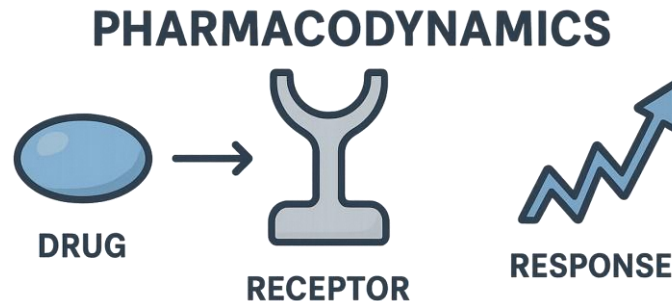


General Pharmacology

Pharmacodynamics

- Pharmacodynamics refers to what the drug does to the body – including the biochemical and physiological effects of drugs and their mechanisms of action.



Principles of Drug Action

1. Stimulation

- Increases the activity of a specific organ or system.
- Example:** Adrenaline stimulates β_1 -receptors to increase heart rate and contractility.

2. Depression

- Decreases the activity of a body system or function.
- Example:** Barbiturates depress the CNS leading to sedation or anaesthesia.

3. Irritation

- Causes mild inflammation or discomfort that leads to a therapeutic response.
- Example:** Capsaicin cream causes irritation that helps relieve pain by depleting substance P.

4. Replacement

- Supplies deficient substances in the body.
- Example:** Insulin in diabetes, thyroxine in hypothyroidism.

5. Cytotoxic Action

- Drugs that selectively kill specific cells, often used in cancer treatment.
- Example:** Chemotherapeutic agents like cyclophosphamide destroy malignant cells.

Mechanisms of Drug Action

Drugs exert their effects by interacting with specific targets:

1. Enzymes

- Drugs may **inhibit** or **activate** enzymes.
- Inhibitors:** Aspirin irreversibly inhibits COX enzymes.

- **Activators:** Some drugs enhance enzyme activity (less common).
- **Example:** Neostigmine inhibits acetylcholinesterase, increasing acetylcholine.

2. Ion Channels

- Drugs can block or open ion channels to affect ion flow across membranes.
- **Example:** Lidocaine blocks sodium channels, preventing nerve conduction (local anesthesia).

3. Transporters (Carriers)

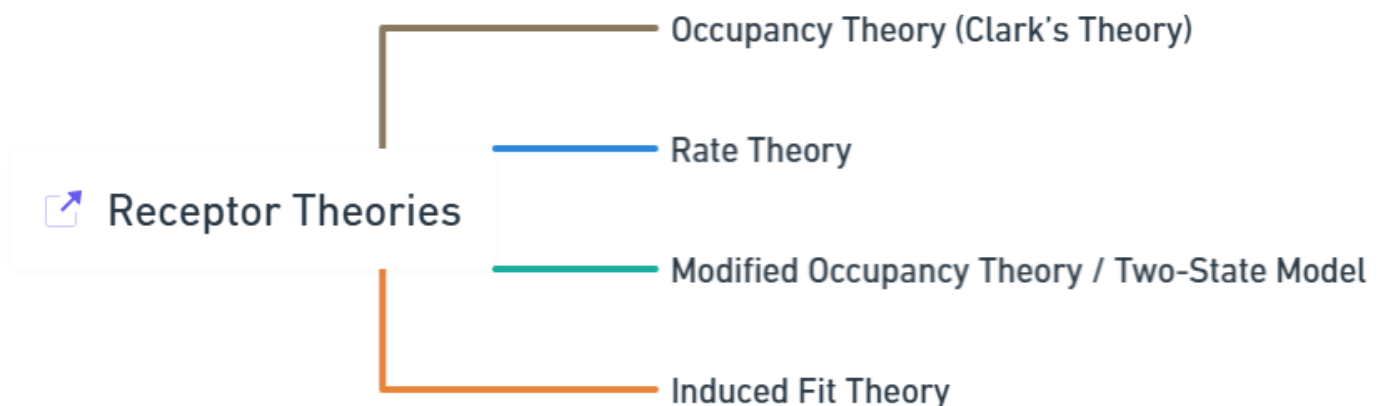
- Drugs can inhibit transporters involved in the movement of substances across membranes.
- **Example:** SSRIs like fluoxetine inhibit serotonin reuptake transporters.

4. Receptors

- The most common drug targets.
- Drugs may act as:
 - **Agonists:** Activate receptors (e.g., Salbutamol on β_2 receptors).
 - **Antagonists:** Block receptors (e.g., Propranolol on β receptors).
 - **Partial agonists:** Partially stimulate receptors (e.g., Buprenorphine).
 - **Inverse agonists:** Reduce basal receptor activity (e.g., Rimonabant on CB1).

Receptor Theories

- These theories explain how drugs interact with receptors and how this leads to a response:
- Historically, various theories have been proposed to explain how drugs interact with receptors and elicit responses:



1. Occupancy Theory (Clark's Theory)

- The intensity of a drug's effect is proportional to the number of receptors occupied by the drug.
- Maximal effect (E_{max}) is achieved when all receptors are occupied.

- Limitations: Does not fully explain partial agonists (drugs that can occupy all receptors but still do not produce a full response).

2. Rate Theory

- Suggests that the response depends on the rate of association and dissociation of the drug-receptor complex.
- Helps to explain why some drugs have an effect that lasts beyond the time they are bound to the receptor.

3. Modified Occupancy Theory / Two-State Model

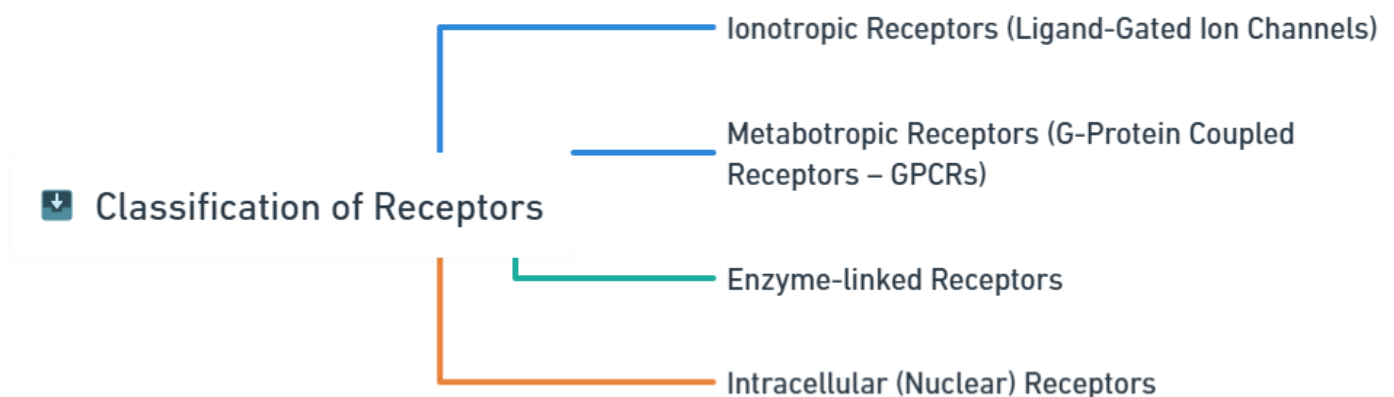
- Proposes receptors exist in two conformations: an inactive (R) and an *active* (R)* state.
- Agonists stabilize the receptor in its active conformation (R*).
- Antagonists do not stabilize either state, preventing activation.
- Inverse agonists stabilize the inactive conformation (R), reducing intrinsic activity.

3. Induced Fit Theory

- The **receptor changes its conformation** upon binding the drug (like a flexible "lock and key").
- Explains how drug binding can activate or inhibit a receptor.

Classification of Receptors

Receptors are classified based on their structure and signal transduction mechanism:



1. Ionotropic Receptors (Ligand-Gated Ion Channels)

- Response in milliseconds.
- **Example:** Nicotinic acetylcholine receptor, GABA-A receptor.

2. Metabotropic Receptors (G-Protein Coupled Receptors – GPCRs)

- Response in seconds.
- Involves G-proteins and second messengers (like cAMP, IP3).
- **Example:** β -adrenergic receptors, muscarinic receptors.



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