STEROID & NON-STEROID DRUGS

Tishk International University
Faculty of Applied Science
Medical Analysis Department

Lecture – 4
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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

- Definition and introduction
- Classification
- Mechanism of action
- Clinical use

Learning Objectives

- Define steroid and non-steroid drugs (NSAIDs).
- Classify based on structure and selectivity.
- Explain their mechanism of action
- Describe their main clinical uses.

Steroid & Non-Steroid Drugs

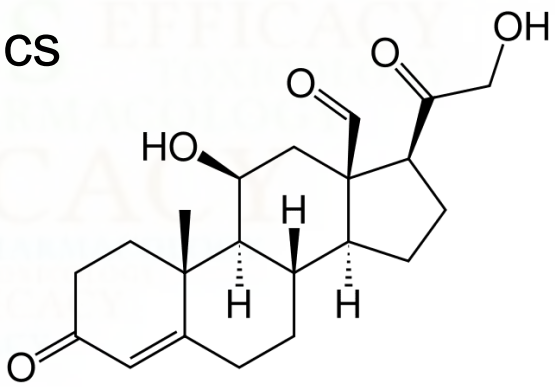
Introduction to Steroid

Steroid Drugs

- Derived from cholesterol backbone
- Lipid-soluble → cross cell membrane easily
- Act via intracellular (nuclear) receptors → gene transcription modulation
- Widely used in inflammation, autoimmune diseases, cancer, and hormone replacement therapy

Steroid & Basic Structure

- Steroid drugs are synthetic or natural compounds that mimic endogenous steroid hormones
- They regulate:
  - Metabolism
  - Immune responses
  - Electrolyte balance
  - Reproductive functions
- Core structure: 4 fused carbon rings
- Modifications determine:
  - Biological activity
  - Potency
  - Pharmacokinetics



Classification of Steroid Drugs

Main Classes:

- **Corticosteroids**
  - Glucocorticoids
  - Mineralocorticoids
- **Sex Steroids**
  - Androgens
  - Estrogens
  - Progestins
- **Anabolic Steroids**

Classification of Steroid Drugs

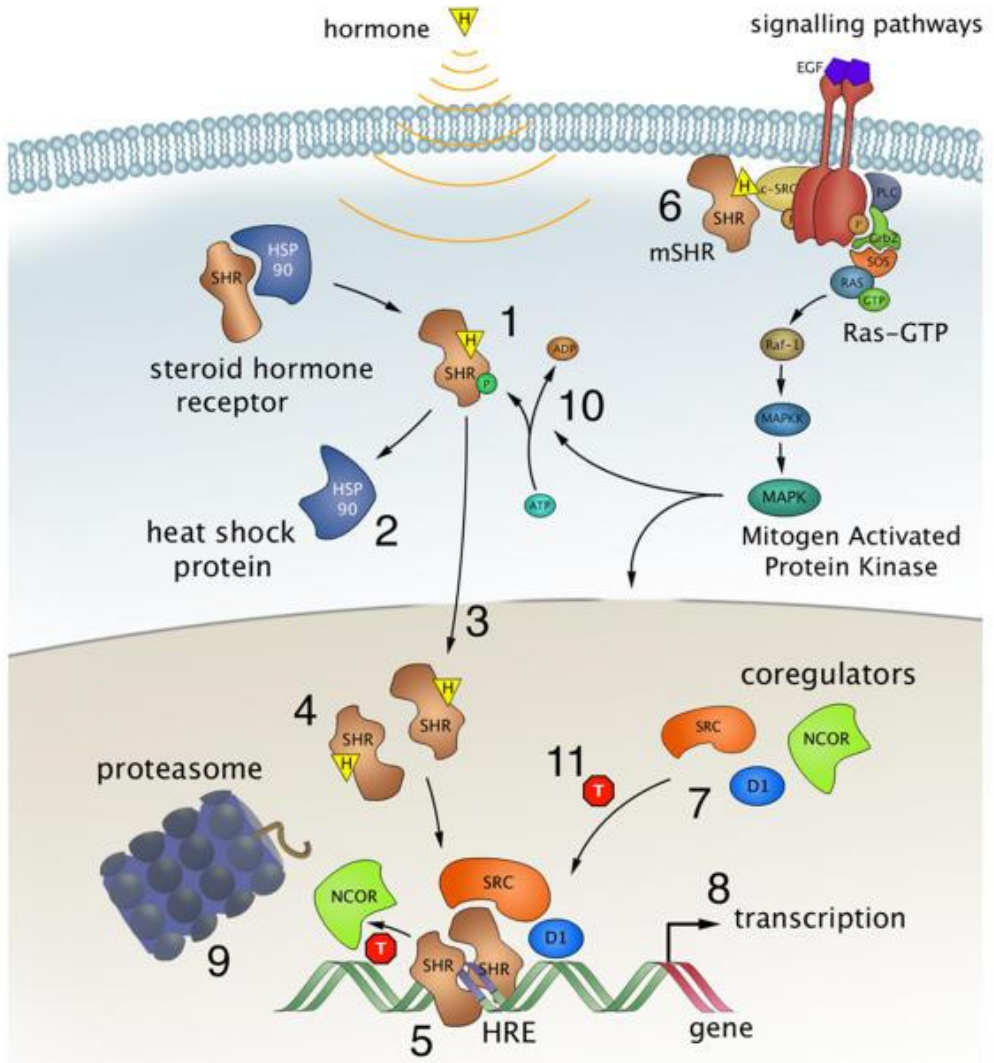
Class	Examples	Primary Functions
Glucocorticoids	Prednisone, Dexamethasone, Hydrocortisone	- Anti-inflammatory - Immunosuppressive - Regulate glucose metabolism
Mineralocorticoids	Aldosterone (physiological), Fludrocortisone	- Regulate electrolyte balance (Na^+ retention, K^+ excretion) - Maintain blood pressure
Sex Steroids	Testosterone (androgen), Estradiol (estrogen), Progesterone (progestin)	- Reproductive development - Secondary sexual characteristics - Menstrual cycle regulation
Anabolic Steroids	Nandrolone, Stanozolol, Oxandrolone	- Promote muscle growth - Increase protein synthesis - Enhance physical performance

Classification of Steroid Drugs

Class	Mechanism of Action	Clinical Uses
Glucocorticoids	Bind intracellular glucocorticoid receptors → regulate gene transcription (↓ pro-inflammatory cytokines, ↑ anti-inflammatory proteins)	- Asthma - Rheumatoid arthritis - Autoimmune diseases - Cancer therapy
Mineralocorticoids	Bind mineralocorticoid receptors in kidney → increase Na^+ reabsorption and water retention	- Addison's disease - Adrenal insufficiency
Sex Steroids	Bind nuclear receptors → regulate genes involved in reproductive and endocrine functions	- Hormone replacement therapy - Contraception - Infertility treatment
Anabolic Steroids	Bind androgen receptors → stimulate protein synthesis and muscle growth	- Muscle wasting diseases - Chronic illness recovery

Mechanism of Action o

Step	Process	Molecular Details
1	**Passive diffusion**	Steroid drugs are lipophilic and diffuse across the phospholipid bilayer without transporters
2	**Receptor binding**	Steroid binds to inactive intracellular receptor in cytoplasm or nucleus
3	**Receptor activation**	Binding causes conformational change and dissociation of heat shock proteins
4	**Nuclear translocation**	Steroid–receptor complex moves into nucleus (if cytoplasmic receptor)
5	**Dimerization**	Two steroid–receptor complexes form a homodimer
6	**DNA binding**	Complex binds to specific DNA sequences called Hormone Response Elements (HREs)
7	**Transcription regulation**	Complex recruits coactivators or corepressors to regulate gene transcription
8	**mRNA synthesis**	Target gene is transcribed into mRNA
9	**Protein synthesis**	mRNA translated into proteins in cytoplasm
10	**Cellular response**	Newly synthesized proteins produce biological effects



Steroid drugs enter cells, bind intracellular receptors, move to th and produce proteins that alter cell function.

Introduction to Non-Steroid Drugs

- Non-steroid drugs are non-lipid compounds that do not have the steroid nucleus
- Most commonly refer to NSAIDs (Non-Steroidal Anti-Inflammatory Drugs)
- Widely used for:
  - Pain relief
  - Fever reduction
  - Anti-inflammatory effects

Classification of Non-Steroid Drugs (Based on Chemical Class)

Chemical Class	Examples	COX Selectivity	Mechanism of Action	Common Clinical Uses
Salicylates	Aspirin	Non-selective (COX-1 > COX-2)	Irreversible inhibition of COX enzymes → ↓ Prostaglandins & ↓ Thromboxane A2	Pain, fever, inflammation, antiplatelet therapy
Propionic Acid Derivatives	Ibuprofen, Naproxen	Non-selective	Reversible COX inhibition → ↓ Prostaglandin synthesis	Mild–moderate pain, arthritis, dysmenorrhea
Acetic Acid Derivatives	Diclofenac, Indomethacin, Ketorolac	Non-selective (some COX-2 preference)	Reversible COX inhibition	Acute pain, gout, RA
Oxicams (Enolic Acids)	Piroxicam, Meloxicam	Preferential COX-2 (Meloxicam)	Long-acting COX inhibition	Chronic arthritis
Fenamates	Mefenamic acid	Non-selective	COX inhibition	Dysmenorrhea
Para-aminophenol Derivative	Paracetamol	Weak peripheral COX inhibition	Central COX inhibition (CNS) → ↓ fever & pain	Fever, mild pain

Classification of Non-Steroid Drugs (Based on COX Selectivity)

Categoryc	Examples	Mechanism of Action	Effect on Platelets
Non-Selective COX Inhibitors	Ibuprofen, Diclofenac, Naproxen	Reversibly inhibit COX-1 & COX-2 → ↓ Prostaglandins	Mild ↓ platelet aggregation
Irreversible COX Inhibitor	Aspirin	Irreversibly inhibits COX-1 → ↓ Thromboxane A2	Strong antiplatelet effect
Selective COX-2 Inhibitors (Coxibs)	Celecoxib, Etoricoxib	Selective inhibition of COX-2 → ↓ inflammatory prostaglandins	No significant effect
Central COX Inhibitor	Paracetamol	Inhibits COX in CNS (minimal peripheral action)	No effect

Pharmacokinetics (PK)

Steroids

- Absorption: Oral, IV, topical
- Distribution: Protein-bound (CBG, albumin)
- Metabolism: Liver
- Excretion: Urine

Non-Steroids

- NSAIDs: Oral absorption, renal excretion
- Opioids: First-pass metabolism
- Antibiotics: Variable routes (oral/IV/IM)

Pharmacodynamics (PD) & Mechanism

Steroids (GENOMIC ACTION)

- Bind cytoplasmic receptor → translocate to nucleus
- Bind GREs → regulate gene transcription
- ↓ Pro-inflammatory cytokines (IL-1, TNF-α)
- ↑ Anti-inflammatory proteins (lipocortin)

Pharmacodynamics (PD) & Mechanism

Non-Steroids (NON-GENOMIC ACTIONS)

NSAIDs

- Inhibit COX-1 / COX-2
- ↓ Prostaglandins → ↓ pain, inflammation

Opioids

- Bind μ-receptors → ↓ neurotransmitter release

Muscle Relaxants

- GABA-B activation → ↓ excitatory signals

Antibiotics

- Target bacteria:
  - Cell wall (penicillin)
  - Protein synthesis (macrolides)

Steroids vs Non-Steroids

Feature	Steroid Drugs	Non-Steroid Drugs
Structure	Cholesterol-derived	Diverse
Solubility	Lipid-soluble	Mostly water-soluble
Receptor	Intracellular	Membrane/enzyme
Mechanism	Gene transcription	Enzyme/receptor
Onset	Slow	Fast
Duration	Long	Short

Clinical Applications

Steroids

- Rheumatoid arthritis
- Asthma & COPD exacerbation
- Cerebral edema
- Autoimmune diseases
- Transplant rejection prevention

Non-Steroids

NSAIDs

- Pain, arthritis, sports injury

Opioids

- Cancer pain, trauma

Muscle Relaxants

- Stroke, spinal injury spasticity

Antibiotics

- Infection control in rehabilitation

Clinical Integration

When to choose steroids?

- Severe inflammation
- Autoimmune disease
- Need long-term immunosuppression

When to choose non-steroids?

- Acute pain
- Infection
- Mild–moderate inflammation

References

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