



SNS COLLEGE OF ALLIED HEALTH SCIENCE
SNS Kalvi Nagar, Coimbatore - 35
Affiliated to Dr MGR Medical University, Chennai



DEPARTMENT OF OPERATION THEATRE AND ANAESTHESIA TECHNOLOGY - II YEAR

COURSE NAME: PHARMACOLOGY

TOPIC - NSAIDS

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Lecturer



- NSAIDs are medications that reduce pain, inflammation, and fever without steroid-related risks.
- Act as analgesics, anti-inflammatories, and antipyretics.
- Used for arthritis, pain, and fever; not opioids or corticosteroids.



CLASSIFICATION



- Salicylates (e.g., Aspirin)
- Propionic Acids (e.g., Ibuprofen, Naproxen)
- Acetic Acids (e.g., Diclofenac, Indomethacin)
- Enolic Acids (Oxicams; e.g., Piroxicam, Meloxicam)
- Anthranilic Acids (Fenamates; e.g., Mefenamic Acid)
- Selective COX-2 Inhibitors (e.g., Celecoxib)



SALICYLATES (E.g., ASPIRIN)



- Mechanism:** Irreversible COX-1/COX-2 inhibition; reduces prostaglandins/thromboxanes.

PharmacoKinetics:

- Metabolism: Hepatic
- Excretion: Renal

PharmacoDynamics:

Anti-inflammatory, analgesic, antipyretic; antiplatelet via thromboxane block.

- Onset/Duration:** 30 min (pain); 4-6h duration; antiplatelet lasts 7-10 days.
- Dosage:** 325-650mg q4-6h (pain; max 4g/day); 75-325mg daily (CV prevention).
- Adverse Effects:** GI ulcers/bleeding, tinnitus, Reye's syndrome, renal impairment.
- Indications:** Pain, fever, inflammation, CV prophylaxis, arthritis.
- Interactions:** Bleeding risk with warfarin/SSRIs; reduces antihypertensives; alcohol increases GI risk.



Propionic Acids (e.g., Ibuprofen, Naproxen)



- **Mechanism:** Non-selective COX-1/COX-2 inhibition; reversible.

PharmacoKinetics:

- **Metabolism:** Hepatic
- **Excretion:** Renal (metabolites).

PharmacoDynamics:

Reduces prostaglandins for pain/inflammation relief.

- **Onset/Duration:** 30 min (pain); 4-8h (ibuprofen short; naproxen >10h long).
- **Dosage:** Ibuprofen: 200-800mg q6-8h (max 3.2g/day); Naproxen: 220-500mg q8-12h (max 1.5g short-term).
- **Adverse Effects:**
 - GI irritation/bleeds, CV risks (less with naproxen), renal toxicity, hearing loss.
- **Indications:** Mild-moderate pain, arthritis, dysmenorrhea, fever.
- **Interactions:** Diuretic/lithium reduction; bleeding with warfarin; hypertension aggravation.



ACETIC ACIDS (E.G., DICLOFENAC, INDOMETHACIN)



• **Mechanism:** Non-selective COX inhibition; potent anti-inflammatory.

PharmacoKinetics :

- Metabolism: Hepatic
- Excretion: Renal/biliary.

PharmacoDynamics :

- Prostaglandin reduction; strong on inflammation.
- **Onset/Duration:** 30 min (pain); 4-6h.
- **Dosage:** Diclofenac: 50-100mg tablets; Indomethacin: 25-50mg q8-12h (max 200mg/day).
- **Adverse Effects:** High GI risk (ulcers), hepatotoxicity (diclofenac), renal failure, CV events.
- **Indications:** Arthritis, gout, postoperative pain, migraine.
- **Interactions:** Methotrexate toxicity; bleeding with anticoagulants; diuretic antagonism.



ENOLIC ACIDS (OXICAMS; E.G., PIROXICAM, MELOXICAM)



- **Mechanism:** Non-selective COX inhibition (meloxicam prefers COX-2).

PharmacoKinetics :

Metabolism: Hepatic

Excretion: Renal.

PharmacoDynamics :

- Long-acting anti-inflammatory via prostaglandin block.
- **Onset/Duration:** Slower onset; >10h (long half-life).
- **Dosage:** Piroxicam: 20mg daily; Meloxicam: 7.5-15mg daily.
- **Adverse Effects:** GI bleeds, renal issues, skin reactions; lower GI risk with meloxicam.
- **Indications:** Chronic arthritis, long-term pain management.
- **Interactions:** Warfarin bleeding; lithium retention; alcohol synergy on GI.



ANTHRANILIC ACIDS (FENAMATES; E.G., MEFENAMIC ACID)



- **Mechanism:** Non-selective COX inhibition.

PharmacoKinetics :

- Metabolism: Hepatic;
- Excretion: Renal.

PharmacoDynamics :

- Analgesic/anti-inflammatory via prostaglandin reduction.
- **Onset/Duration:** 30 min; 4-6h.
- **Dosage:** 500mg initial, then 250mg q6h (max 7 days).
- **Adverse Effects:** GI upset, diarrhea, renal toxicity, hematologic issues.
- **Indications:** Dysmenorrhea, mild pain.
- **Interactions:** Anticoagulants (bleeding); antihypertensives reduction.



ELECTIVE COX-2 INHIBITORS (E.G., CELECOXIB)



- Mechanism:** Selective COX-2 inhibition; spares COX-1 (less GI impact).
- PK:** Absorption: GI (2-3h peak); Distribution: Protein-bound; Metabolism: Hepatic CYP2C9; Excretion: Fecal/renal.
- PD:** Anti-inflammatory without platelet/GI effects.
- Onset/Duration:** 1-2h; 12h.
- Dosage:** 100-200mg q12-24h (max 400mg/day).
- Adverse Effects:** CV risks (MI/stroke), renal issues; lower GI bleeds.
- Indications:** Arthritis, pain in patients with GI risk.
- Interactions:** Sulfonamide allergy; warfarin; fluconazole (CYP inhibition)

NSAID Class	Mechanism	Onset	Duration	Side Effects	Indications
Salicylates (Aspirin)	Irreversible COX-1/COX-2 inhibition	30 min	4-6h	GI bleeding, tinnitus, renal issues, Reye's syndrome	Pain, fever, arthritis, CV prophylaxis
Propionic Acids (Ibuprofen)	Non-selective COX inhibition	30 min	4-8h	GI upset, CV risk, renal dysfunction	Pain, arthritis, dysmenorrhea, fever
Acetic Acids (Diclofenac)	Non-selective COX inhibition	30 min	6-8h	GI bleeding, hepatotoxicity, CV risk	Arthritis, gout, migraines
Enolic Acids (Piroxicam)	Non-selective COX inhibition	30 min	12-24h	GI irritation, renal effects, CV risk	Chronic arthritis, pain
Fenamates (Mefenamic Acid)	Non-selective COX inhibition	30 min	6-8h	GI distress, diarrhea, renal issues	Dysmenorrhea, mild pain
COX-2 Selective (Celecoxib)	Selective COX-2 inhibition	30 min	12-24h	CV risk, renal effects, lower GI risk	Arthritis, pain in GI risk patients



THANK YOU