# Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

## Mechanism of Action

Nonsteroidal anti-inflammatory drugs (NSAIDs) exert their therapeutic effects through the inhibition of prostaglandin production. Prostaglandins are lipid mediators derived from arachidonic acid through the cyclooxygenase pathway. Cyclooxygenase enzymatically converts arachidonic acid into prostaglandins. NSAIDs interrupt this pathway through the acetylation of cyclooxygenase[1]. Two forms of cyclooxygenase exist; COX-1 and COX-2. Inhibition of COX-2 is responsible for the adverse effects of nonselective NSAIDs.

| Cyclooxygenase Isoforms | |
|---|---|
| COX-1 | COX-2 |
| Constitutive Isoform | Inducible Isoform |
| Platelet aggregation | Fever |
| Renal function | Inflammation |
| Gastric protection | Pain |

## Pharmacokinetics

- Highly protein bound
- Low volume of distribution (low tissue binding)
- Weakly acidic (except COX-2 inhibitors)
- Elimination by hepatic oxidation or conjugation

## Common Medications

NSAIDs can be categorized into six chemical classes:

| Classification | Medication | Inhibition |
|---|---|---|
| Salicylic acid derivatives | aspirin | COX-1, COX-2 |
| Propionic acid derivatives | Ibuprofen, ketoprofen, naproxen, oxaprozin | COX-1, COX-2 |
| Acetic acid derivatives | diclofenac, etodolac, ketorolac, indomethacin, nabumetone, sulindac | COX-1, COX-2 |
| Para-aminophenol derivatives | acetaminophen | Weak COX-1, COX-2 |
| Enolic acid derivatives | meloxicam | COX-2 > COX-1 |

 

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| Selective COX-2 Inhibitors | celecoxib, etoricoxib | COX-2 |

References

1. Deer, T., Leong, M. and Buvanendran, A. (2013). *Comprehensive treatment of chronic pain by medical, interventional, and integrative approaches*. New York, NY: Springer.

 