrenoceptors
B) Phenylephrine at α1-adrenoceptors
C) Aripiprazole at dopamine receptors
D) Naloxone at opioid receptors
E) Diazepam at GABA receptors

86. How can the inhibition caused by a competitive antagonist be overcome?
A) By increasing the concentration of the antagonist
B) By increasing the concentration of the agonist
C) By decreasing the concentration of the agonist
D) By decreasing the concentration of the antagonist
E) By changing the receptor’s structure

87. What is the primary effect of an antagonist when no agonist is present?
A) It enhances the biological function of the receptor
B) It inhibits receptor activation by blocking agonist binding
C) It mimics the effect of the endogenous ligand
D) It activates the receptor even in the absence of an agonist
E) It has no effect on biological function

88. What is the primary characteristic of an irreversible antagonist?
A) It binds to the receptor reversibly and competes with the agonist
B) It binds covalently to the active site, permanently reducing the number of receptors available
C) It has high intrinsic activity and mimics the effect of the agonist
D) It increases the Emax without changing the EC50
E) It can be overcome by increasing the agonist concentration

89. What is the effect of an irreversible antagonist on the Emax?
A) It causes an increase in Emax
B) It causes a downward shift in Emax
C) It causes an upward shift in Emax
D) It has no effect on Emax
E) It causes a shift in Emax without affecting the EC50

90. How does an irreversible antagonist differ from a competitive antagonist?
A) Irreversible antagonists reduce agonist potency, while competitive antagonists reduce agonist efficacy
B) Irreversible antagonists cannot be overcome by increasing the concentration of the agonist, while competitive antagonists can
C) Irreversible antagonists increase the Emax, while competitive antagonists decrease it
D) Irreversible antagonists increase the EC50, while competitive antagonists decrease it
E) Irreversible antagonists mimic the effect of the agonist, while competitive antagonists block it

91. What happens when the concentration of agonist is increased in the presence of an irreversible antagonist?
A) The effect of the irreversible antagonist is reversed
B) The efficacy of the agonist is increased
C) The EC50 of the agonist decreases
D) The effect of the irreversible antagonist cannot be overcome
E) The receptor becomes more sensitive to the agonist

92. Which of the following statements about irreversible antagonists is true?
A) They can bind reversibly to receptors, affecting both potency and efficacy
B) They reduce the number of available receptors permanently by covalently binding to them
C) They reduce the agonist potency by shifting the EC50 to the right without changing Emax
D) They increase the Emax of the agonist without affecting the EC50
E) They mimic the effect of the endogenous ligand

93. What is the primary characteristic of an allosteric antagonist?
A) It binds to the agonist-binding site to prevent receptor activation
B) It binds to a site other than the agonist-binding site and prevents receptor activation
C) It mimics the agonist's effect at the receptor
D) It increases the Emax without changing the EC50
E) It decreases the EC50 without affecting the Emax

94. What is the effect of an allosteric antagonist on the Emax of an agonist?
A) It increases the Emax
B) It causes no change in the Emax
C) It causes a downward shift in the Emax
D) It causes an upward shift in the Emax
E) It increases the EC50

95. What is an example of an allosteric antagonist?
A) Picrotoxin at the GABA receptor
B) Terazosin at the α1-adrenoceptor
C) Naloxone at opioid receptors
D) Phenylephrine at α1-adrenoceptors
E) Aripiprazole at dopamine receptors

96. What happens when picrotoxin binds inside the GABA-controlled chloride channel?
A) Chloride ions can pass through the channel more easily
B) The receptor becomes activated by GABA
C) Chloride ions cannot pass through the channel, even when GABA occupies the receptor
D) The chloride channel opens without GABA binding
E) The receptor is activated by an agonist at an allosteric site

97. What is functional antagonism?
A) An antagonist that binds to the same receptor as the agonist to reduce its effect
B) An antagonist that binds to a different receptor and produces opposite effects to the agonist
C) An agonist that produces an effect opposite to that of another agonist
D) An antagonist that prevents receptor activation without affecting the agonist's action
E) An agonist that enhances the effect of another agonist

98. Which of the following is an example of functional antagonism?
A) Epinephrine causing vasodilation at α1-adrenoceptors
B) Histamine causing bronchoconstriction at H1 receptors and epinephrine causing bronchodilation at B2-adrenoceptors
C) Naloxone blocking opioid receptors
D) Terazosin blocking α1-adrenoceptors to lower blood pressure
E) Aripiprazole acting as a partial agonist at dopamine receptors
bronchodilation at B2-adrenoceptors
99. What is the mechanism of action of epinephrine in functional antagonism against histamine?

A) It binds to H1 receptors and reduces bronchoconstriction
B) It binds to B2-adrenoceptors and causes relaxation of bronchial smooth muscle
C) It blocks the effects of histamine on H1 receptors
D) It enhances the action of histamine on bronchial smooth muscle
E) It increases the production of histamine

100. What does functional antagonism demonstrate in terms of receptor interaction?
A) That two agonists can have the same effect at different receptors
B) That the same receptor can be involved in both the agonist and antagonist effects
C) That an antagonist can work by blocking the action of the agonist at a separate receptor
D) That a receptor's activity can only be affected by direct agonist binding
E) That antagonists are only effective at the same site as the agonist

101. What does a quantal dose-response curve describe?
A) The relationship between drug dose and the proportion of a population that experiences a specific effect
B) The maximum effect of a drug in an individual patient
C) The relationship between drug dose and the magnitude of response in an individual
D) The therapeutic range of a drug in a specific population
E) The total number of receptors occupied by a drug

102. In a quantal dose-response curve, the ED50 represents what?
A) The dose that causes the maximum possible response in the population
B) The dose at which all individuals respond to the drug
C) The dose that causes a therapeutic response in half of the population
D) The dose required to achieve a graded response
E) The dose at which the drug causes adverse effects in 50% of the population

103. How can graded dose-response data be transformed into quantal responses?
A) By calculating the mean dose for the entire population
B) By determining a predetermined level of graded response as the threshold for a positive response
C) By measuring the maximum response for each individual
D) By analyzing the receptor binding data for the drug
E) By examining the time it takes for a response to occur

104. Which of the following is true regarding quantal dose-response curves?
A) They represent individual variations in drug responses within a population
B) The curves are used to determine the maximum achievable effect of a drug in a population
C) The ED50 is the dose that causes the full therapeutic effect in all individuals
D) The shape of the curve is similar to that of log dose-response curves
E) Quantal dose-response curves are only useful for determining toxic doses in individuals

105. What does the therapeutic index (TI) of a drug measure?
A) The drug's effectiveness at producing a therapeutic response
B) The ratio of the dose that causes toxicity to the dose that produces the desired effect
C) The time it takes for the drug to reach its peak effect
D) The amount of drug required to occupy 50% of the receptors
E) The duration of the drug's effect on the body

106. What does a higher therapeutic index (TI) indicate about a drug?
A) The drug has a greater likelihood of causing side effects
B) The drug is more toxic at therapeutic doses
C) The drug has a wider margin between effective and toxic doses, indicating greater safety
D) The drug is more potent in small doses
E) The drug has a longer duration of action

107. What does a low therapeutic index (TI) indicate about a drug?
A) The drug has a large margin of safety between effective and toxic doses
B) The drug has a high risk of causing adverse effects at therapeutic doses
C) The drug is highly effective with minimal risk of toxicity
D) The drug's therapeutic effect is not influenced by the dose
E) The drug should not be used in clinical practice

108. Why are drugs with a low therapeutic index (TI) sometimes used in clinical practice?
A) Because they are highly effective for treating serious diseases, and the risk of adverse effects is outweighed by the risk of leaving the disease untreated
B) Because they are safer than drugs with high TI
C) Because they have no risk of toxicity
D) Because they have a predictable and non-overlapping effective and toxic dose range
E) Because they have minimal side effects

109. Which of the following is an example of a drug with a low therapeutic index?
A) Penicillin
B) Warfarin
C) Ibuprofen
D) Paracetamol
E) Aspirin

110. What does clinical experience with drugs that have a low therapeutic index help determine?
A) The range of doses that are most effective in treating diseases
B) The optimal time for administering the drug
C) The exact number of adverse effects associated with the drug
D) The safe margin between effective and toxic doses based on real-world use
E) The drug's interaction with other medications

111. Why is warfarin considered a drug with a low therapeutic index (TI)?
A) Its dose-response curve shows a wide margin between therapeutic and toxic doses
B) Small changes in dose or bioavailability can cause significant changes in therapeutic effects, leading to serious side effects
C) It has a high degree of effectiveness with minimal adverse effects
D) The therapeutic effect can be easily predicted in all patients
E) It has a large margin of safety, so the risk of toxicity is low

112. What is the desired therapeutic effect of warfarin in patients?
A) A decrease in blood pressure by 10 mmHg
B) A two- to threefold increase in the international normalized ratio (INR)
C) A rapid increase in heart rate
D) A reduction in cholesterol levels
E) A fivefold increase in platelet aggregation

113. What is a potential risk of increasing the dose of warfarin too much?
A) The drug becomes ineffective in preventing clot formation
B) It can cause hemorrhage due to excessive anticoagulation in some patients
C) It may lead to hypertension
D) It may cause liver toxicity
E) It may cause hyperglycemia

114. Why is bioavailability critically important for drugs with a low therapeutic index like warfarin?
A) Because even small changes in absorption can cause significant alterations in the drug's effects
B) It determines the half-life of the drug
C) It regulates the drug’s ability to bind to receptors
D) It affects the metabolism rate of the drug
E) It only impacts the drug’s side effect profile

115. Why does penicillin have a large therapeutic index?
A) It has a narrow margin between the effective and toxic doses
B) The therapeutic effects of penicillin are highly dependent on bioavailability
C) It is safe to administer doses much higher than the minimum required to achieve a desired effect without significant risk of adverse effects
D) It has no side effects regardless of the dose
E) The drug’s effects are unpredictable, even at therapeutic doses

116. For drugs like penicillin with a large therapeutic index, what role does bioavailability play in clinical use?
A) Bioavailability is critical in determining the therapeutic effect
B) Changes in bioavailability significantly alter the clinical effects of the drug
C) Bioavailability does not critically affect the therapeutic or clinical effects of the drug
D) It directly determines the rate at which the drug is metabolized
E) Bioavailability is irrelevant to the overall treatment outcome