

# NSAID Pharmacology Cheat Sheet

by happyfeet2020 via cheatography.com/144934/cs/31540/

#### How Do NSAIDs Work?

NSAIDs have anti-inflammatory, antipyretic, analgesic and anti-platelet properties.

Goal is to inhibit the cox-mediated generation of pro-inflammatory eicosanoids and to limit extent of inflammation, pain and fever.

They do this by blocking the site in cyclooxygenase enzyme in which substrate arachidonic acid binds to

Most NSAIDs are metabolized in the liver by oxidation and conjugation to inactive metabolites which are typically excreted in urine \*patient sensitive to one NSAID may be sensitive to any other NSAID. Studies show that meloxicam can be a good option for NSAID intolerant patients

#### **COX-1 Function**

Contributes to homeostasis

Ongoing constitutive physiologic "house keeping", vascular homeostasis, maintenance of renal, myocardial and GI blood flow, platelet function, intestinal mucosal proliferation, antithrombogenesis

### **COX-1 Inhibition**

Decreases mucosal defense

Increase GI acid, and decreases GI mucus

Decreases HCO2 secretion

Decreases mucosal blood flow

### Special Consideration for NSAIDs

- 1. Both ibuprofen and naproxen may reduce the effects of furosemide (diuretic) and may reduct the effectiveness of several antihypertensive agents
- 2. Indomethacin is the NSAID most likely to cause nephrotoxicity

### COX 2 Inhibitors

Due to sometimes severe GI adverse effects associated with long-term NSAID therapy, selective cox 2 inhibitors are used Inhibition of the chemical mediators responsible for inflammation while maintaining the cytoprotective effects of the products of COX-1 activity

#### Meloxicam

Inhibition of COX-2 may generate some problems in wound healing, angiogenesis and the resolution of inflammation

Lower GI and renal problems

Cox 2 may induce hypertension, renal failure and cardiac failure

Naproxen has some cardioprotective properties

### Contraindication for NSAIDs (cont)

Compro
NSAIDs reduce renal blood flow
mised and therefore may further

Renal reduce renal function which may
have an impact on the effects of
concurrent meds and elimination of the NSAID and other
meds and toxins

Compro Most NSAIDs are metabolised mised in liver

Liver

**Function** 

**Hypers-** Happens more in asthmatics **ensitivity** 

### **COX 2 Function**

Works at the site of pain and inflammation Source of prostacyclin- platelet stability/dilate blood vessels

### **COX 2 Inhibition**

Decrease pain and inflammation

Increases CV risk as it shifts the balance between platelet production TxA2 and PGI2, predisposing to platelet aggregation, thrombus formation and vasoconstriction

### Salicylates

#### Salicylates (cont)

A single administration of aspirin decreases for several days the amount of thromboxane that can be generated, shifting the vascular TxA2-PGI2 balance toward PGI2 mediated vasodilation, platelet inhibition, and antithrombogenesis

Long term use of aspirin can lead to GI ulceration and hemorrhage, nephrotoxicity and hepatic injury

Two unique toxicities of aspirin: induced airway hyperactivity in asthmatics and reye's syndrome

### **NSAID Drug to Drug Interaction**

### Contraindication for NSAIDs

Reye's

For patients under 18

Syndone

years old

Pregnancy

Includes Aspirin which acts in an irreversible manner by acetylating the active site serine residue in both COX-1 and COX-2

Daily low dose aspirin is used as an anti-thrombogenic agent for prophylaxis and post event management of MI and stroke

Aspirin is antithrombogenic because of its irreversible inhibition of COX, which prevents platelets from biosynthesizing TxA2

Within an hour of aspirin, the effects of COX-1 activity on newly formed platelets is irreversibly destroyed (acetylated) therefore TXA2 cannot be produced

Wendy's LAMP	mnemonic for remembering drug interactions
Warfarin	May increase risk of bleeding- Monitor PT and INR
Lithium	May increase lithium plasma levels and decrease its clearance renally- need to monitor
ACE Inhibitors	may decrease antihyperten- siuve effects so need to monitor BP and CV function
	NSAIDs increase BP and decrease affects of diuretics, ACE inhibitors and ARB which all relax blood vessels as NSAIDs inhibit cox-2 in kidneys which decreases sodium excretion due to a decrease in prostaglandins
	NSAIDs may increase fluid retention and decrease blood flow to the kidneys as they block prostaglandins which dilate blood vessels and allow O2 to reach kidneys
Methot- rexate	May lead to an increase in methotrexate toxicity- don't administer within 10 days of high dose methotrexate
Probenecid	May lead to reversal or

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uricosuric effects



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### Non-Selective COX 1 and COX 2 Inhibition

GI irritation due to decreased protection of gastric mucosa.- N&V, GI ulcer, diarrhea

Skin reactions - mild rash, hives, photosensitivity

Inhibition of platelet function- increase risk of bleeding

Decreased renal blood flow- decreases GFR can cause renal ischemia- look out for pts w/ renal disease

CVD risk

Respiratory- bronchospasm- look out for asthmatic pts

### **Risk Factors for GI complications**

Over 60 in age

history of peptic ulcer

use of anti coagulants or corticosteroids

History of pylori infection

High NSAID dose or use of two NSAIDs

Severe illness

### Reducing GI risks

Misoprostol

Synthetic prostaglandin-Protects gastric mucosa from

irritation

**Protein** 

Long lasting reduction of gastric

Pump

acid production

**Inhibitors** 

Drugs end in -"prazole"

Receptor Antago-

H2

Blocks the action of histamine on parietal cells in the stomach decreasing the production of

nists acid by these cells

Drugs end in -"dine"

Not enough evidence that these alone will work in reducing GI

issues

COX-2

Allow continued protective

Inhibitor

COX-1 function

### Acetaminophen

Not an NSAID

Has analgesic and antipyretic effects similar to aspirin

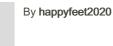
anti-inflammatory effects are insignificant because of its weak inhibition of COX

May be a third functional COX isoform (COX-3)

MOA believed to involve the prostaglandin pathways within the CNS with little influence on peripheral prostaglandin synthesis

Adverse effect= hepatotoxicity as it is metabolized by hepatic cytochrome p450 enyzmes which produces a reactive molecule which is normally detoxified by conjugation with glutathione. An overdose of acetaminophen can overwhelm glutathione stores, leading to cellular and oxidative damage and in severe cases to acute hepatic necrosis

4000mg daily limit for adults. For those who are alcoholics or multiple medication patients and patients with liver disorders, even doses within the therapeutic range may be hepatotoxic



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