PHARMACODYNAMICS

Lecture – 3

Second Semester

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Course Description

This course introduces the fundamental principles of pharmacology, focusing on:

- Drug classification systems
- Mechanisms of drug action
- Pharmacokinetics (ADME)
- Pharmacodynamics
- Drug–drug interactions
- Toxicology and drug safety

Week	Topic
1	Introduction to Pharmacology
2	Pharmacokinetics (ADME)
3	Pharmacodynamics
4	Steroid & Non-Steroid Drugs
5	Nervous System Pharmacology
6	Cardiovascular Pharmacology
7	Antimicrobial Agents
8	Endocrine & Metabolic Drugs
9	Hematology & Chemotherapy
10	General Toxicology
11	Clinical Toxicology & Drug Safety
12	Student Presentations & Review

COURSE SYLLABUS

Outline

- Outline the processes studied by pharmacodynamics.
- Describe how the drug’s pharmacological characteristics affect its therapeutic effect.
- Explain how interactions with other drugs, food and diseases affect the drug's effectiveness.

Learning Objectives

- Overview of Pharmacodynamics
- Mechanisms of drug actions
- Pharmacological characteristics
- Drug interactions

Overview of pharmacodynamics

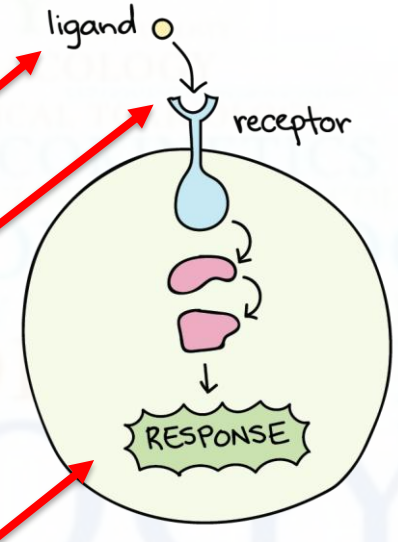
PD is what the drug does to the body

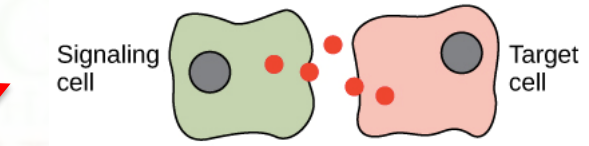
- **Mechanism of drug action**
  - drug-receptor interaction
  - post-receptor effect
- **Pharmacological characteristics**
  - therapeutic effect
  - drug interactions

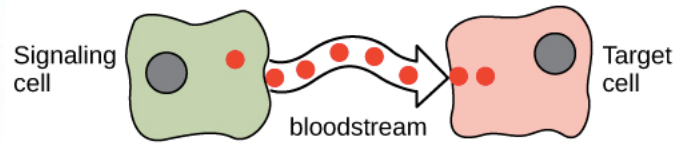
Mechanism Of Drug Action

Drug Action

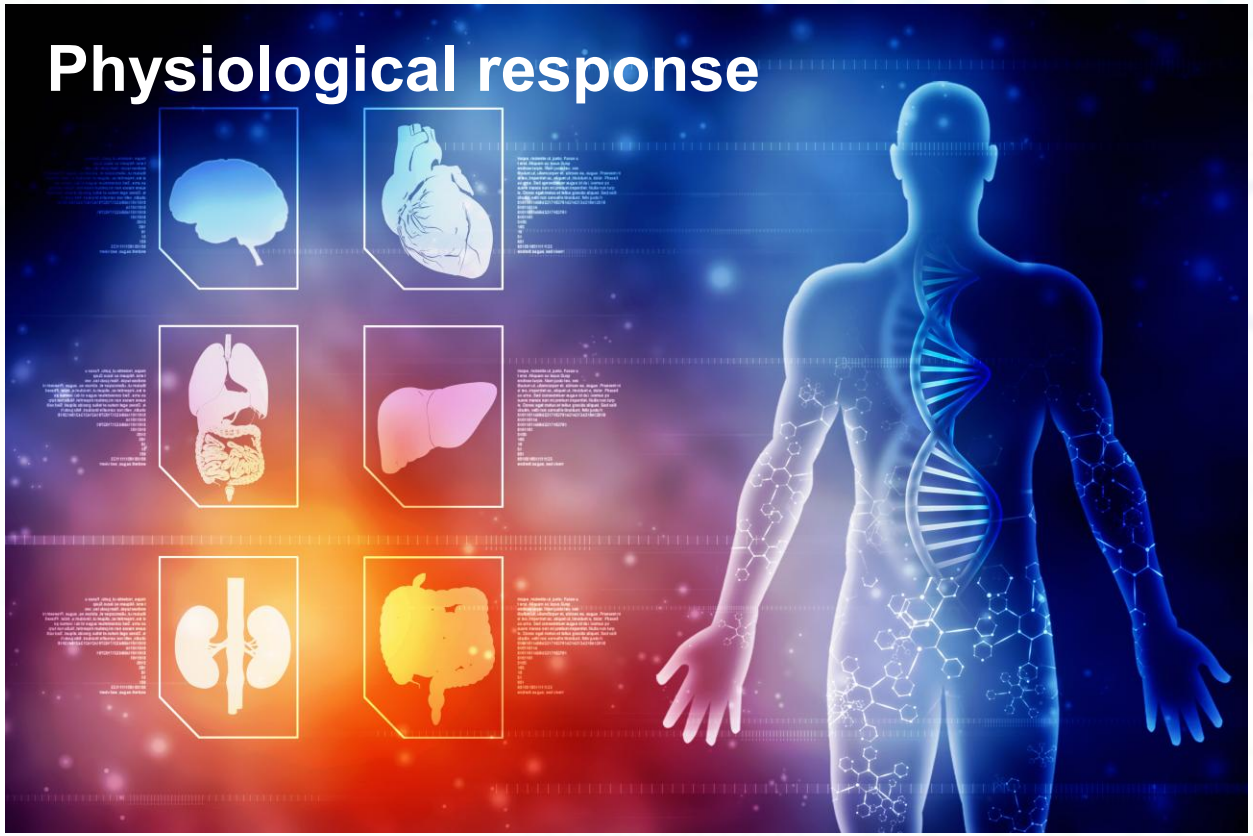
- **Ligand** is any molecule which attaches selectively to particular receptors.
- Most drugs are ligands, i.e. they bind to cellular receptors.
- Drug-receptor binding triggers a chain of changes:
  - Initiate biochemical reactions inside the cell, i.e. signal transduction
  - Communication between cells – local or distant
  - Alteration in the associated physiological processes

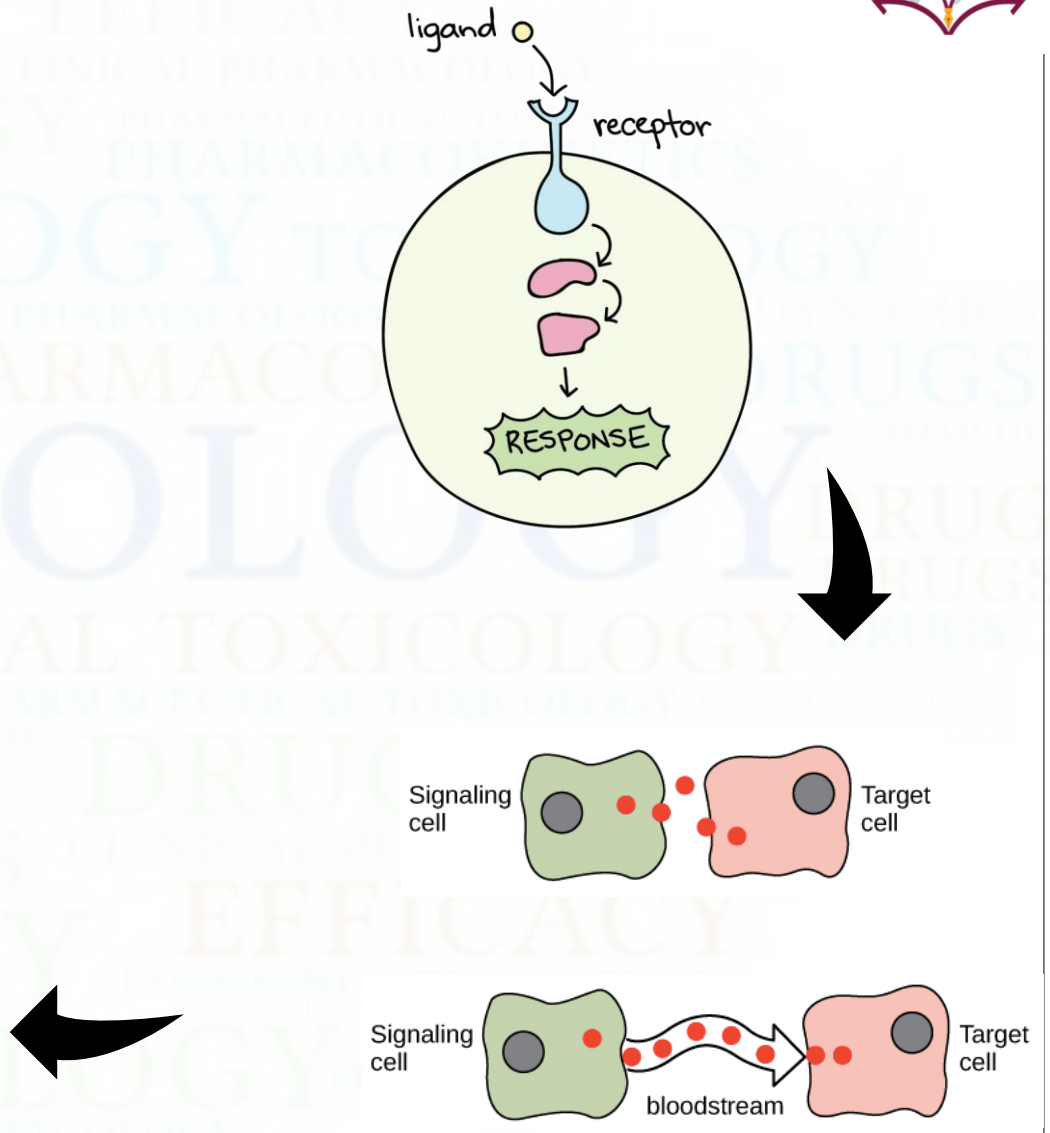






Drug Action

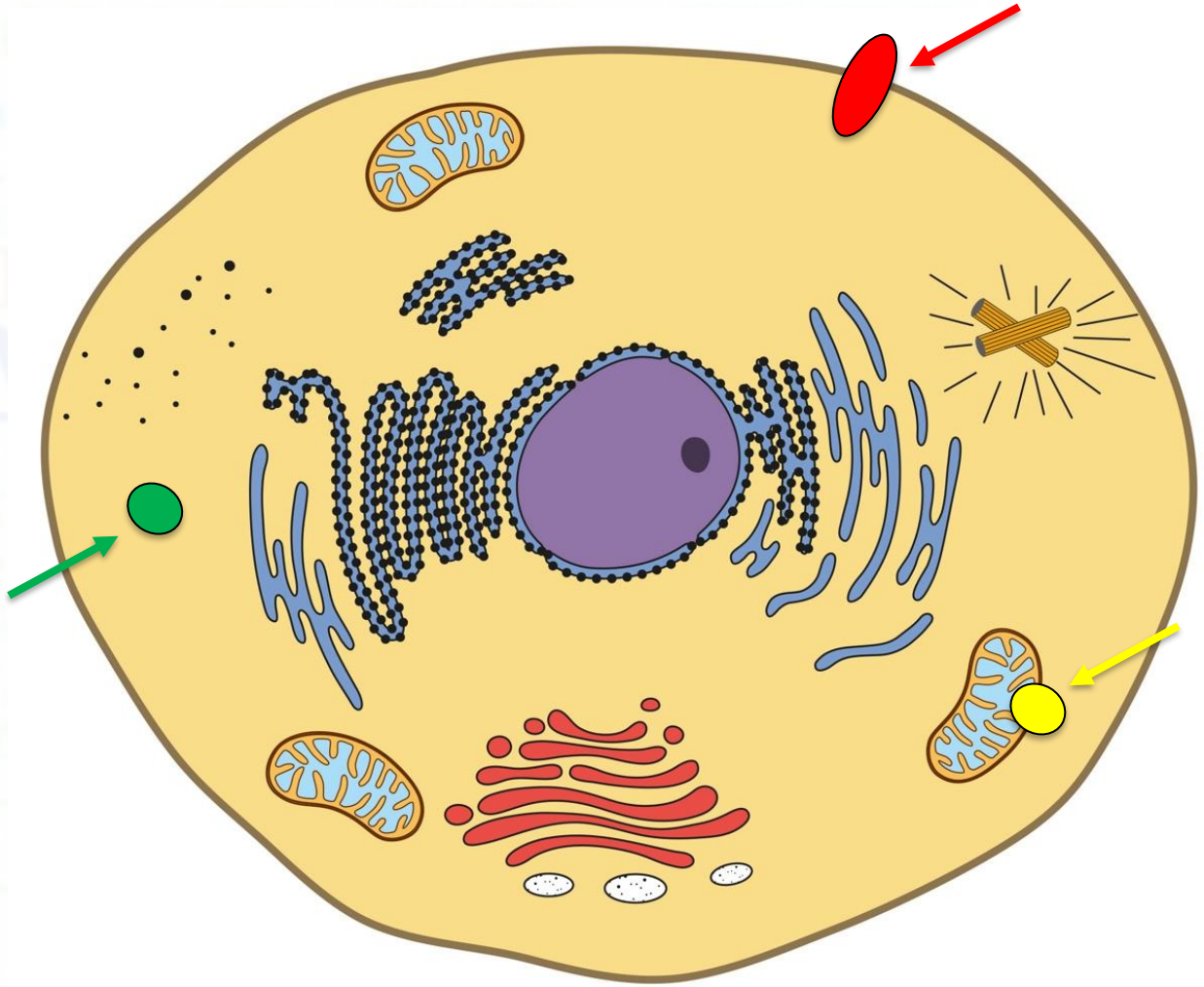

Physiological response


ligand
receptor
RESPONSE
Signaling cell
Target cell
Signaling cell
bloodstream
Target cell

Receptors

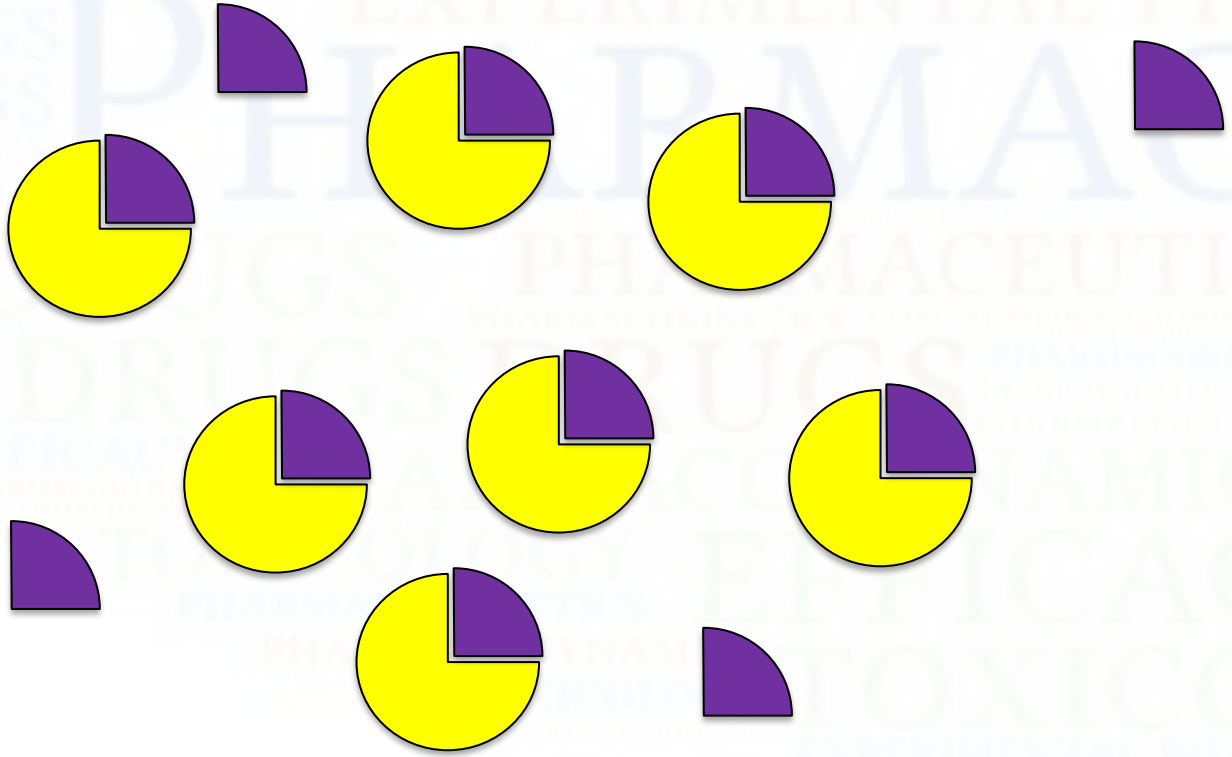
Proteins or glycoproteins

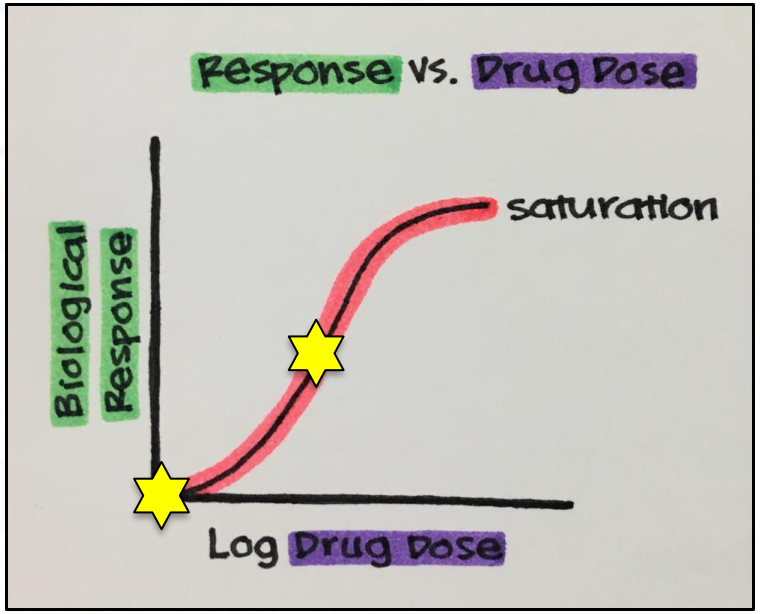
- present on the cell membrane
- on an organelle within the cell
- free in the cytoplasm



Receptors

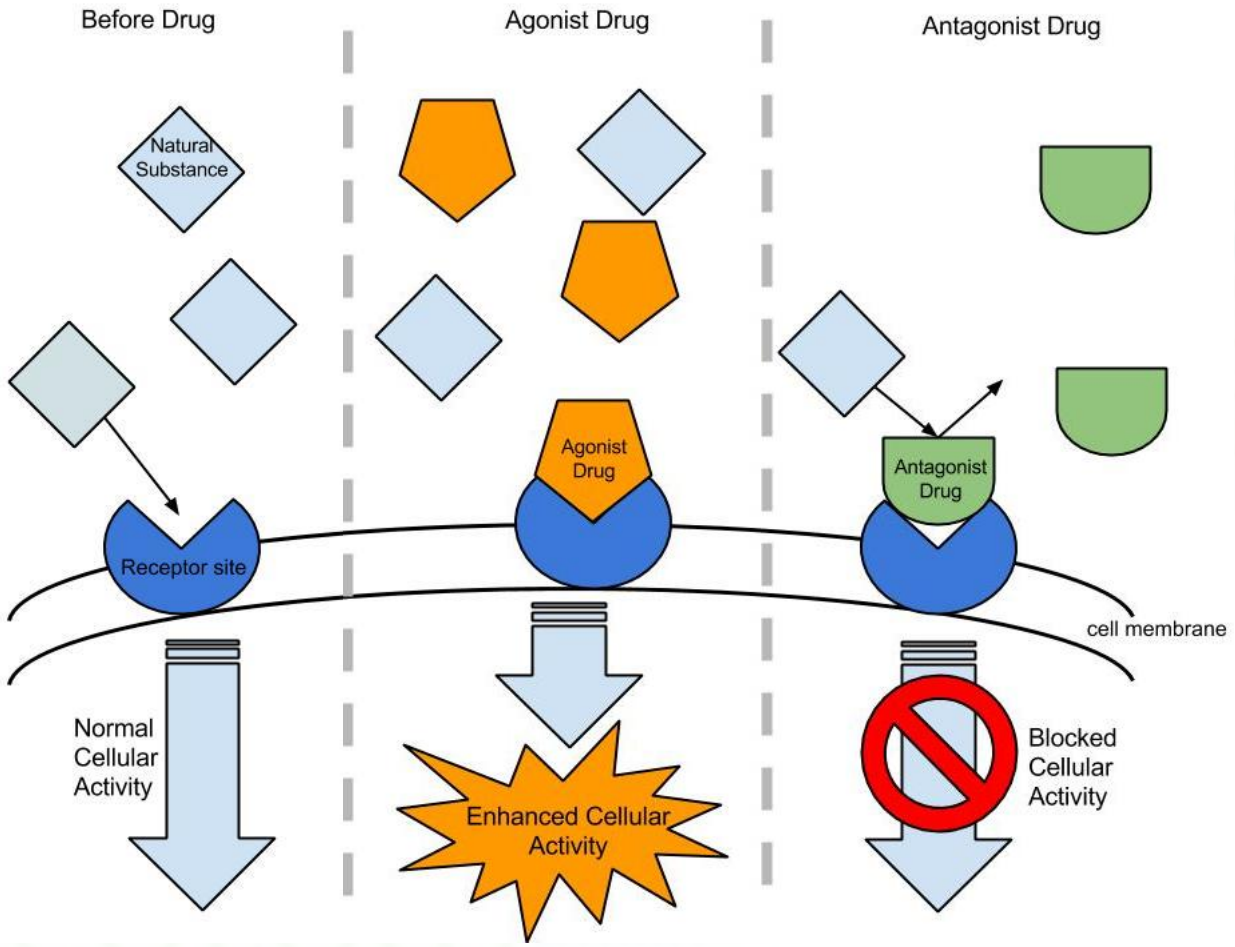
- Every cell has a finite number of receptors.
- Receptor mediated responses plateau upon saturation of all receptors.





Effect on Cellular Processes

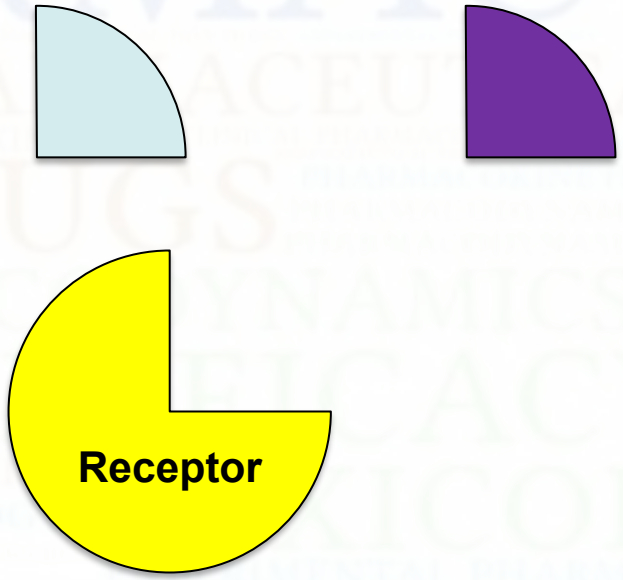
- **Agonists**
  - enhance the relevant cellular process
  - partial agonists enhance the process but don't produce the maximum effect even when all of the receptors are occupied
- **Antagonists**
  - inhibit or block the relevant cellular function



Types of Antagonists

COMPETITIVE ANTAGONISTS

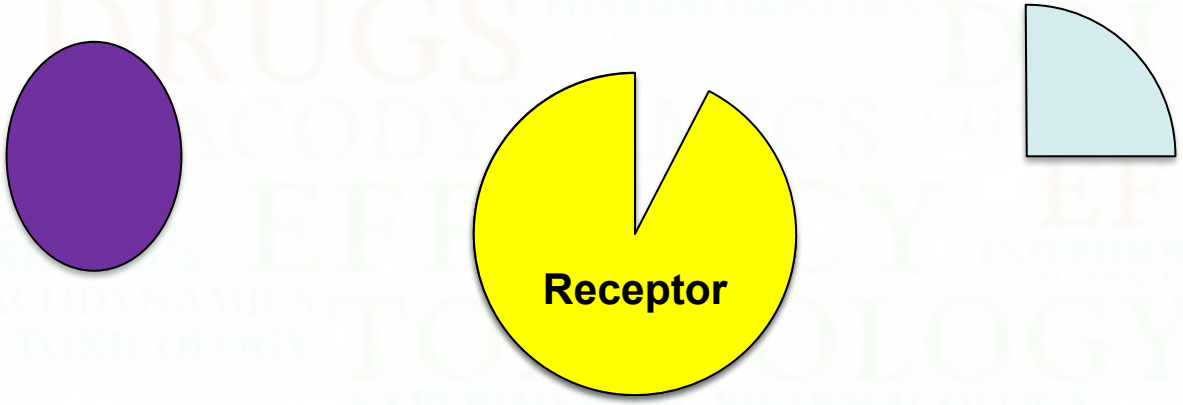
- Compete with a natural ligand or drug-agonist for receptors.
- High doses of an agonist can generally overcome antagonist.



Types of Antagonists

NONCOMPETITIVE ANTAGONISTS, aka ALLOSTERIC ANTAGONISTS

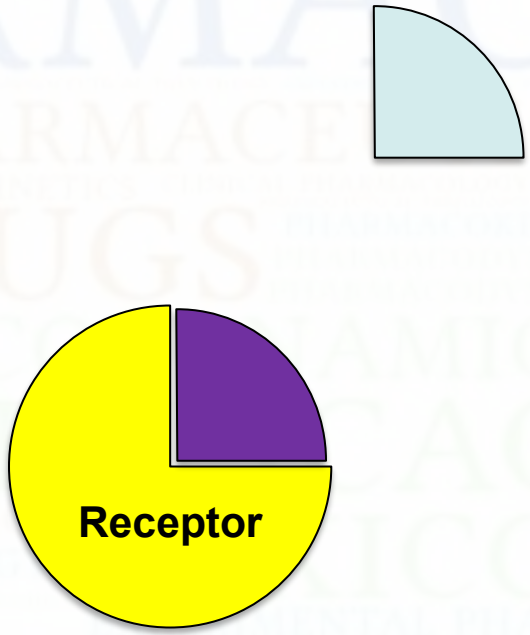
- Bind to a site other than the agonist-binding domain
- Induce a conformation change in the receptor such that the agonist no longer "recognizes" the agonist binding site.
- High doses of an agonist do not overcome the antagonist in this situation.



Types of Antagonists

IRREVERSIBLE ANTAGONISTS

- Bind permanently to the receptor binding site.
- Therefore, their action cannot be overcome by agonists.



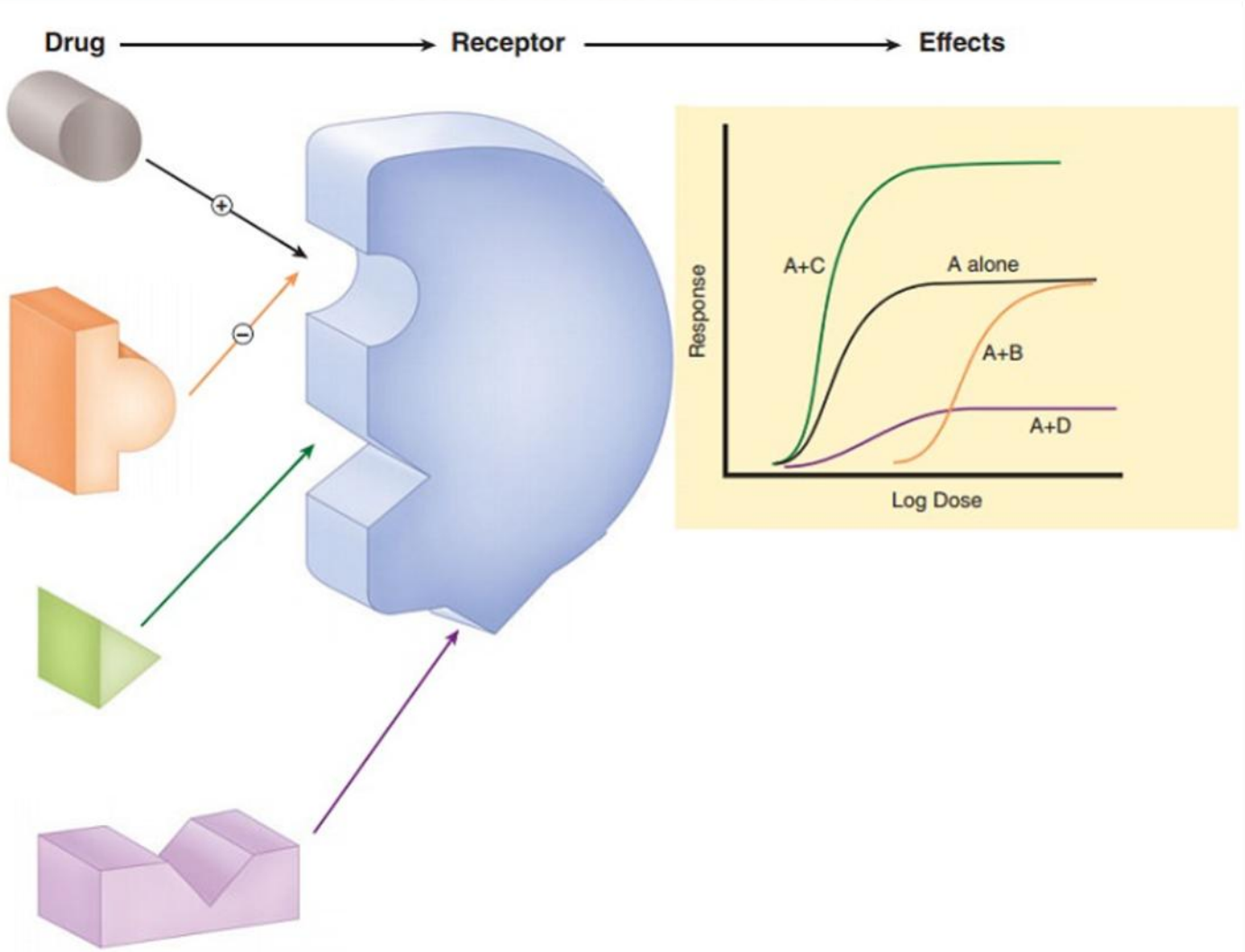
Response to Agonists & Antagonists

A. natural ligand

B. competitive antagonist

C. allosteric agonist

D. allosteric antagonist



Post-receptor Effect

The binding between a drug and a receptor can affect various post-receptor pathways. For example, drug-receptor interactions can lead to:

- Opening or closing ion channels
- Activation of a secondary messenger
  - cAMP, cGMP, Ca^{++}, inositol phosphates, etc.
  - Initiates a series of chemical reactions
- Inhibition or enhancement of a normal cellular process
- Complete turn off of a cellular process

Play

Pharmacological Characteristics

Characteristics of Drug-Receptor Binding

AFFINITY

- Refers to the strength of binding between a drug and a receptor.
- High affinity means the drug can easily bind to the receptor.
- Affinity determines how much drug is required for a physiological effect.

SPECIFICITY

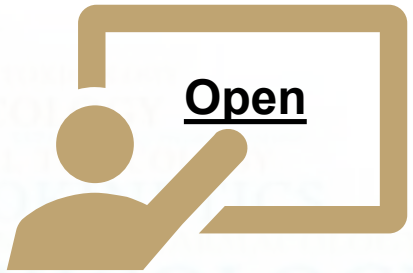
- The ability of a drug to selectively interact with a single type of receptor.
- High specificity means the drug binds one or very few receptors.
- Specificity correlates with the number of side effects.

Physiological Effect of Drugs

- **POTENCY**
  - Amount of drug required to produce 50% of its maximum response
  - Used to compare compounds within classes of drugs
- **EFFICACY**
  - Degree to which a drug is able to produce the desired response

Play

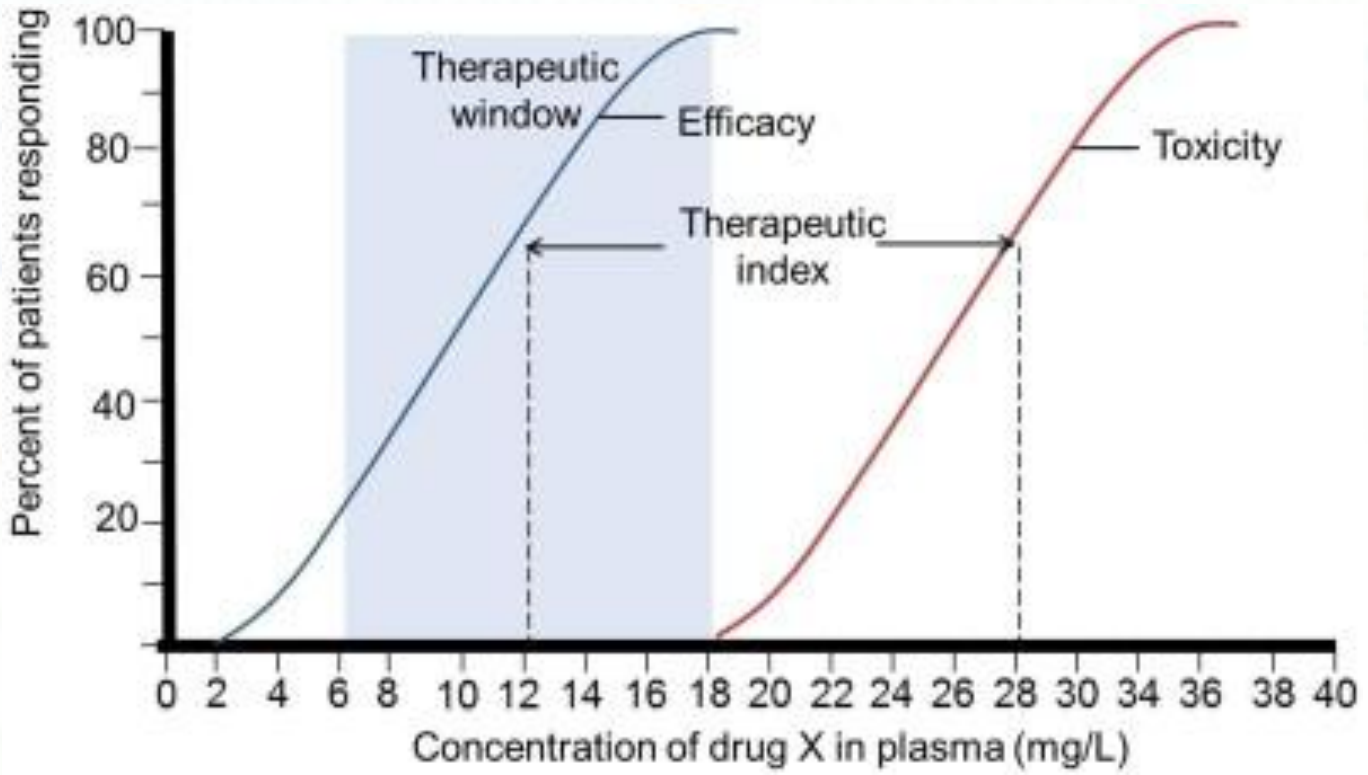
Physiological Effect of Drugs



- **EFFECTIVE CONCENTRATION 50% (EC_{50})**
  - Concentration of the drug which induces a specified clinical effect in 50% of subjects
- **LETHAL DOSE 50% (LD_{50})**
  - Concentration of the drug which induces death in 50% of subjects
- **THERAPEUTIC INDEX**
  - Measure of the safety of a drug
  - Shows the ration between lethal dose and effective concentration
  - Calculation: LD_{50}/EC_{50}

Dose-Response Effect

- Drug induced responses are not an “all or nothing” phenomenon
- Increase in dose may:
  - Increase therapeutic response
  - Increase risk of toxicity
  - Therapeutic window is the range of doses with highest therapeutic effect and minimal adverse effect



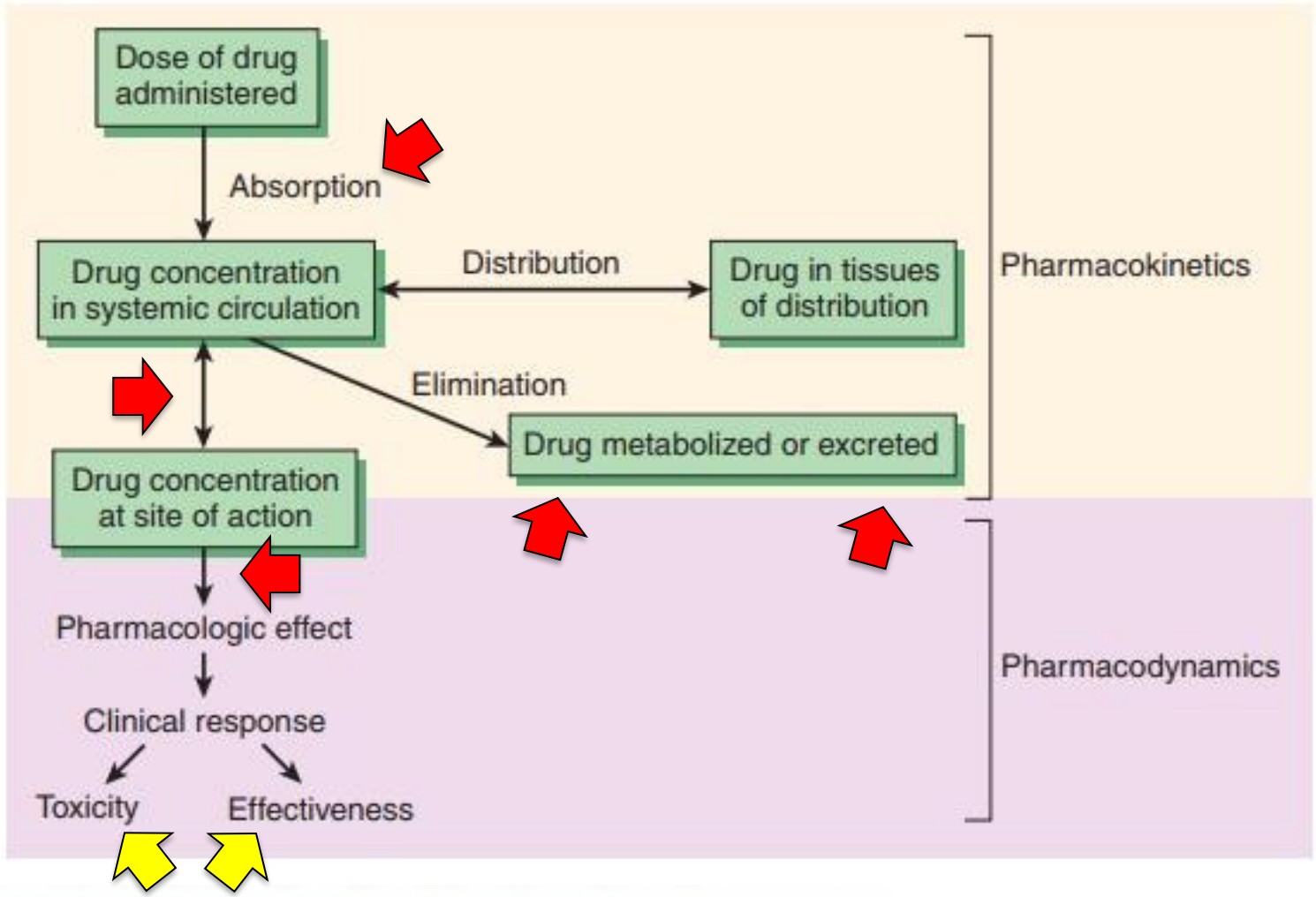
Drug interactions

Drug-Drug Interactions

Presence of other drugs can affect the PK & PD of the drug of interest:

- absorption
- metabolism
- distribution
- elimination
- receptor binding

Drug interactions can affect **toxicity** & **effectiveness.**



Drug-Food Interactions

- Food is essentially a mixture of drugs, each of which has the potential to interfere with medical drugs.
- Food can affect the same processes of PK and PD as drugs: ADME, drug-receptor binding, and post-receptor processes.
- These drugs are commonly affected by food:
  - antihypertensive drugs
  - antibiotics
  - analgesics & antipyretics
  - bronchodilators
  - antihistamines

Play

Drug-Disease Interactions

ABSORPTION

- Enterally administered drugs may have altered absorption due to:
  - alterations in gastric pH,
  - edema of GI mucosa,
  - delayed or enhanced gastric emptying,
  - alterations in blood flow,
  - presence of an ileus.

Drug-Disease Interactions

DISTRIBUTION

- Altered organ perfusion due to hemodynamic changes affects the transport of drugs from the blood to the organs.
- Inflammation & changes in capillary permeability may enhance the delivery of drugs to a site.

Drug-Disease Interactions

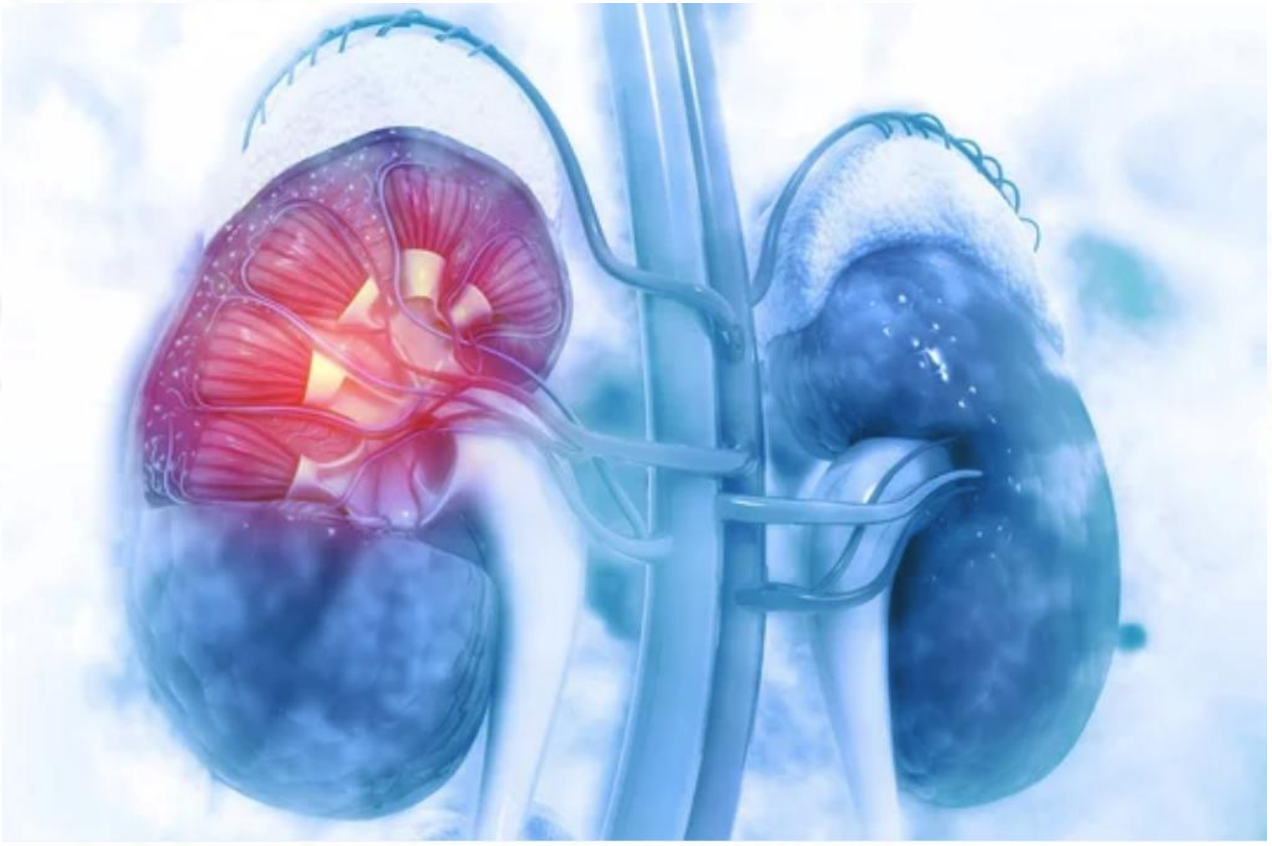
METABOLISM

- Infection & inflammation can decrease drug metabolism, which may prolong the half-life of the drug and the duration of drug action.
- Altered hepatic function directly affects drug metabolism.

Drug-Disease Interactions

ELIMINATION

- Liver diseases such as cirrhosis can affect the metabolism and excretion of drugs.
- Biliary excretion may also be altered by conditions such as obstructive jaundice.
- In compromised renal function, excretion is diminished, which can reduce the clearance of drugs.



Drug-Disease Interactions

RECEPTOR BINDING

- Genetic mutations can change receptor binding, alter the level of binding proteins, or decrease receptor sensitivity.
- Malnutrition can decrease the level of albumin, a protein that binds to many drugs.
- In insulin-resistant diabetes mellitus, insulin receptor sensitivity is decreased, which affects drug-receptor binding and alters the pharmacodynamic effects of insulin.

Pharmacology in Clinical Trials

- PK & PD inform the optimal route of administration, dosage & regimen.
- Pharmacological data help reduce animal testing, shorten the drug development time & help prevent suboptimal study designs in Phase II and III trials by minimizing costs, maximizing efficacy & minimizing toxicity.
- Pharmacometrics utilizes PK and PD data to generate models for studying drug efficacy to guide study design, efficacy comparisons & dose optimization.

Play

Question Samples

Pharmacokinetics (Lecture 2 – ADME)

1. Pharmacokinetics studies:

A) What the drug does to the body
B) Drug toxicity only
C) What the body does to the drug
D) Drug classification

Answer: C

2. Which of the following is NOT part of ADME?

A) Absorption
B) Distribution
C) Metabolism
D) Receptor binding

Answer: D

3. First-pass metabolism mainly occurs in:

A) Kidney
B) Liver
C) Lung
D) Brain

Answer: B

4. First-pass metabolism reduces:

A) Drug efficacy
B) Drug bioavailability
C) Drug potency
D) Drug affinity

Answer: B

Question Samples

5. Lipid-soluble drugs are usually:
A) Rapidly excreted by kidney
B) Converted to water-soluble metabolites in liver
C) Not metabolized
D) Eliminated unchanged
Answer: B

6. The kidney primarily eliminates:
A) Lipid-soluble bound drugs
B) Polar unbound drugs
C) Protein-bound drugs
D) Non-metabolized fat-soluble drugs
Answer: B

7. Which route bypasses first-pass metabolism?
A) Oral
B) Rectal
C) Intravenous
D) Enteral
Answer: C

8. Drug distribution depends on:
A) Blood flow
B) Protein binding
C) Membrane transport
D) All of the above
Answer: D

Question Samples

9. LC-MS is used to:
A) Measure blood pressure
B) Identify drug fragments by mass-to-charge ratio
C) Detect glucose
D) Measure heart rate
Answer: B

10. Enterohepatic recycling refers to:
A) Renal excretion
B) Drug reabsorption from bile
C) Lung elimination
D) Protein binding
Answer: B

Pharmacodynamics (Lecture 3)
11. Pharmacodynamics studies:
A) Drug elimination
B) Drug absorption
C) What the drug does to the body
D) Drug excretion
Answer: C

12. A ligand is:
A) A toxic metabolite
B) A molecule that binds selectively to a receptor
C) An enzyme
D) A transporter protein
Answer: B

Question Samples

13. When all receptors are occupied, the response:
A) Increases infinitely
B) Plateaus
C) Decreases immediately
D) Stops metabolism
Answer: B

14. A partial agonist:
A) Produces maximum effect
B) Produces no effect
C) Produces less than maximal effect even when all receptors are occupied
D) Blocks receptors permanently
Answer: C

15. A competitive antagonist:
A) Binds irreversibly
B) Binds at a different site
C) Can be overcome by high agonist concentration
D) Increases efficacy
Answer: C

16. An irreversible antagonist:
A) Can be displaced by agonist
B) Binds permanently to receptor
C) Is always competitive
D) Has no effect
Answer: B

Question Samples

17. Affinity refers to:
A) Maximum drug response
B) Drug toxicity
C) Strength of drug-receptor binding
D) Drug metabolism rate
Answer: C

18. Potency is defined as:
A) Maximum effect a drug can produce
B) Amount of drug needed to produce 50% of maximum effect
C) Drug half-life
D) Drug elimination rate
Answer: B

18. Potency is defined as:
A) Maximum effect a drug can produce
B) Amount of drug needed to produce 50% of maximum effect
C) Drug half-life
D) Drug elimination rate
Answer: B

19. Therapeutic Index is calculated as:
A) EC50 / LD50
B) LD50 / EC50
C) Dose / Time
D) Absorption × Distribution
Answer: B

Question Samples

20. Noncompetitive (allosteric) antagonists:

A) Bind to the same site as agonist

B) Can be overcome by high agonist dose

C) Bind to a different site and change receptor shape

D) Increase receptor sensitivity

Answer: C

References

• **Brunton LL, Hilal-Dandan R, Knollmann BC.**
Goodman & Gilman's The Pharmacological Basis of Therapeutics. 14th ed. McGraw-Hill; 2023.
→ Gold standard reference for mechanism of action, receptor pharmacology, dose–response, therapeutic index.

• **Rang HP, Ritter JM, Flower RJ, Henderson G.**
Rang & Dale's Pharmacology. 10th ed. Elsevier; 2023.
→ Excellent for receptor theory, agonists/antagonists, competitive vs noncompetitive inhibition.

• **Katzung BG, Trevor AJ.**
Basic & Clinical Pharmacology. 15th ed. McGraw-Hill; 2021.
→ Very suitable for undergraduate students; strong explanations of EC50, LD50, therapeutic window.

• **Neal MJ.**
Medical Pharmacology at a Glance. Wiley-Blackwell; latest edition.
→ Good for simplified conceptual understanding.