"Get rid of all the medical journals in the waiting room. They give my patients too many ideas."

Common OTC & Rx Drugs

OTC Confusion


Active Ingredient


TOFU CHEWS
COMPARE VALUE

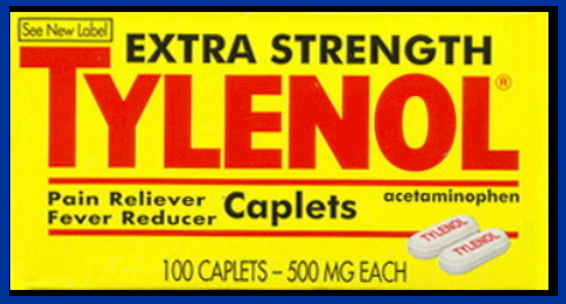
Analgesics

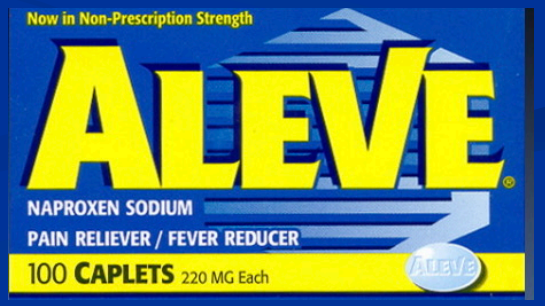
- Analgesics are drugs which relieve pain without loss of consciousness.

Analgesics / Antipyretics

- Some analgesics are also antipyretics → reduce fever.

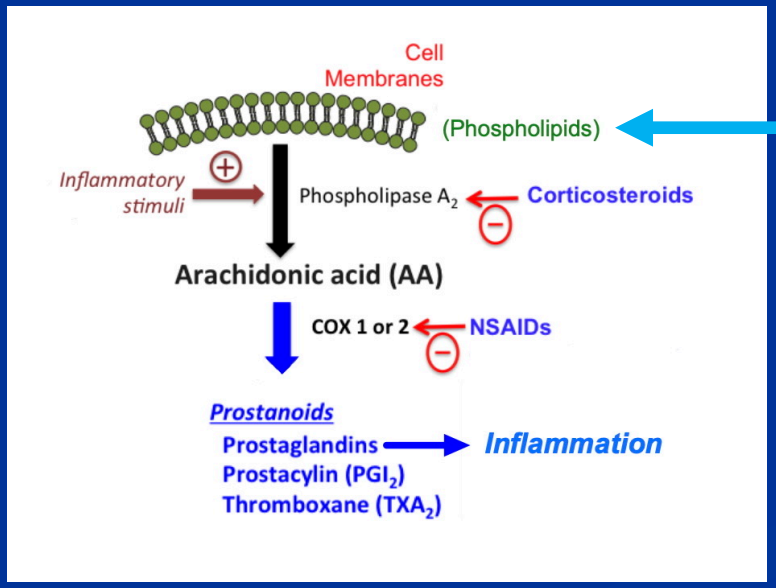
(1) Acetaminophen (Tylenol, APAP)
(2) Aspirin (ASA)
(3) NSAIDs
  - Ibuprofen (Motrin, Advil)
  - Naproxen (Aleve, Naprosyn)

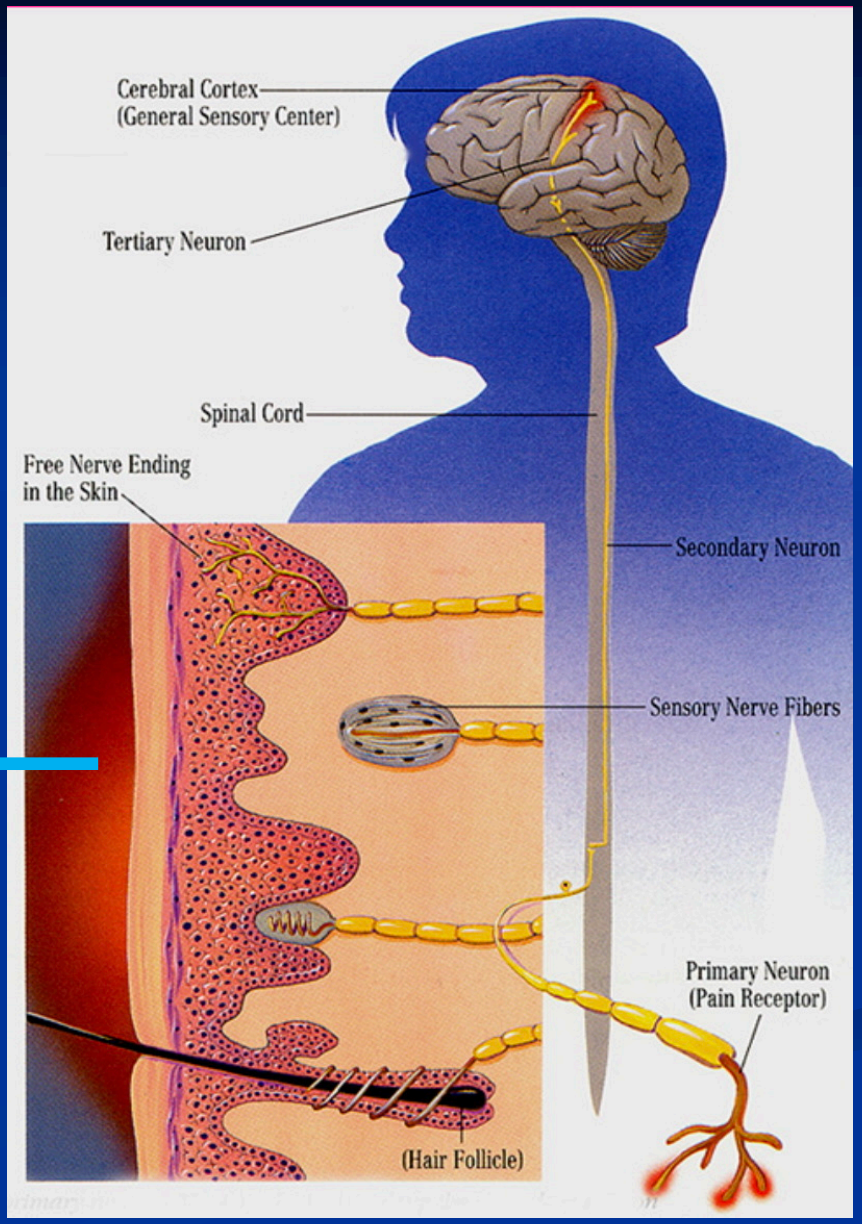




NSAIDs

- Mechanism of Action (MOA)
  → inhibit PG production
  → reduce inflammation
  → reduce pain





NSAIDs

↓

inhibit COX

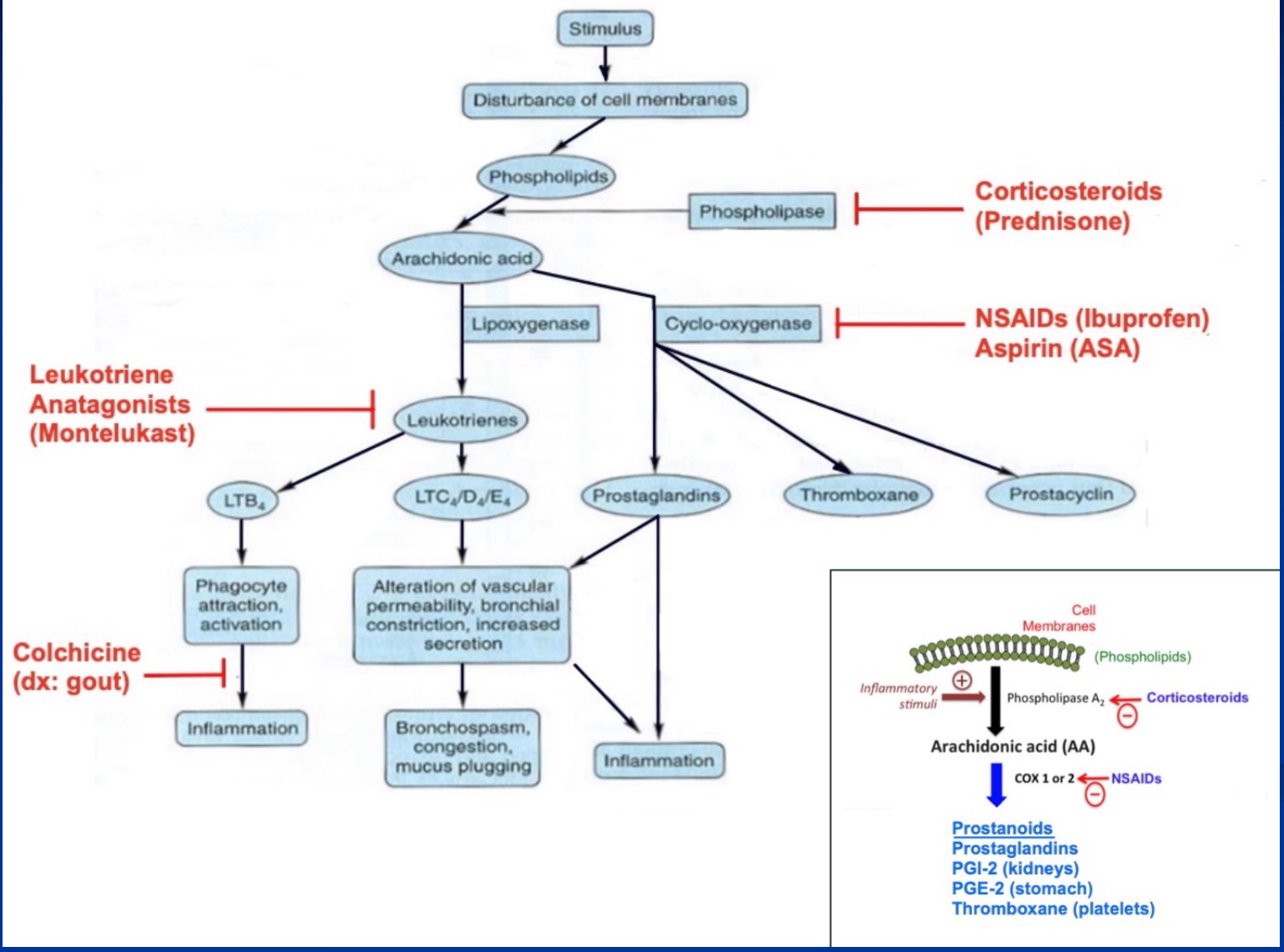
↓

inhibit PG production

↓

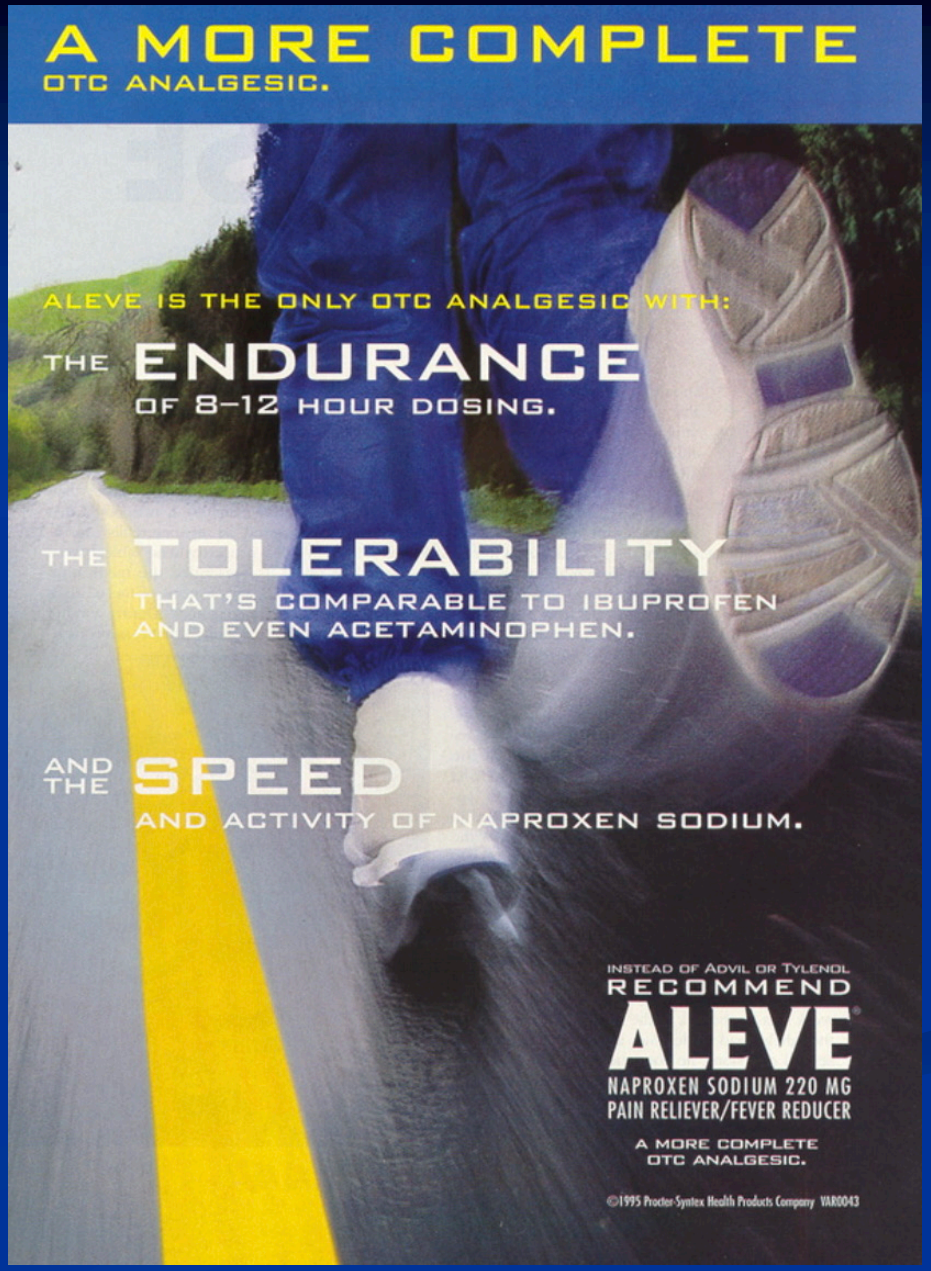
reduce inflammation and pain

Cyclooxygenase (COX) Pathway



Naproxen (Aleve) versus Ibuprofen (Advil)

- Naproxen offers the convenience of twice daily (BID) dosing.



NSAIDs & Kidney Function

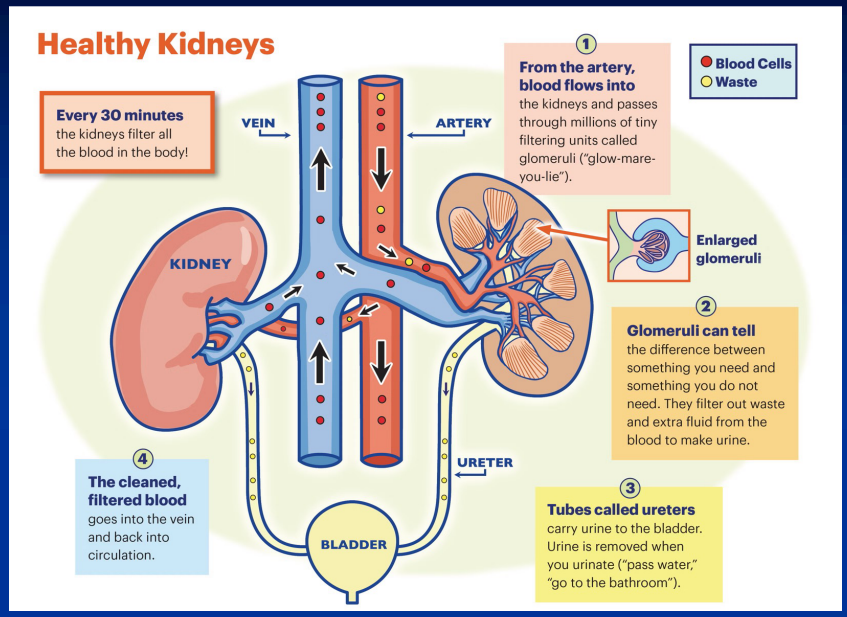
NSAIDs inhibit renal prostaglandin (PGI_2)

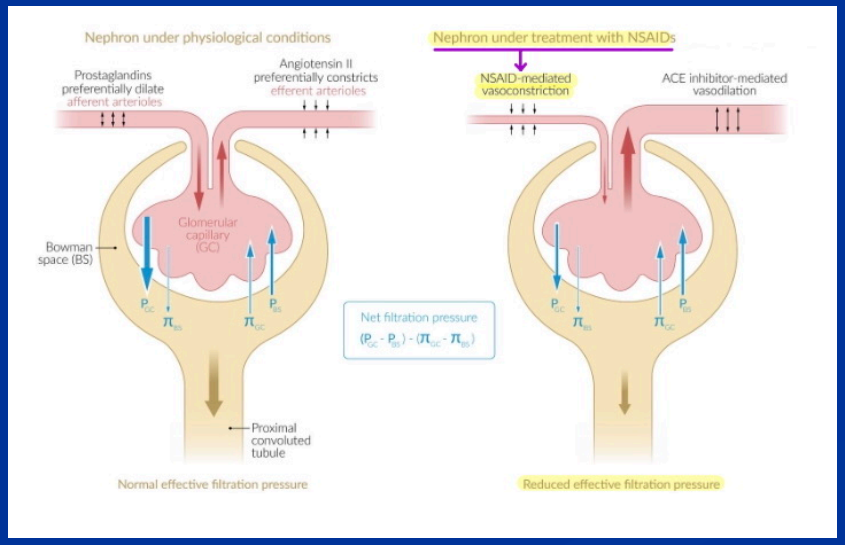
→ reduce kidney blood flow by constricting afferent arteriole of the nephron

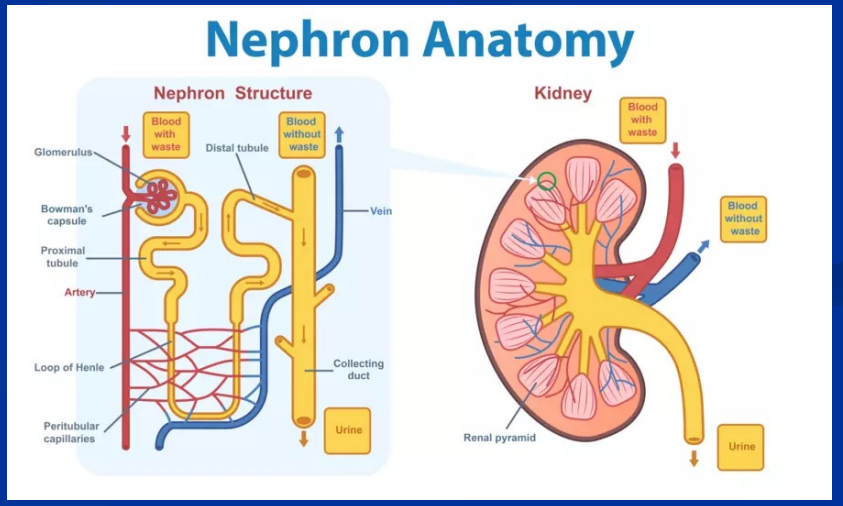
→ reduce glomerular filtration (GFR)

→ increase sodium and water retention, especially in geriatric patients

→ exacerbate hypertension (HTN), heart failure, and chronic kidney disease (CKD)





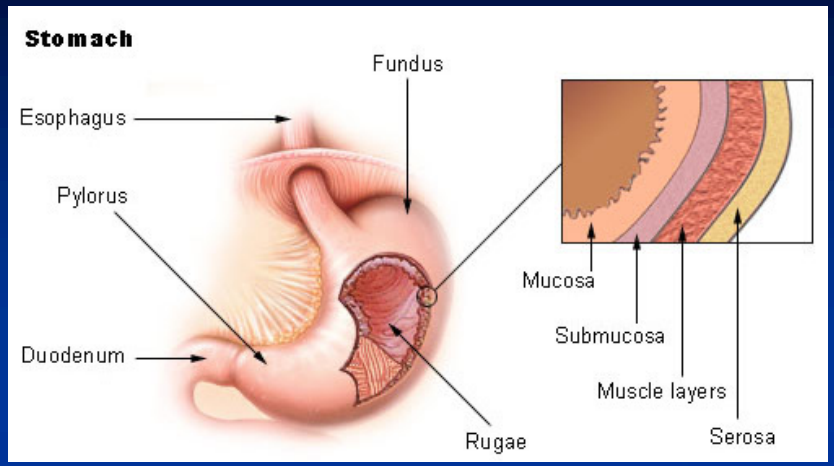


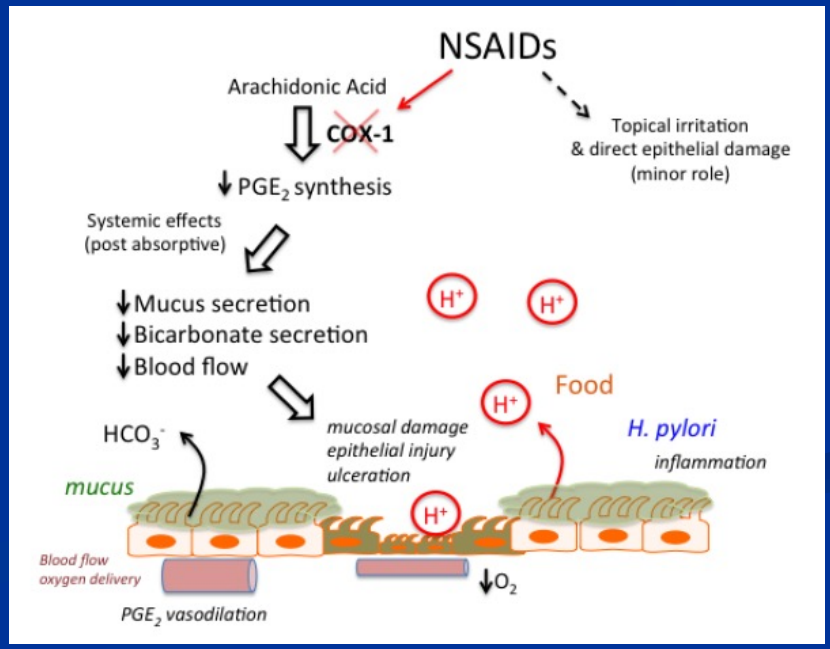
NSAIDs & GI Adverse Effects

NSAIDs inhibit PGE_2 (stomach)

→ decrease secretion of mucus and bicarbonate protective layers → thinning of mucoprotective lining of the stomach

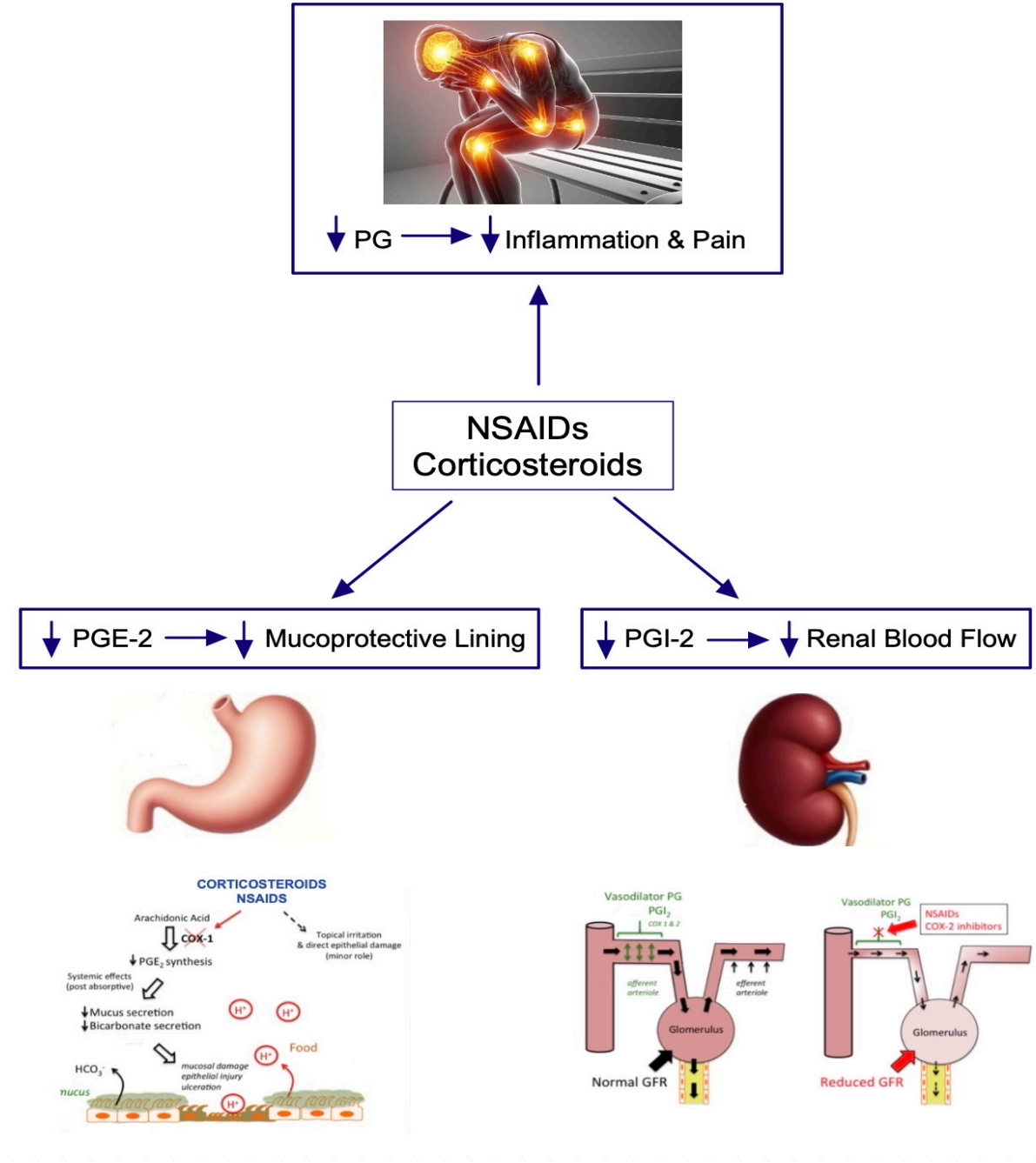
→ increase risk of gastric ulcers and GI bleeding





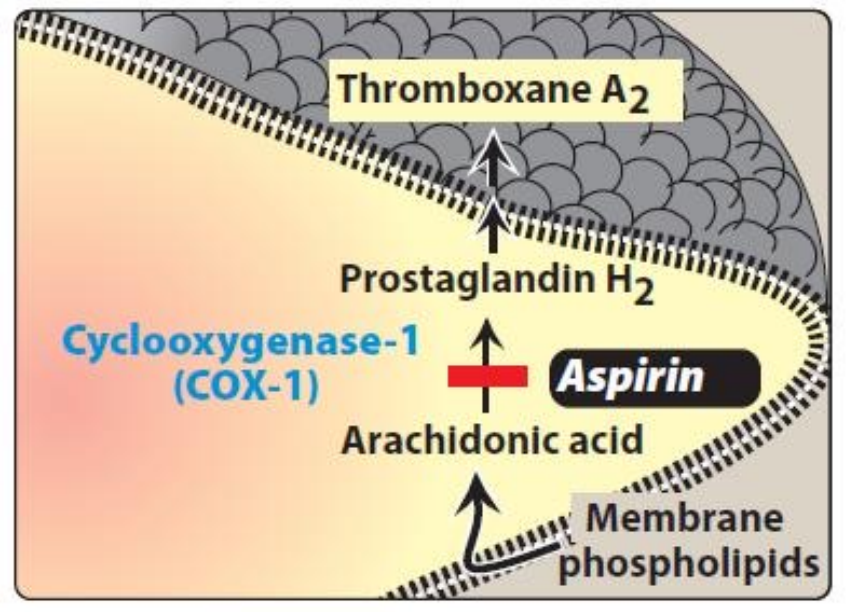
Summary of NSAID-Induced Adverse Effects in the GI Tract and Kidneys.

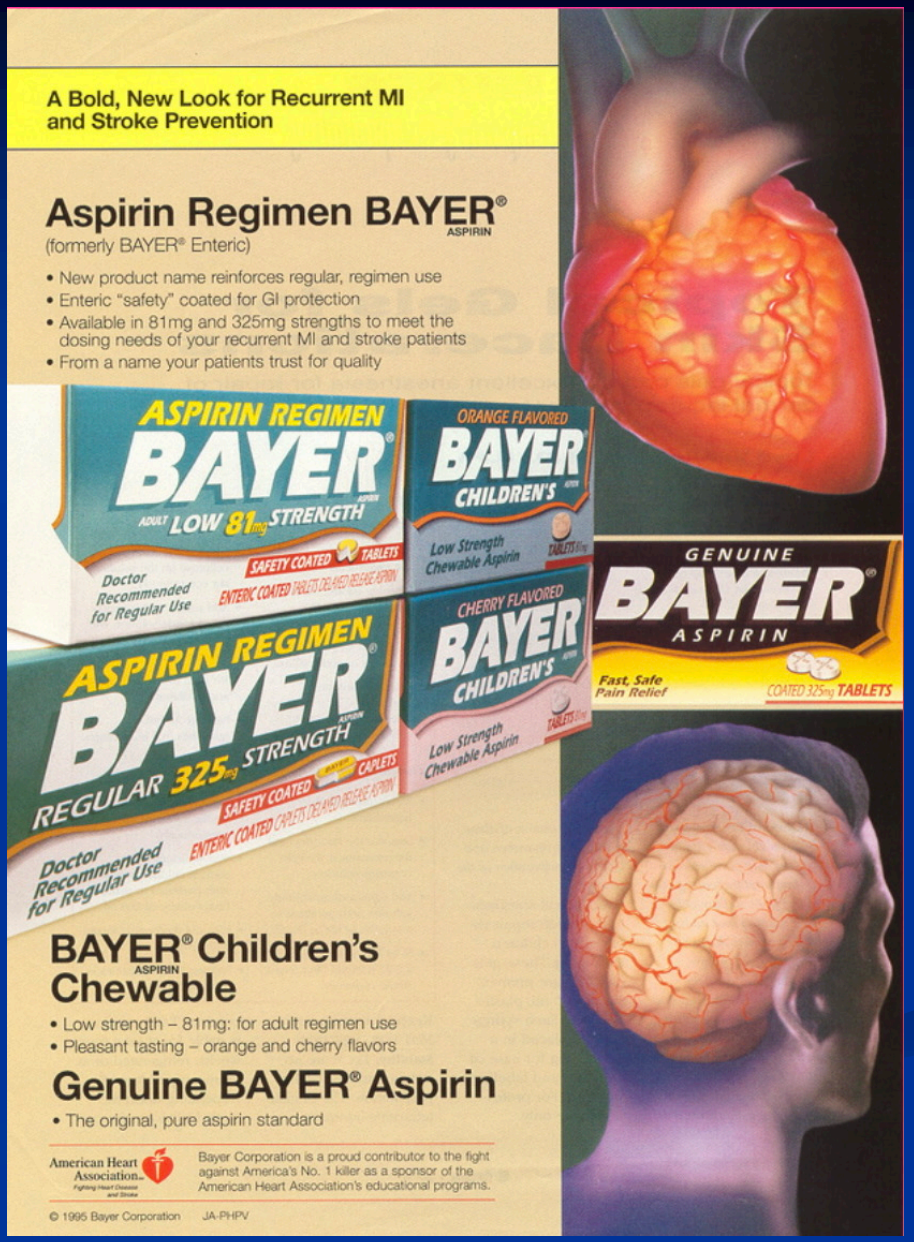
- NSAIDs & Corticosteroids inhibit PGE_2 → decrease muco-protective lining of the stomach → increase risk of gastritis and peptic ulcer disease (PUD).
- NSAIDs & Corticosteroids inhibit PGI_2 (prostacyclin) in kidneys → decrease renal blood flow → decrease GFR → increase Na^+/H_2O retention → exacerbate hypertension (HTN), heart failure, and chronic kidney disease (CKD)



Aspirin (ASA)

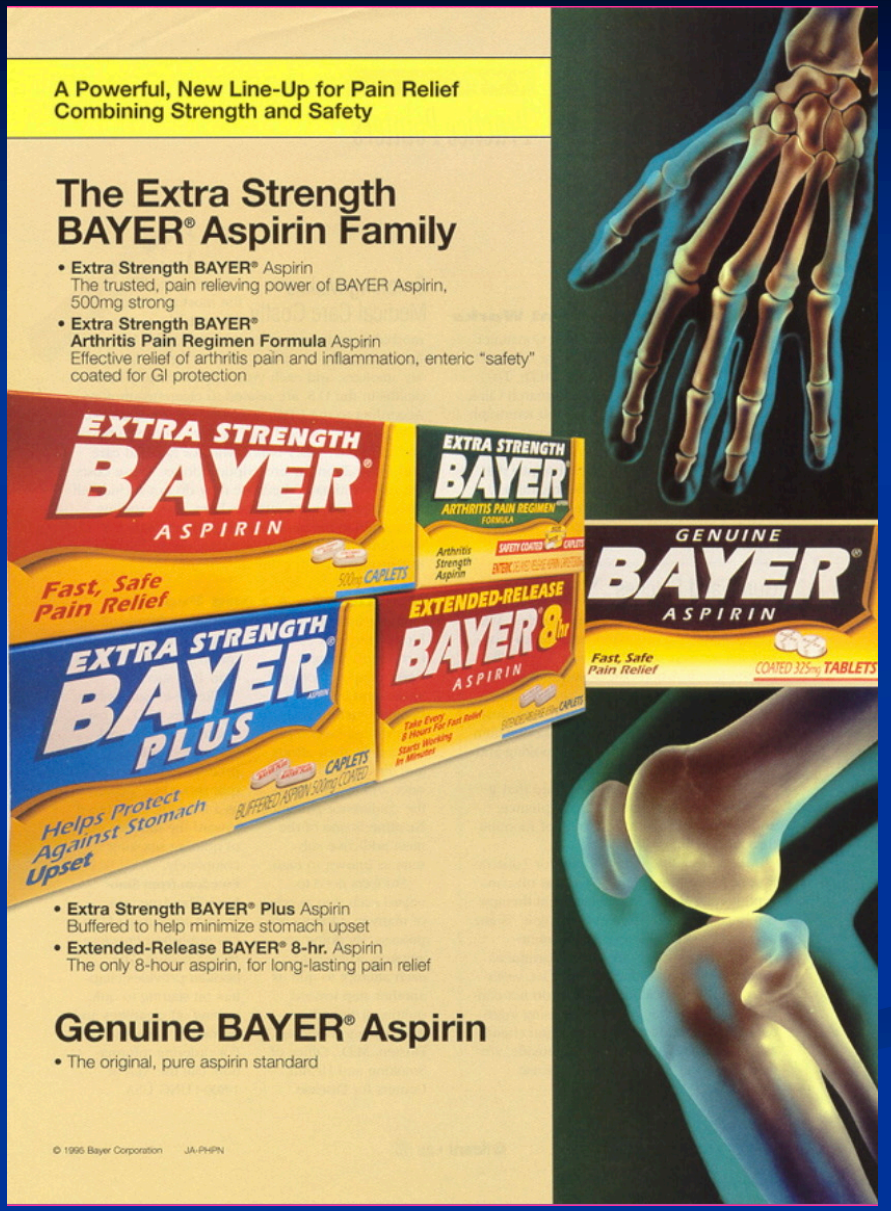
- ASA (81 mg/day) irreversibly inhibits COX-1 in platelets.
  - → inhibits platelet aggregation
  - → prevents thromboembolic events (i.e., MI and stroke)





Aspirin (ASA)

- The properties of aspirin are dose-dependent.
  - antiplatelet effect: 81-162 mg/day
  - analgesic / antipyretic effect: 325-650 mg / dose
  - anti-inflammatory effect: 1000 mg / dose



Buffered Aspirin Products: Bufferin and Ascription

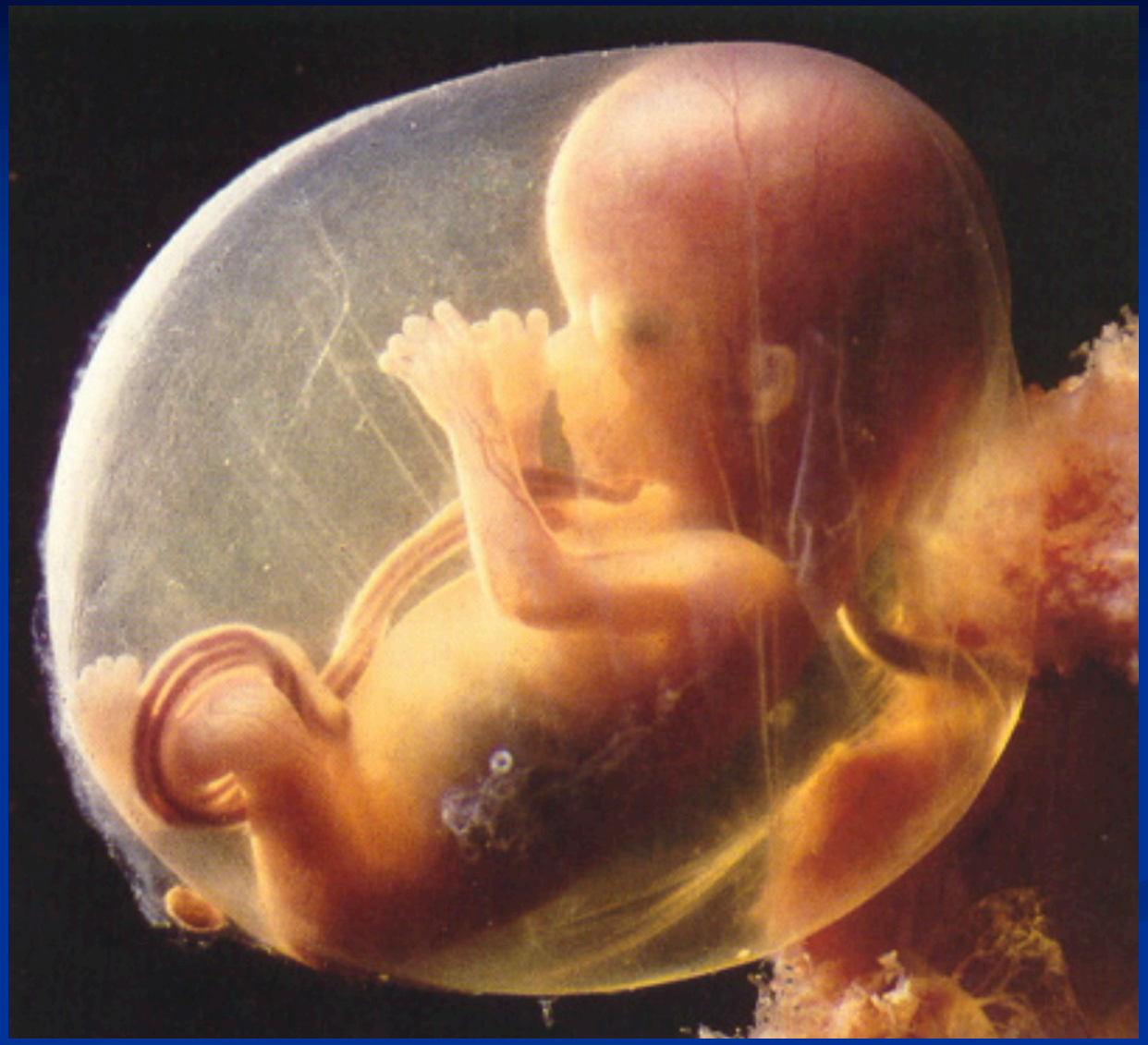
- buffered ASA products contain aspirin plus a dose of an antacid to prevent GI upset

Enteric-Coated Aspirin: Ecotrin

- Enteric-coated aspirin tablets dissolve and get absorbed in the alkaline environment of the small intestine.
- Enteric-coated aspirin reduces gastric irritation and ulcers, especially in patients requiring long-term aspirin therapy for cardiovascular prevention.

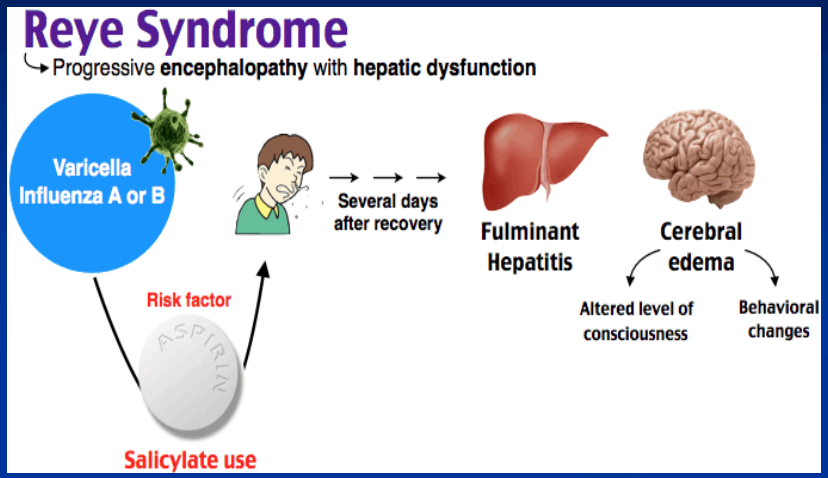
Aspirin & Pregnancy

Aspirin and NSAIDs increase bleed risk in both mother and fetus during pregnancy (due to antiplatelet effect), especially during the 3rd trimester.



Aspirin & Reye's Syndrome

Reye's Syndrome is a rare potentially fatal condition characterized by acute encephalopathy and liver dysfunction in children recovering from a viral illness (such as influenza or varicella / chickenpox), when treated with aspirin.

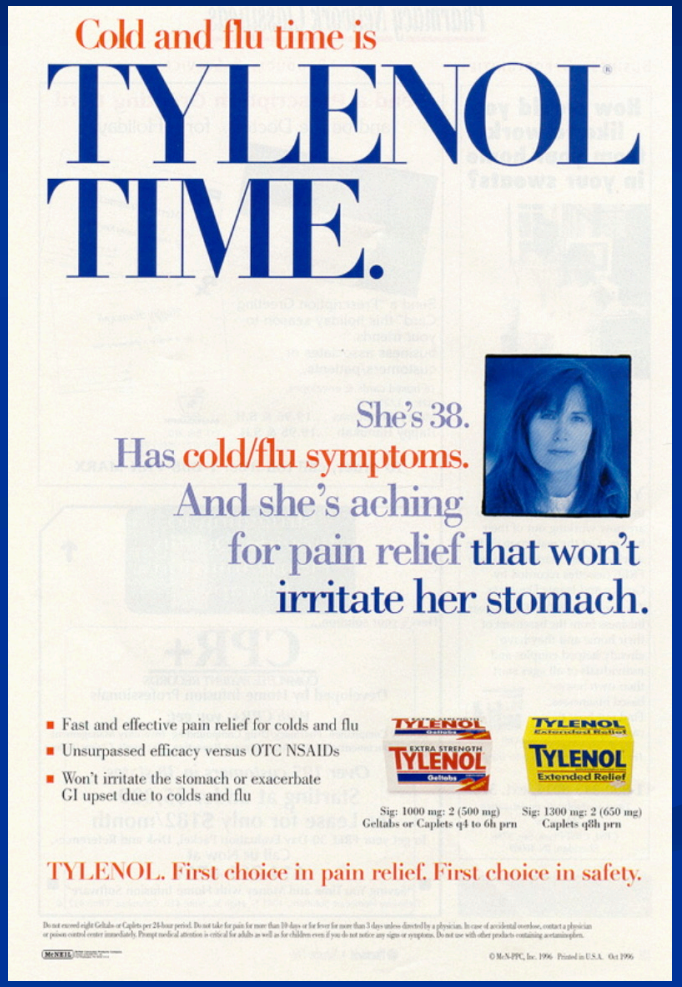


Pediatric patients should be treated with APAP (Tylenol) or ibuprofen (Motrin) for fever or cold & flu.

Acetaminophen (Tylenol, APAP)

- Advantage over NSAIDs and aspirin (ASA) → no GI upset
- Disadvantage: no peripheral anti-inflammatory properties in conditions such gout, musculoskeletal injuries, osteoarthritis, etc …

Property	Acetaminophen	NSAIDs (e.g., ibuprofen, aspirin)
Analgesic (pain relief)	✅ Yes	✅ Yes
Antipyretic (fever reduction)	✅ Yes	✅ Yes
Anti-inflammatory	❌ *Minimal to none*	✅ Strong
Mechanism	Weak COX inhibition in CNS	Strong COX-1/2 inhibition peripherally

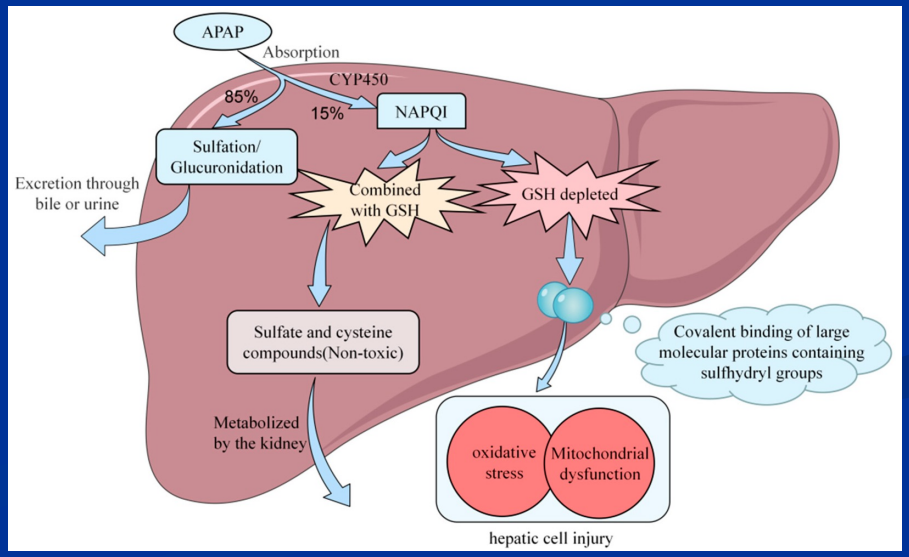


Acetaminophen Overdose & Hepatoxicity

- The liver breaks down most of the APAP using sulfation and glucuronidation to excrete APAP.
- 10-15% of APAP is metabolized by CYP2E1 into a toxic metabolite known as NAPQI, which is hepatotoxic.
- Glutathione is needed for the reactions that detoxify NAPQ1.
- N-acetylcysteine (NAC), a treatment of APAP overdose, is a precursor for glutathione.

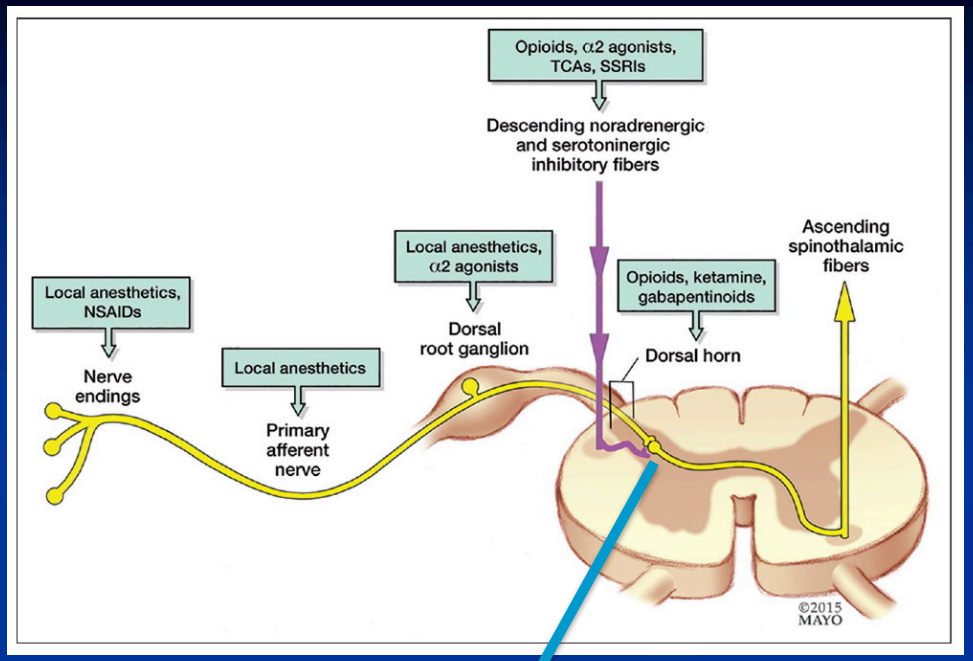
Stages of Acetaminophen Toxicity

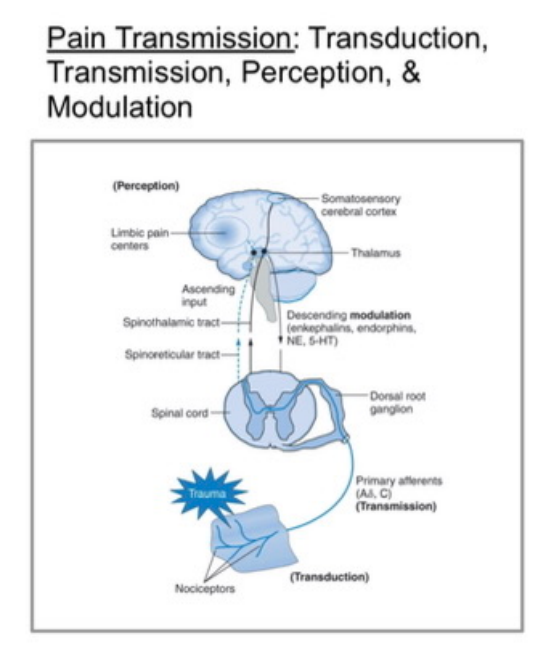
Stage	Signs & symptoms
Stage I (0.5 – 24 hours)	• **Nausea, vomiting, diaphoresis, pallor, lethargy, & malaise** • Some patients remain asymptomatic • **Laboratory studies are usually normal**
Stage II (24 – 72 hours) 	• Initially, subclinical elevations of AST, ALT develop • **patients develop right upper quadrant pain, with liver enlargement and tenderness. Elevations of prothrombin time (PT) and total bilirubin, oliguria, and renal function abnormalities may become evident**
Stage III (72 – 96 hours)	• Liver function abnormalities peak • **Symptoms of stage I reappear with jaundice, confusion (hepatic encephalopathy).** • ALT and AST levels > 10,000 IU/L • **Prolongation of the PT/INR, hypoglycemia, lactic acidosis, total bilirubin concentration above 4.0 mg/dL Death most commonly occurs in this stage**
Stage IV (4 days – 2 weeks) Recovery	• Patients who survive stage III enter a recovery phase • **Recovery can be slower in severely ill patients**



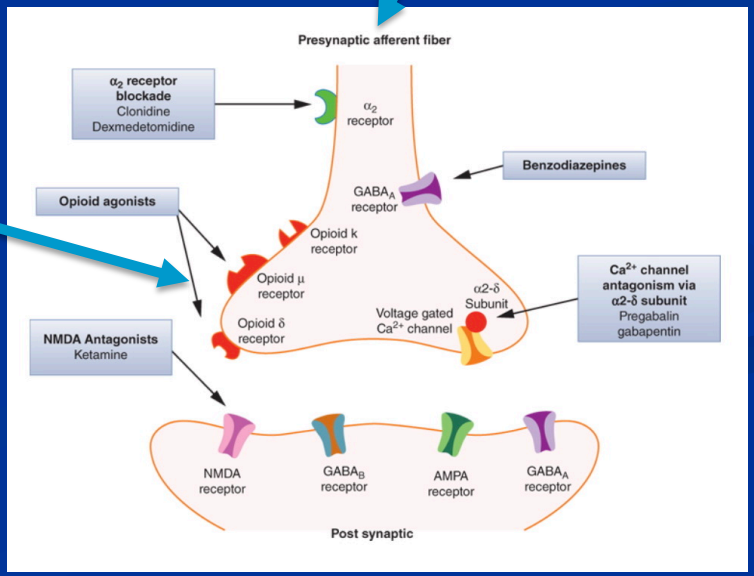
Narcotic Analgesics

- Opioids alleviate pain primarily by inhibiting neurotransmitter release at multiple levels of the pain pathway in the CNS, while collateral actions occur in the respiratory centers, GI tract, and reward circuitry to produce the adverse effects and abuse potential (i.e., euphoria).



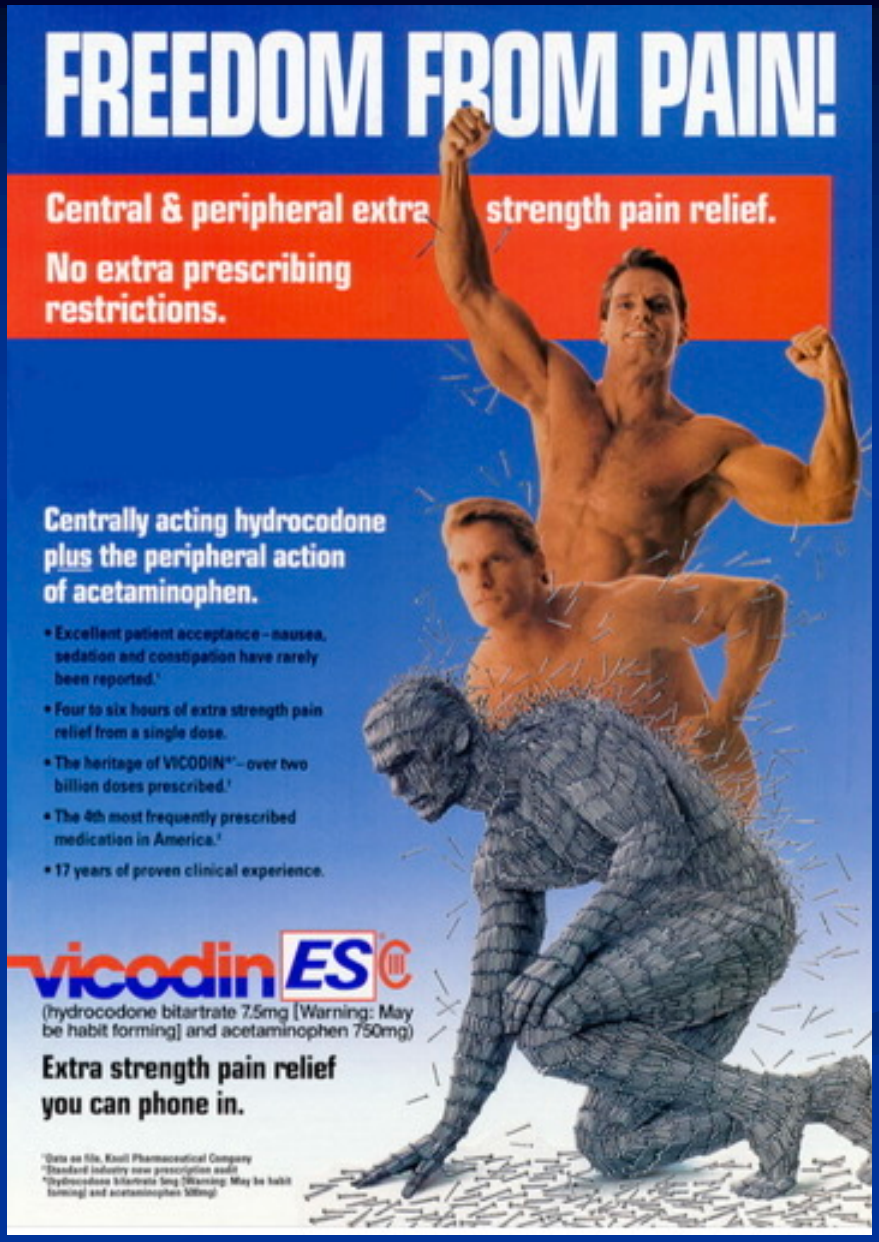


opioid receptors

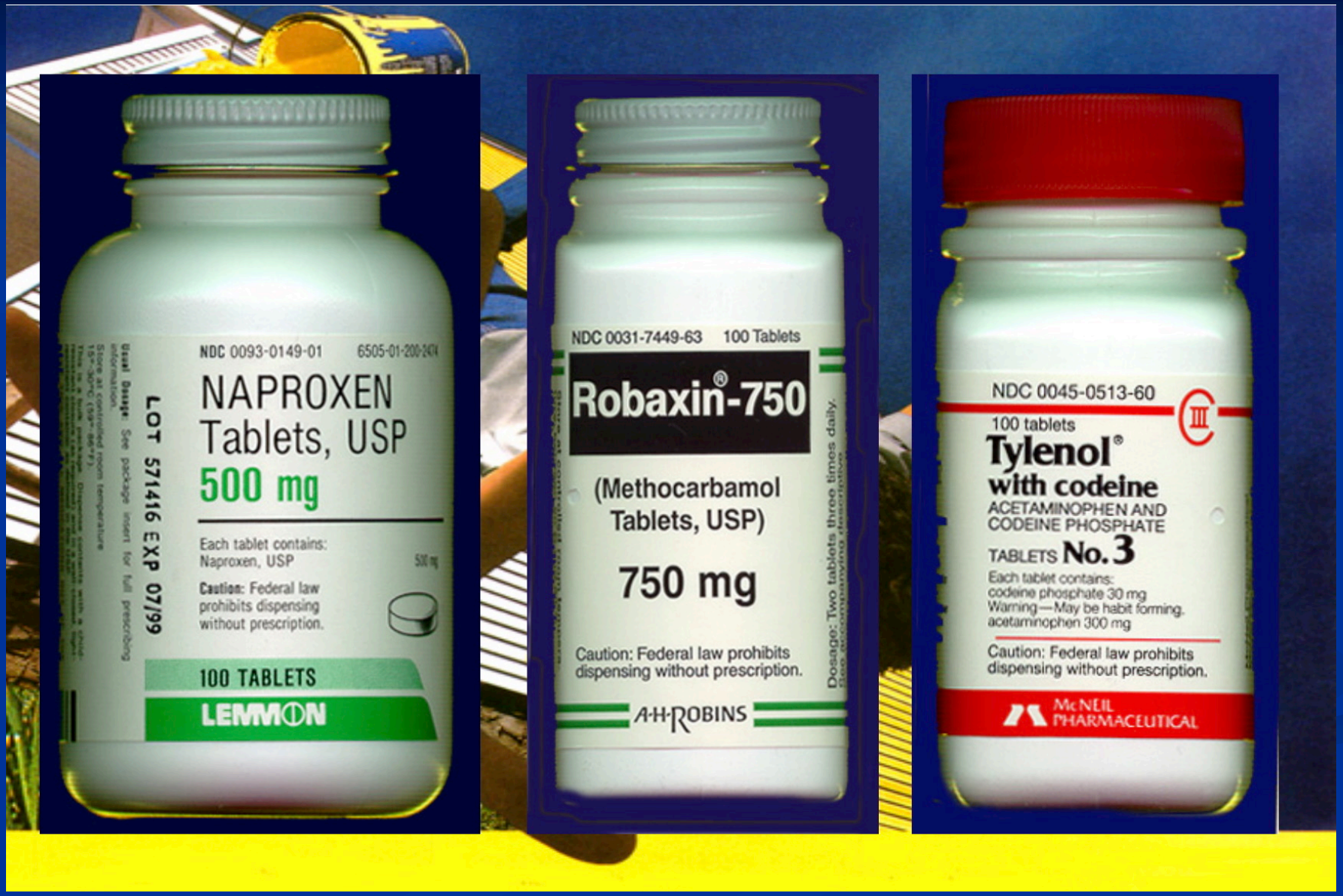


Narcotic Analgesics

- Narcotics are very potent analgesics; however, they do not offer antipyretic and anti-inflammatory properties.
- Risks: drug tolerance, drug dependence, and potential for opioid addiction.

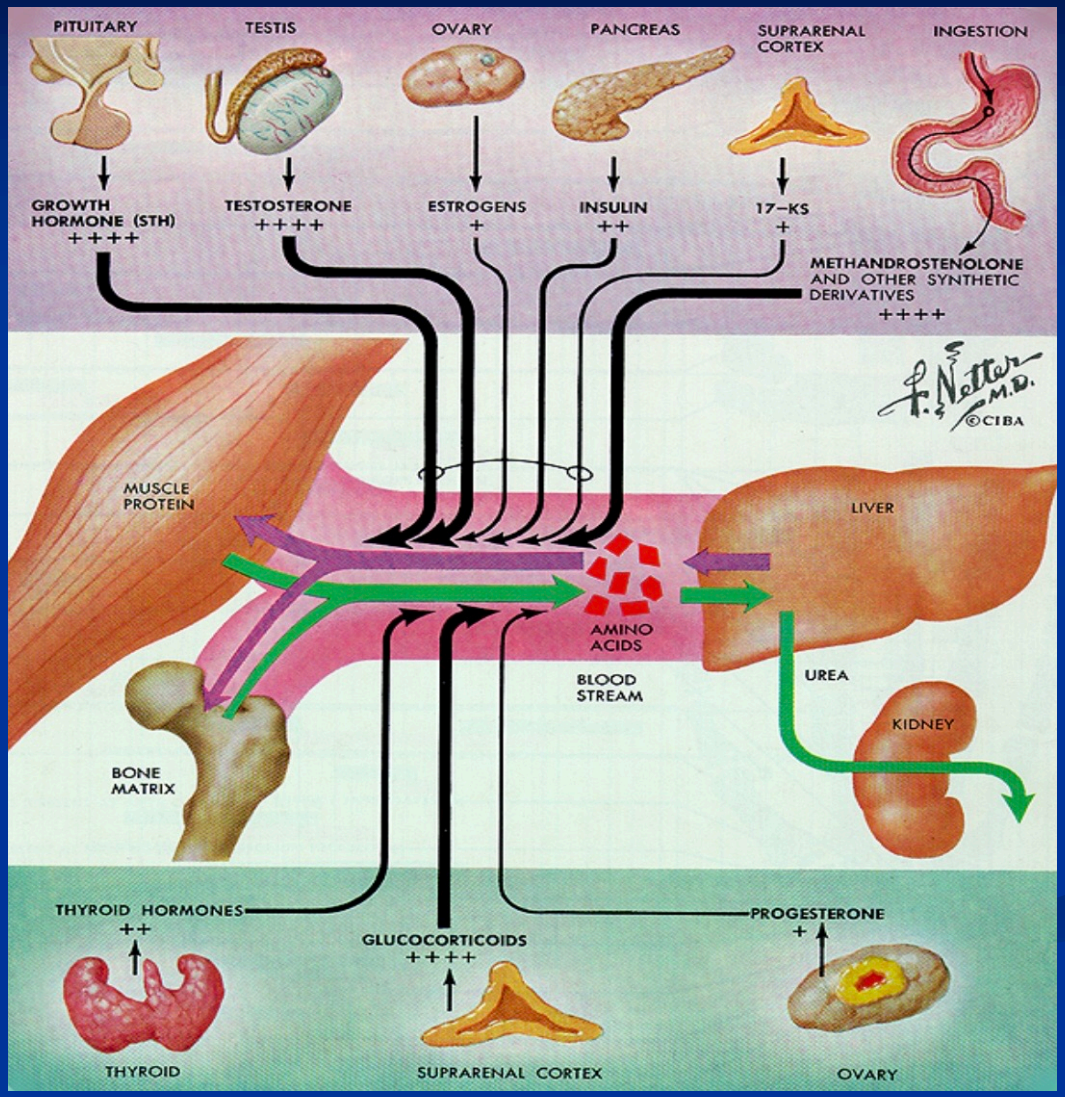


Treatment Options: NSAIDs, Muscle Relaxants, & Narcotic Analgesics

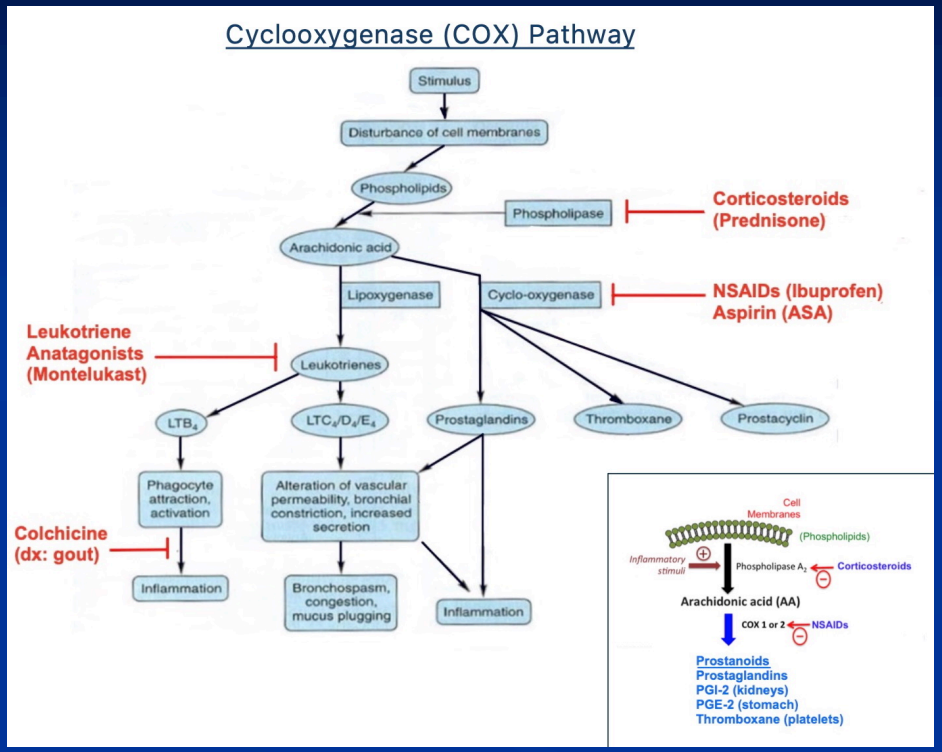


Corticosteroids

- Corticosteroids, also known as glucocorticoids, are potent anti-inflammatory agents.
- Disadvantages
  - PGE_2 inhibition → decreases muco-protective lining (stomach) → GI upset / ulceration.
  - PGI_2 (prostacyclin) inhibition in kidneys → decreases renal blood flow → decreases GFR → increases Na^+/H_2O retention → exacerbates HTN, heart failure, & CKD.
  - Systemic adverse effects (short-term and long-term): HPA-axis suppression, hypertension, hyperglycemia, cataract formation, immunosuppression, osteoporosis, myopathy, weight gain, etc…



Corticosteroids: Risks versus Benefits



Major Adverse Effects Associated with Systemic (PO/IV) Glucocorticoid Therapy

Metabolic & Endocrine Hyperglycemia Adrenal Insufficiency (i.e., HPA-Axis Suppression) Immune System Immunosuppression (risk of infection) Hematologic Leukocytosis Cardiovascular Fluid Retention Hypertension	Neuropsychiatric Dysphoria/Depression Mania/Psychosis Euphoria Insomnia Ophthalmologic Elevated Intraocular Pressure Cataract Formation Exophthalmos Gastrointestinal Gastritis Peptic Ulcer Disease (PUD)	Bone & Muscle Osteoporosis Myopathy Dermatologic & Appearance Cushingoid Appearance Facial Erythema Skin thinning Weight Gain Hirsutism Acne Striae

Summary Statements: ASA, Acetaminophen, NSAIDs, Glucocorticoids, and Opioids

Acetaminophen (Tylenol)

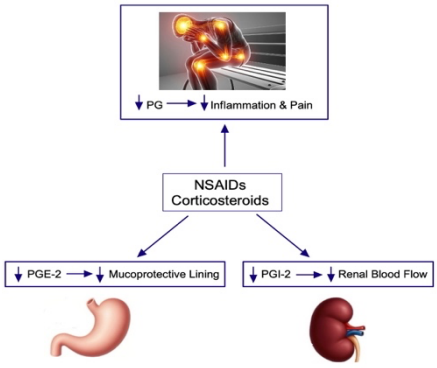
- Properties: antipyretic and analgesic
- Disadvantage: no anti-inflammatory properties
- Advantage: does not cause GI upset, gastritis, GI bleeding/ulcers
- Acetaminophen (APAP) does not exert antiplatelet effect
- APAP overdose --> hepatoxicity

Aspirin (ASA)

- Pharmacologic effects are dose-dependent
  - ASA 81 mg/day --> antiplatelet effect --> reduces risk of recurrent thromboembolic events in post-stroke / post-MI patients.
  - ASA 325-500 mg/dose --> analgesic effect (e.g., headache)
  - ASA 1000 mg/dose --> anti-inflammatory effect
- Disadvantages
  - PGE_2 inhibition (stomach) --> decreases muco-protective lining --> GI upset, gastritis, GI bleeding / ulcers
  - PGI_2 (prostacyclin) inhibition --> decreases renal blood flow --> decreases GFR --> increases sodium/water retention --> exacerbates HTN / CHF

NSAIDs: Ibuprofen (Motrin, Advil) and Naproxen (Naprosyn)

- Properties: antipyretic, analgesic, and anti-inflammatory.
- Disadvanages
  - PGE_2 inhibition (stomach) --> decreases muco-protective lining --> GI upset, gastritis, GI bleeding / ulcers
  - PGI_2 (prostacyclin) inhibition (kidneys) --> decreases renal blood flow --> decrease GFR --> increases sodium/water retention --> exacerbates HTN/CHF
- Naproxen (Aleve is OTC, Naprosyn is Rx) is a more potent NSAID than ibuprofen (Advil, Motrin)
- Naproxen (BID dosing) has a longer duration of action than ibuprofen (TID-QID dosing).



Glucocorticoids = Corticosteroids = Anti-Inflammatory Steroids (Example: Prednisone)

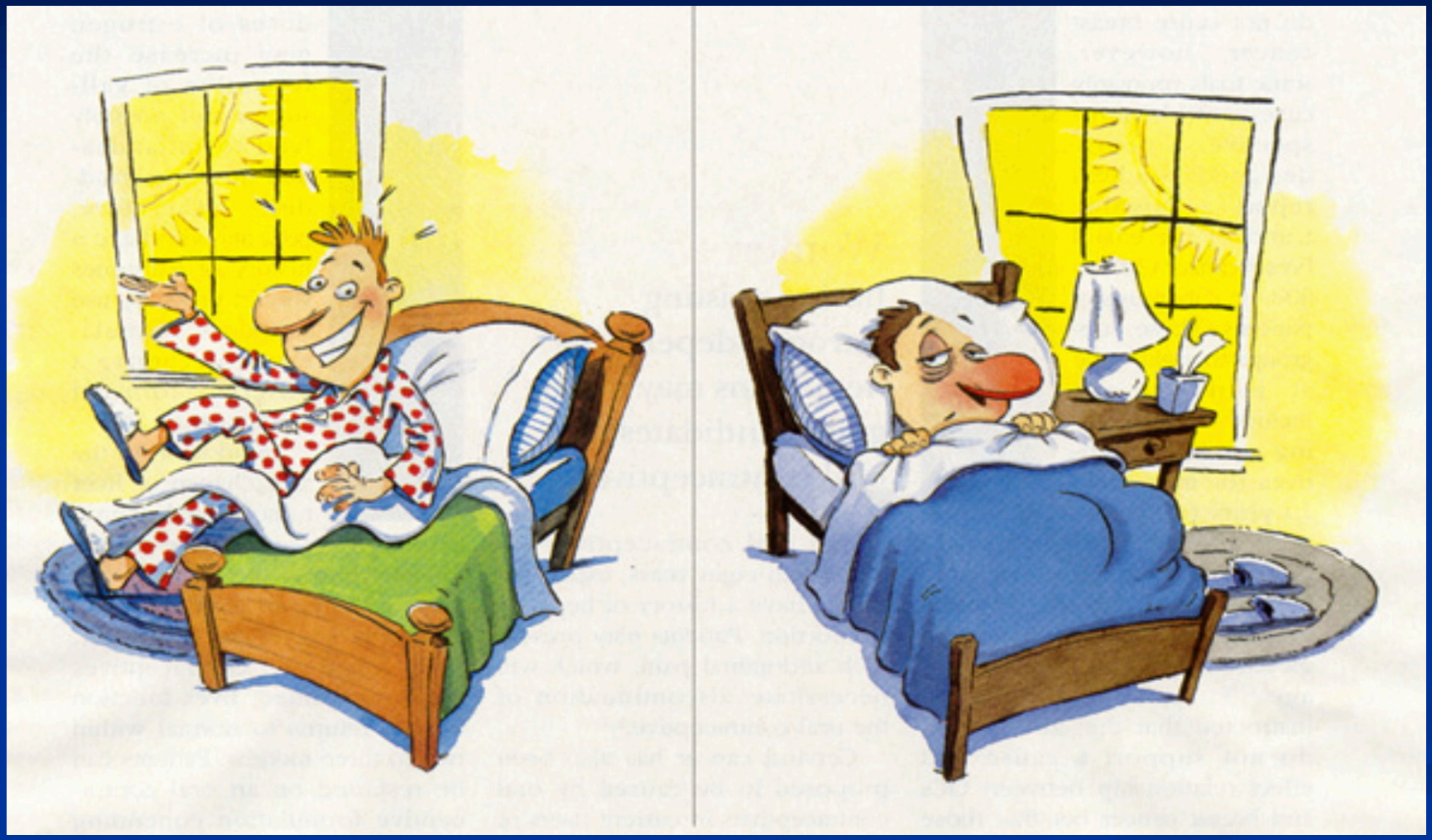
- Properties: potent anti-inflammatory agents; no antipyretic effects.
- Disadvantages
  - PGE_2 inhibition --> decreases muco-protective lining (stomach) --> GI upset, gastritis, GI bleeding / ulcers
  - PGI_2 (prostacyclin) inhibition (kidneys) --> decreases renal blood flow --> decreases GFR --> increases sodium/water retention --> exacerbates HTN / CHF
  - Systemic adverse effects with short-term and long-term use: HPA-axis suppression, immunosuppression, cataract formation, osteoporosis, myopathy, weight gain, hypertension, hyperglycemia, etc …

Opioids: Morphine, Codeine, Hydrocodone, etc…

- Properties: potent analgesic effects; no antipyretic effects and no anti-inflammatory properties.
- Disadvantages: drug tolerance, drug dependence, and potential for opioid addiction.

COLD & FLU

Treatment Considerations for Cold & Flu



Last night, he took his pharmacist's advice.

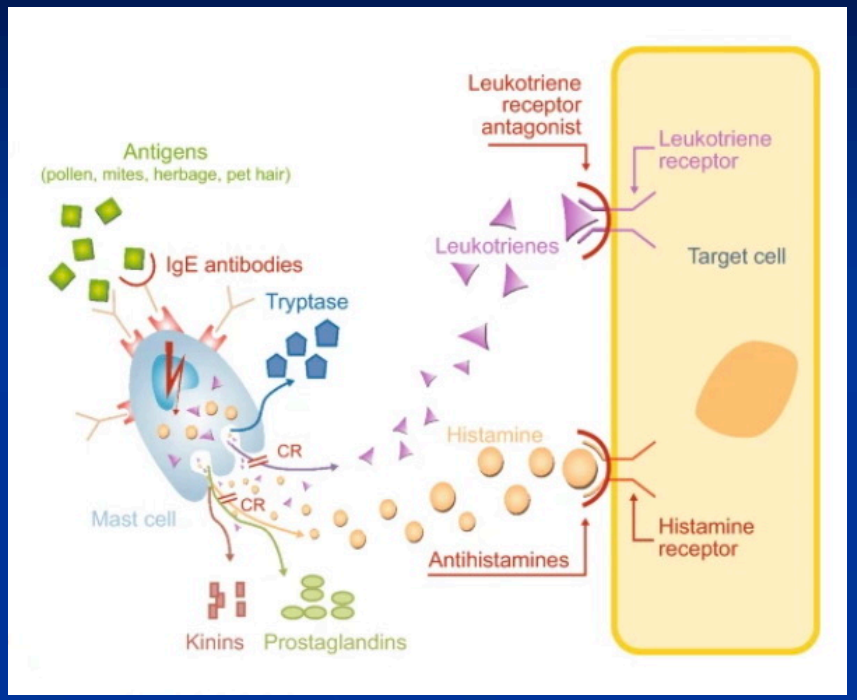
He didn't.

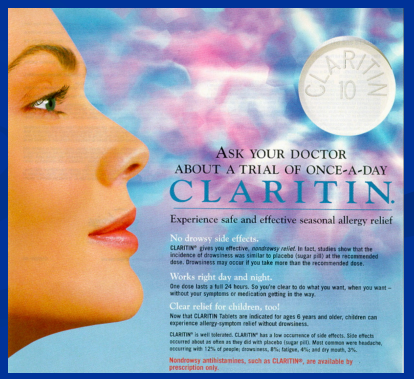
Cold & Flu Considerations

- What are the advantages and disadvantages of using multi-symptom combination products such as Nyquil?

Antihistamines

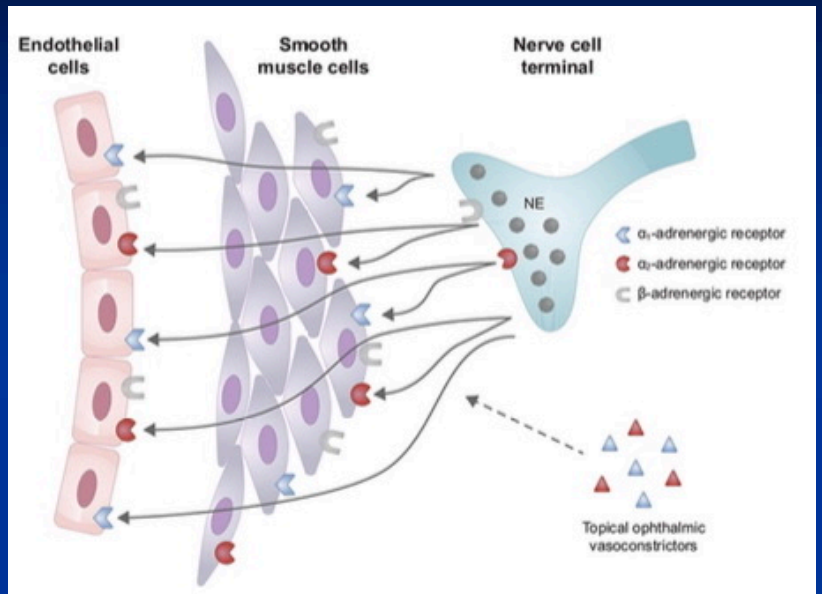
- MOA: Antihistamines have been reclassified as "inverse agonists," rather than H_1 receptor antagonists; therefore, antihistamines induce a conformational change in the structure of the H_1 receptor and prevent histamine from binding and activating it.
- Antihistamines are divided into 1st and 2nd generation agents.
  - 1st-generation antihistamines include: diphenhydramine (Benadryl), chlorpheniramine (Chlor-Trimeton), hydroxyzine (Atarax, Vistaril), promethazine (Phenergan).
  - 2nd generation (minimally-sedating) antihistamines include: cetirizine (Zyrtec), fexofenadine (Allegra), desloratadine (Clarinex), loratadine (Claritin), levocetirizine (Xyzal).





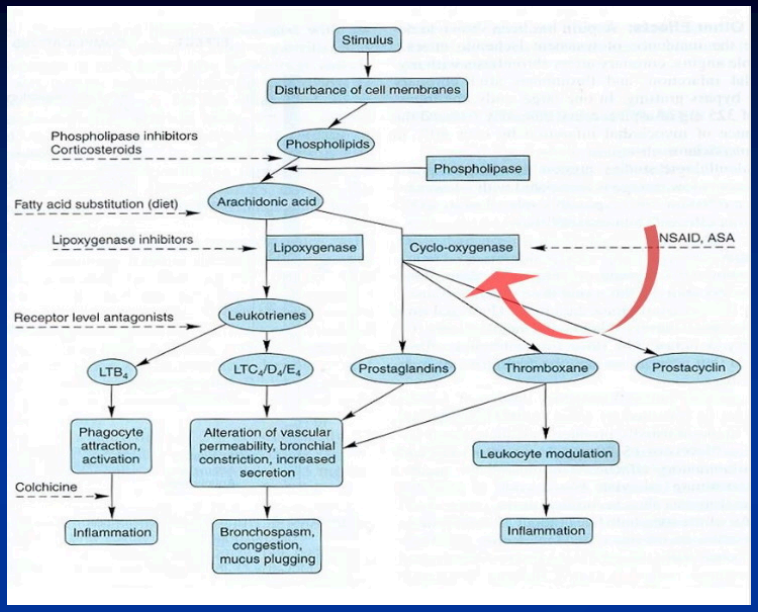
Decongestants

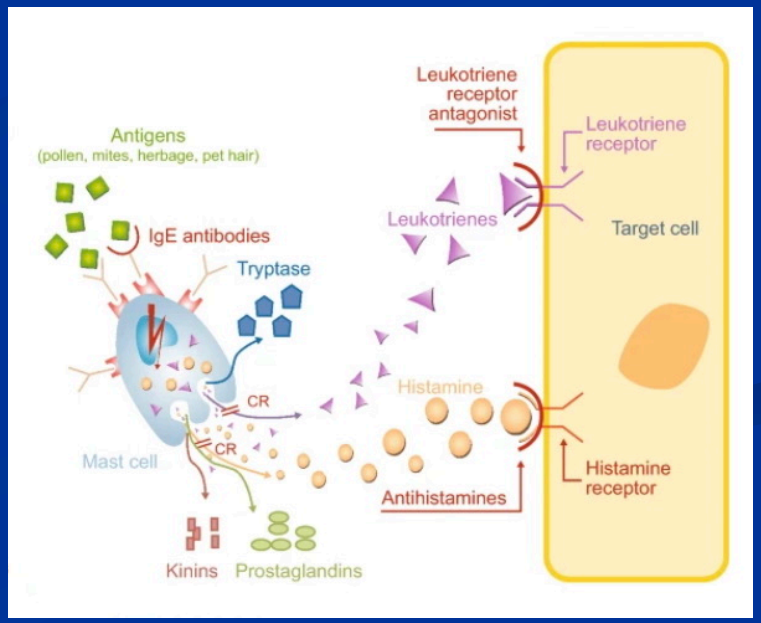
- MOA: Nasal decongestants are alpha$_1$ receptor agonists → vasoconstriction of nasal vessels→ reduce mucus production and edema → decrease congestion.
- OTC nasal decongestant products include: phenylephrine (Neo-Synephrine) and oxymetazoline (Afrin).
- Caution: Nasal decongestants sprays can cause *rhinitis medicamentosa* (i.e., rebound congestion) after 5-7 days of continuous use.



Glucocorticoid Nasal Sprays

- MOA: Inhaled glucocorticoids are non-specific suppressors of inflammation: Glucocorticoids inhibit arachidonic acid metabolism, resulting in the decreased production of leukotrienes (LT) and prostaglandins (PG).
- Glucocorticoid nasal sprays, such as fluticasone (Flonase), are the most effective pharmacologic agents for allergic rhinitis and are recommended for moderate-severe allergic rhinitis.

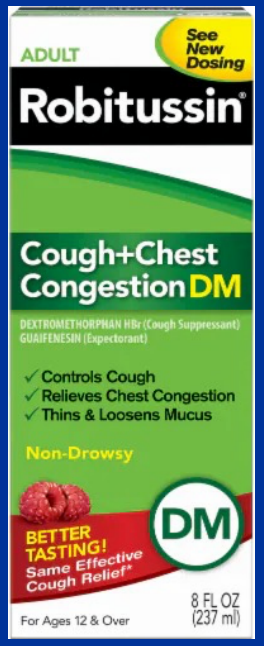




Antitussives

- Antitussives, known as cough suppressants, relieve coughing by acting on the cough center in the brain to suppress the cough reflex.

Dextromethorphan (Robitussin DM)

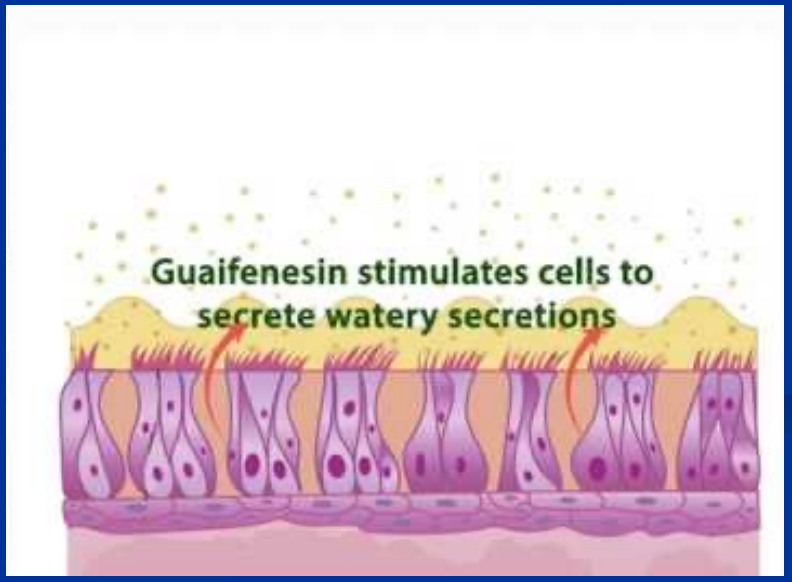


Codeine Syrup (Robitussin AC)

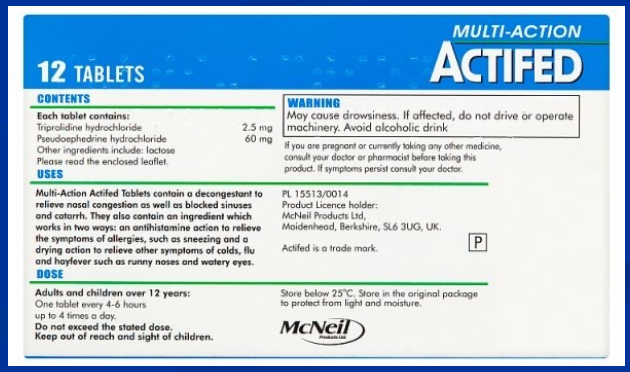
Diphenhydramine (Benylin)

Expectorants

- Guaifenesin is the most common OTC expectorant in products such as Robitussin (Plain) Syrup and Mucinex.
- Expectorants thin and loosen mucus and airway secretions, making it easier to cough up and expel.



Combination Cold/Flu Products Considerations



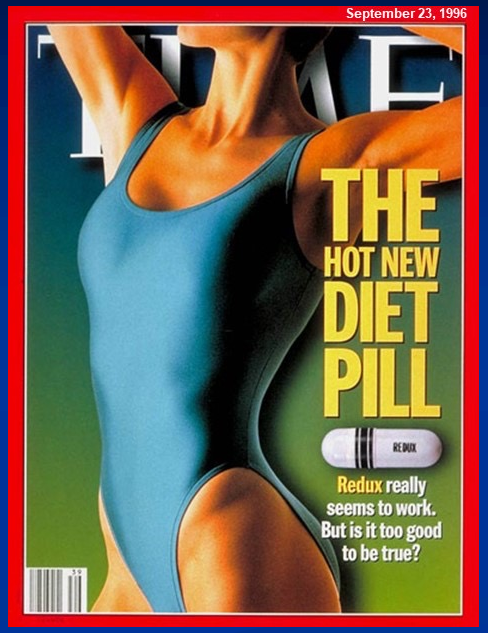
Hypnotics

- Sedatives are used to reduce anxiety symptoms; whereas hypnotics are used to treat insomnia by inducing sleep.

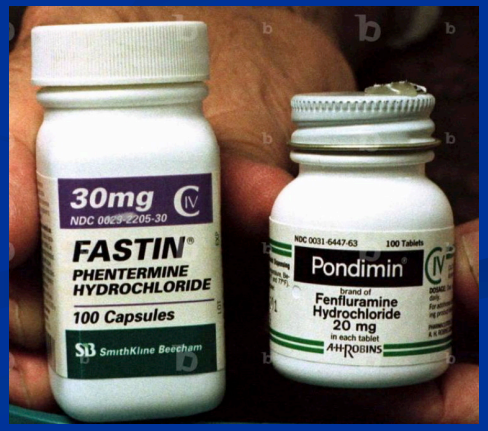
Diphenhydramine 25 mg softgels

APAP 500 mg + Diphenhydramine 25 mg caps

Appetite Suppressants



- Phen-Fen, a popular combination appetite suppressant regimen during the 1990s, consisted of Fastin (phentermine) plus Pondimin (fenfluramine).
- Redux (Dexfenfluramine), an isomer of fenfluramine, was also a popular appetite suppressant marketed during the Phen-Fen fad.

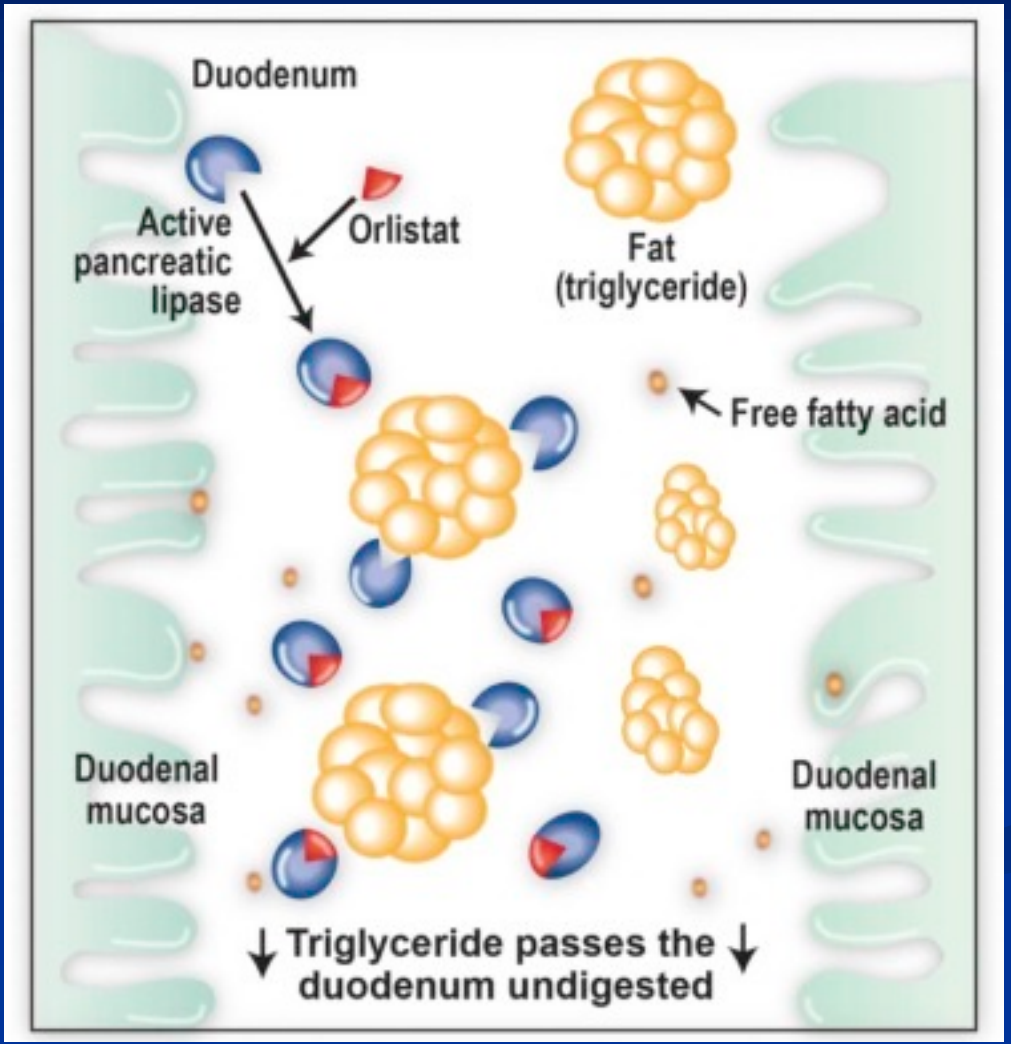


Phen-Fen Diet → Key Points …

- Phen-Fen consisted of phentermine (Fastin) plus fenfluramine (Pondimin), a potent synergistic combination product.
- Phentermine increases norepinephrine (NE) in appetite center of the brain → reduces appetite.
- Fenfluramine increases serotonin in appetite center → reduces appetite.
- High doses of fenfluramine (Pondimin) and dexfenfluramine (Redux) results in pulmonary hypertension.

Xenical (Orlistat)

- MOA: orlistat inhibits pancreatic lipases → inhibits breakdown of fats into fatty acids + glycerol → reduces absorption of fatty acids (9 kcal/gm) → reduces body weight.
- Side Effects: steatorrhea, fluid and electrolyte depletion, and fat-soluble vitamin deficiencies (i.e., vitamins A, D, E, K).



Appetite Suppressants

GLP-1 Receptor Agonists

Semaglutide (Ozempic, Wegovy)
Tirzepatide (Zepbound, Mounjaro)
Dulaglutide (Trulicity)
Liraglutide (Victoza)

↓

Increase Insulin Release

+

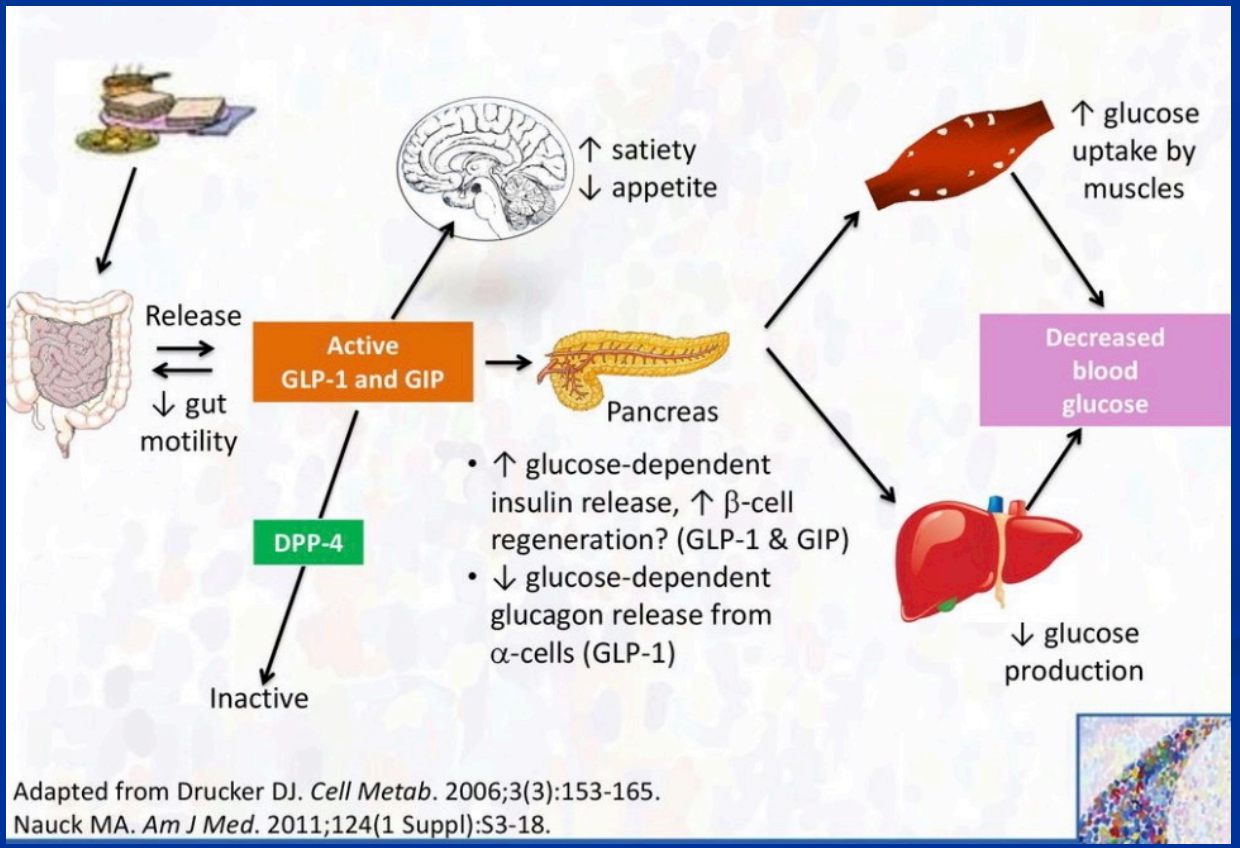
Decrease Glucagon Release

+

Increase Satiety

+

Delay Gastric Emptying Time



Lifestyle Modifications

Dietary Considerations & Exercise

Sample Caloric Requirement (BEE) Calculation for Stressed Patients

Female: 655 + (9.6 x wt. in kg) + (1.85 x ht. in cm) – (4.7 x age)

Male: 66 + (13.7 x wt. in kg) + (5.00 x ht. in cm) – (6.8 x age)

Sample Calculation (based on patient-specific parameters: ht, wt, age, and disease state)

S.Y. is a 64 year-old female patient with major sepsis. Calculate her caloric requirement based on her pathologic condition. Her height is 5'4" and body weight is 140 pounds.

Conversion Factors:

- body weight from pounds to kg. : 140 lbs / 2.2 = 63.64 kg
- height from inches to cm. : 5'4" = 64 inches x 2.54 = 162.56 cm

BEE = 655 + (9.6 x **63.64**) + (1.85 x **162.56**) – (4.7 x **64**)

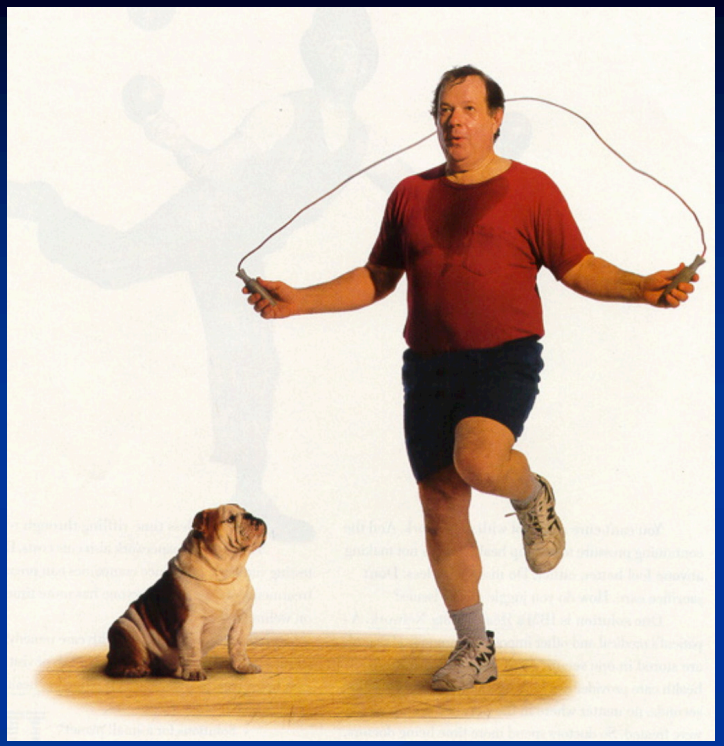
= (655 + 610.94 + 300.74) – (300.8)

= 1265.88 kcal / day

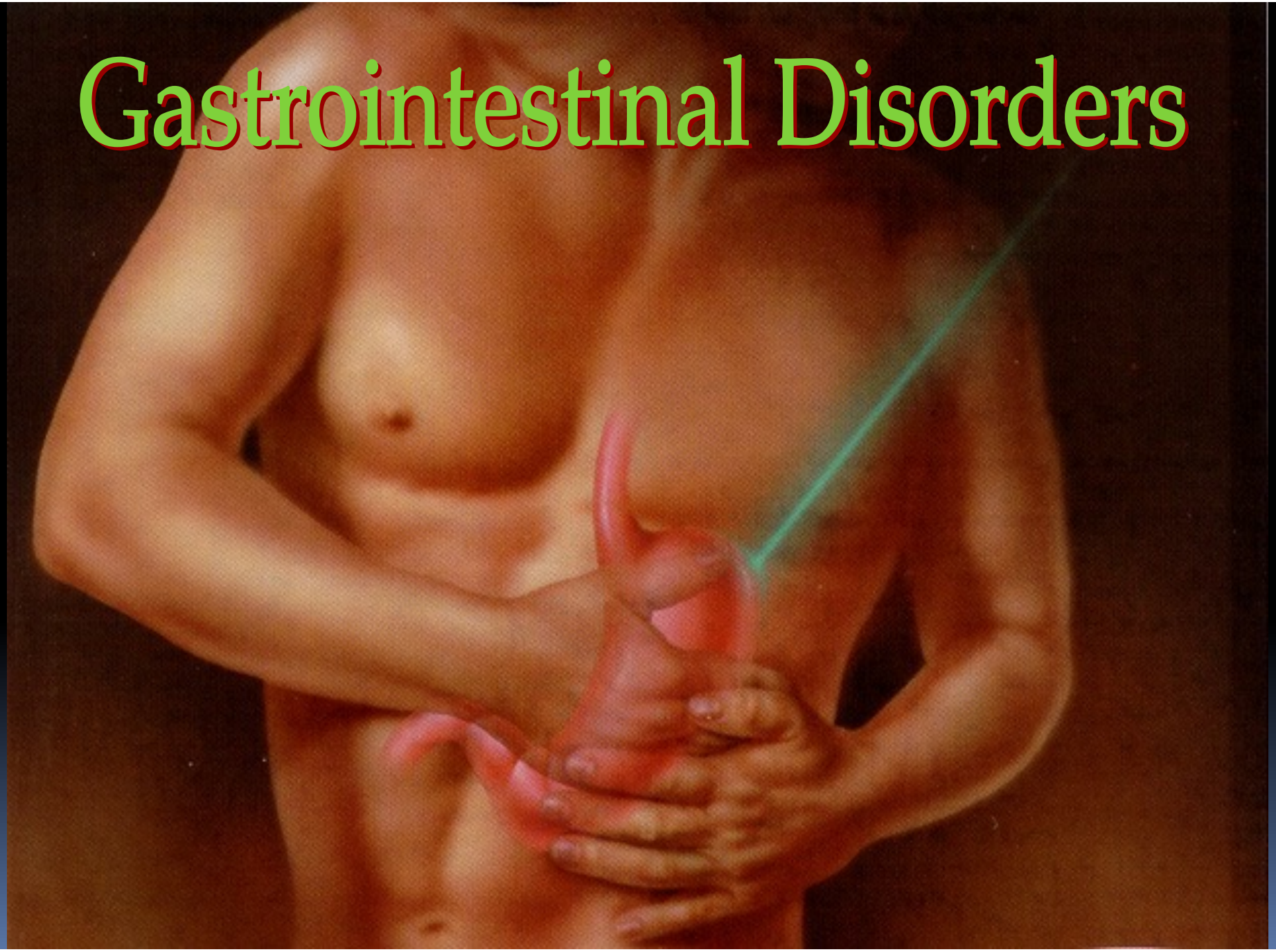
Multiply the BEE value by the appropriate "disease stress factor", which provides additional calories to account for the degree of physiologic stress (based on increased metabolic requirement during pathologic condition – i.e., major sepsis).

BEE for major sepsis = 1.5 x 1265.88

Answer → 1898.82 kcal / day



Gastrointestinal Disorders



Antacids: Maalox / Mylanta

MOA: Neutralize Gastric Acidity

Active Ingredients
(neutralizing agents)

(1) magnesium
(SE: osmotic diarrhea)

(2) aluminum
(SE: constipation)

* SE = Side Effect(s)

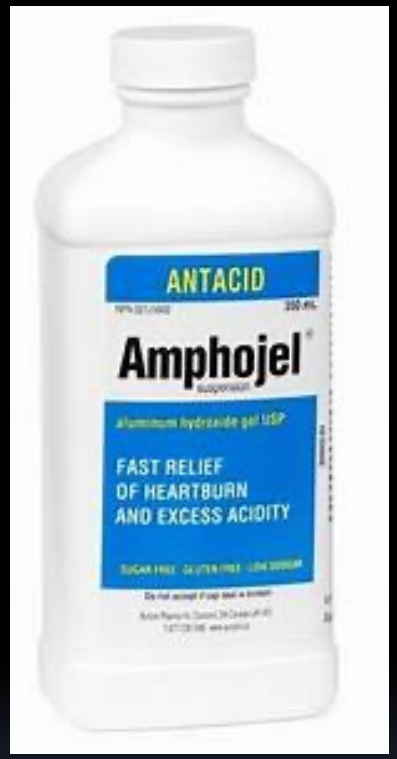
Antacids (cont.)

Milk of Magnesia (magnesium)

- magnesium → neutralizes hyperacidity
- magnesium → treats constipation

Amphojel (aluminum hydroxide)

- aluminum → neutralizes hyperacidity
- neutralizing agent → treats diarrhea



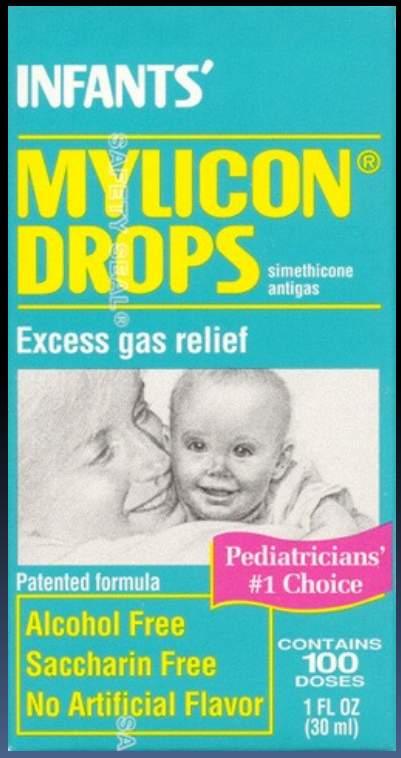
Antacids (Maalox, Mylanta)

- Onset: immediate (minutes)
- Duration: 30 minutes on an empty stomach, but 3 hours when taken within 1 hour of meals
- Alginic acid may be added to antacids → forms a viscous solution that floats on top of gastric contents → protects the esophageal mucosa from acid reflux
- Simethicone (surfactant) may be added to antacids → ”breaks up” gas bubbles → relieves gas
- Caution: small amounts of aluminum and magnesium are absorbed and can accumulate in renal insufficiency → toxicity
  - Magnesium: avoid in patients with CrCl < 30 ml/min
  - Aluminum: avoid in patients with renal failure (CrCl < 15 ml/min)

Antacids (cont.)

Simethicone (Mylicon)

Simethicone (surfactant) → decreases surface tension of gas bubbles → breaks up gas bubbles → relieves gas



Antacids (cont.)

Calicum Carbonate (TUMS)

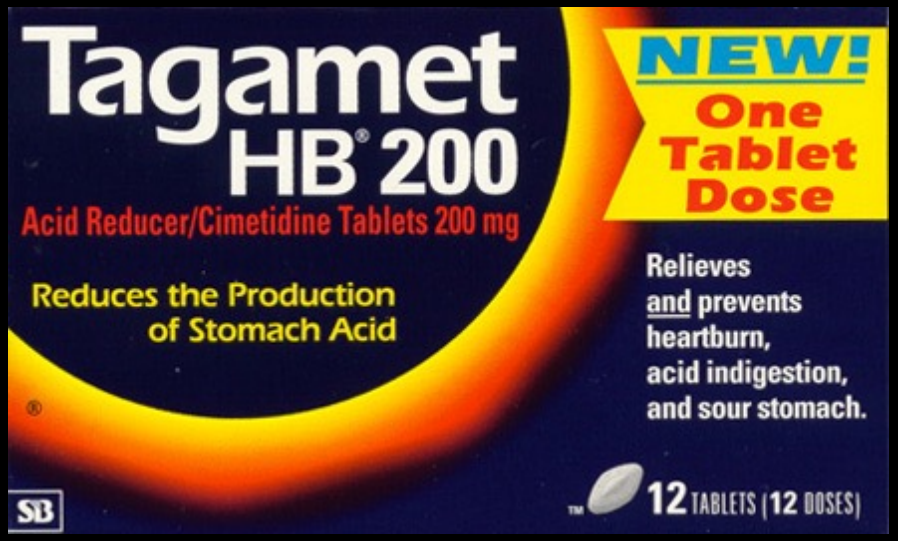
- moderate neutralizing capacity, compared to Maalox/Mylanta
- $CaCO_3$ → gas formation
  → burping / flatulence
- high-doses (4-8 grams/day)
  → hypercalcemia / metabolic alkalosis
  "milk-alkali syndrome" → kidney failure

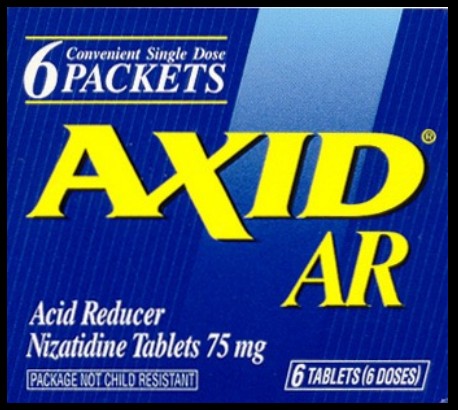
Antacids (cont.)

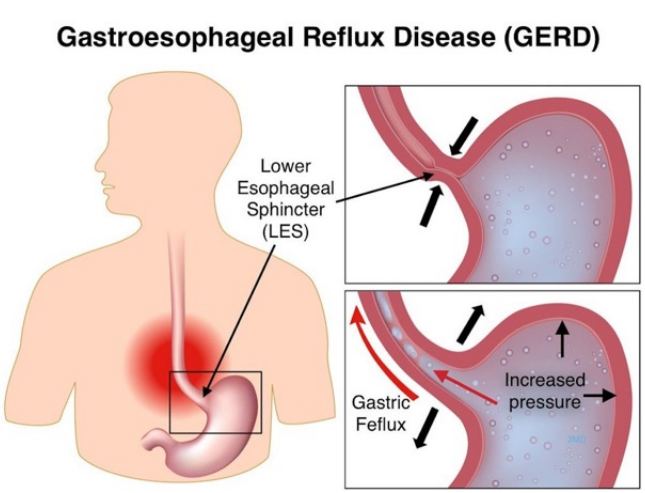
Sodium Bicarbonate
(Alka-Seltzer)

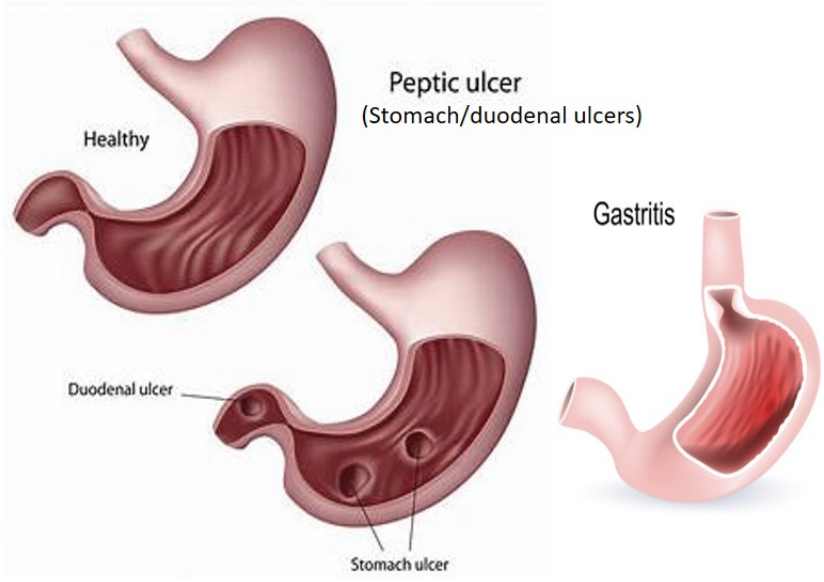
High sodium content (567 mg per tablet) → Na^+/H_2O retention → exacerbates hypertension, heart failure, chronic kidney disease

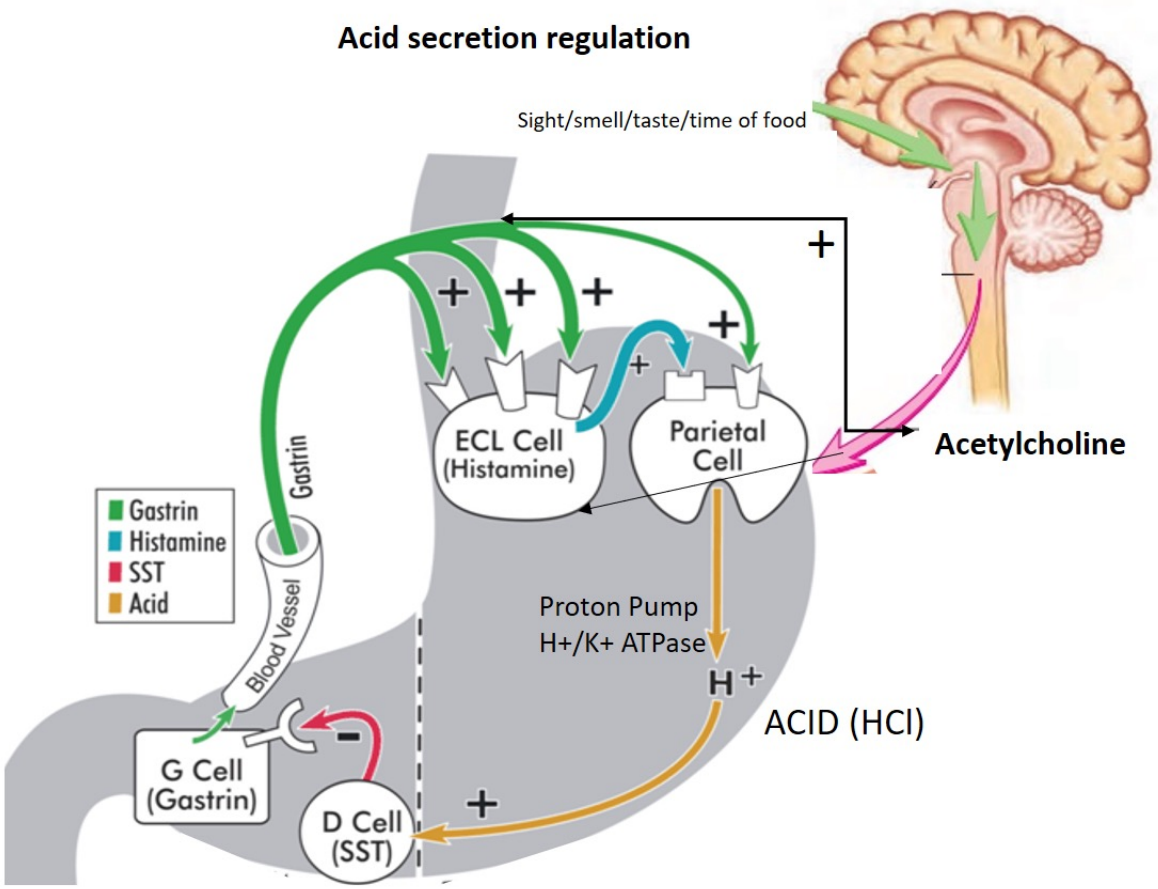
H_2 Receptor Antagonists (H_2RAs)





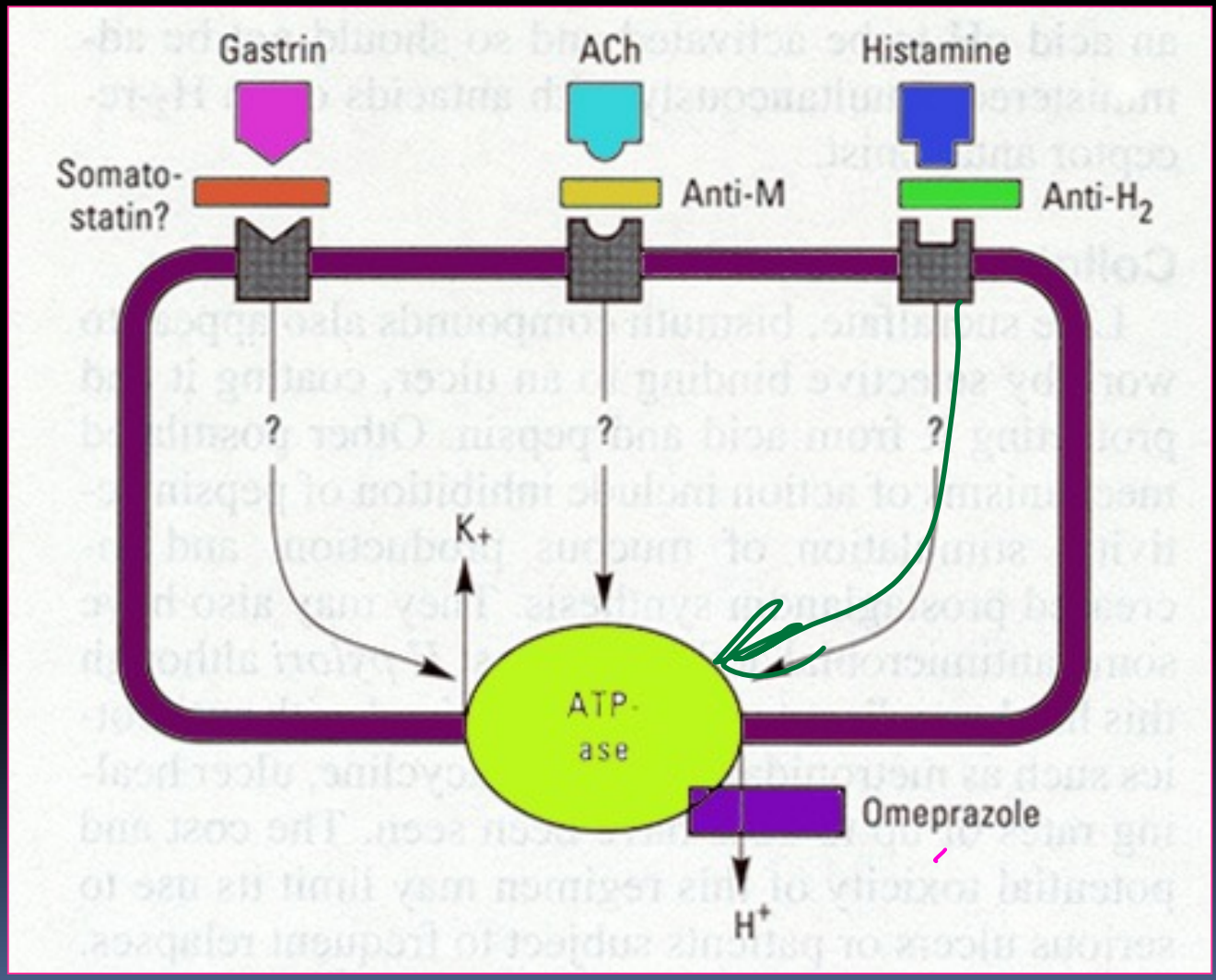






G-cells (antrum) → produce gastrin. D-cells protect stomach from overproduction of gastric acid by releasing somatostatin (SST) → inhibits production of gastrin. ACh and gastrin → increase release of histamine-2 from enterochromaffin-like (ECL) cells. Gastrin, Histamine-2, ACh (acetylcholine) → bind to receptors on parietal cells → gastric acid secretion

H_2 Receptor Antagonists (H_2RAs)



Histamine-2 Receptor Antagonists (H_2RA)

- H_2RAs are remarkably safe
- Oral absorption is rapid → peak serum drug concentration: 1-3 hours
- Side Effects (SEs)
  - GI Discomfort: diarrhea, constipation
  - CNS Effects: headache, dizziness, drowsiness, lethargy
  - Dermatologic Effects: rash
  - Hematologic Effect: thrombocytopenia (1%) is reversible upon discontinuation of H_2RA
- Cimetidine (Tagamet) has the greatest potential for drug-drug interactions → inhibits hepatic cytochrome P-450 isoenzymes
  → inhibits metabolism of theophylline, phenytoin, warfarin
  → drug toxicities

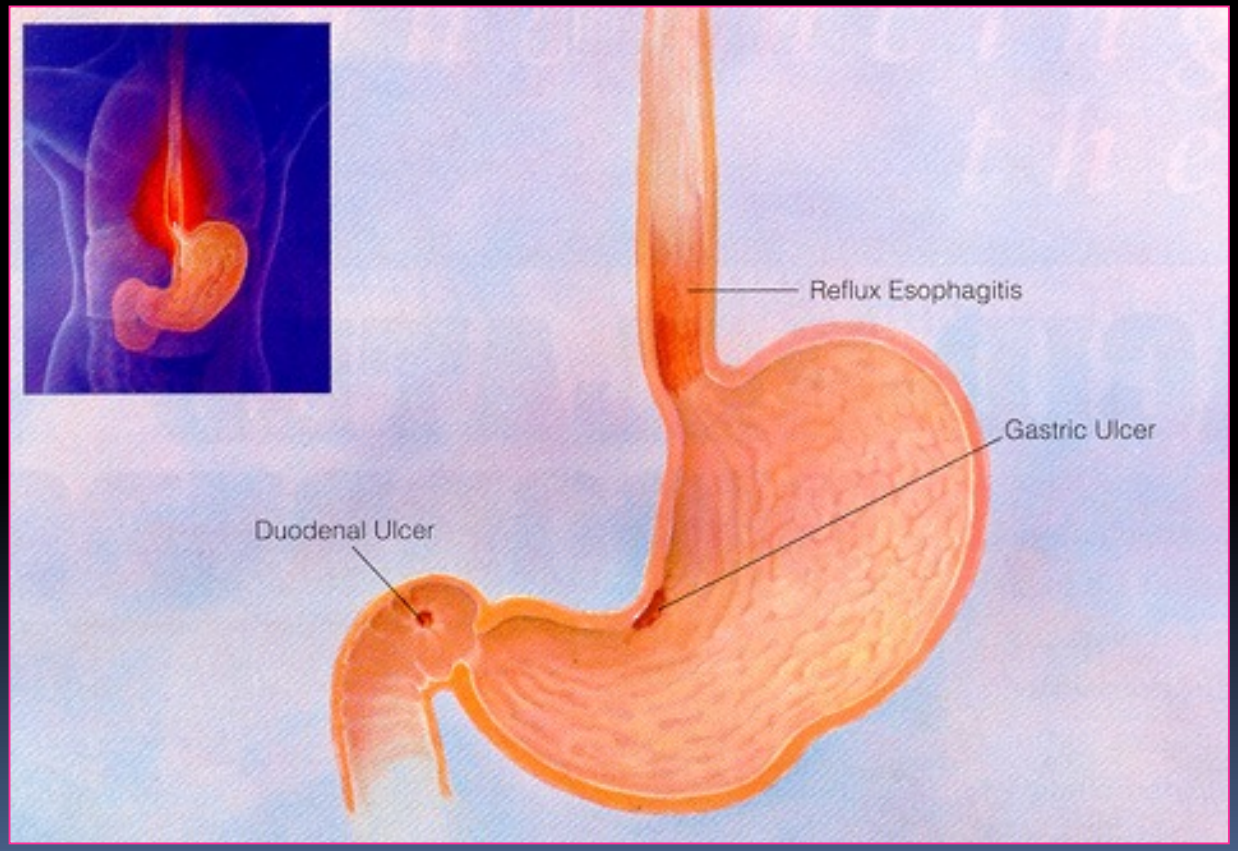
Histamine-2 Receptor Antagonists (H_2RA)

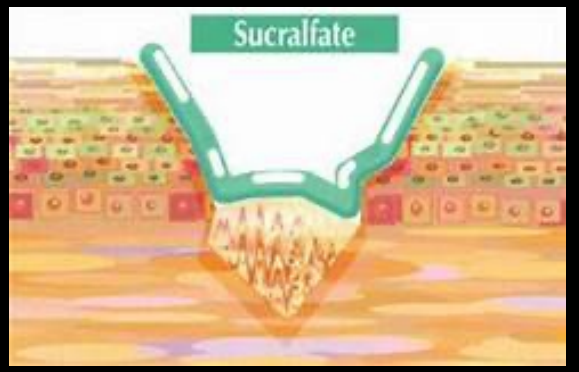
- Tachyphylaxis or tolerance may develop after 2-6 weeks of H_2RA therapy due to upregulation of H_2 receptor sites.
- Development of tachyphylaxis limits the use of H_2RAs in management of GERD and other conditions requiring long-term therapy.

Sucralfate (Carafate)

(cytoprotective agent)

MOA: binds to gastric ulcer forming a protective barrier



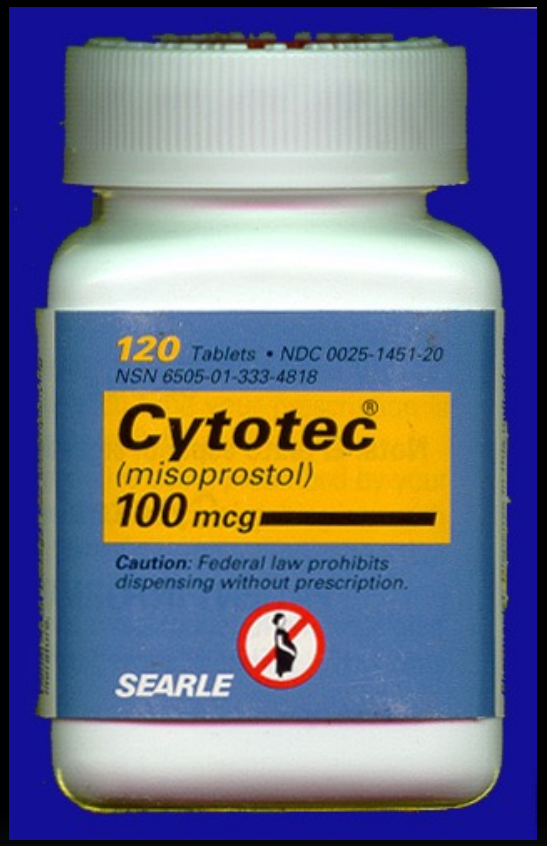


Sucralfate (Carafate)

- Sucralfate may also have protective affect by stimulating release of mucosal prostaglandins (PGE)
- SE: constipation (1-3%) due to aluminum content
- Caution: aluminum content may accumulate in patients with renal insufficiency → "aluminum encephalopathy" (i.e., dementia), and anemia

- Aluminum binds dietary phosphate (GI tract) → hypophosphatemia
- Sucralfate tablets are large and may be difficult for geriatrics to swallow → use liquid formulation

Misoprostol (Cytotec)



MOA: synthetic prostaglandin (PG) analog

- stimulates the production of mucus and bicarbonate ("mucoprotective shield")
- improves mucosal blood flow
- reduces mucosal cell turnover
- mildly inhibits gastric acid secretion (less than H_2RAs)

SE: diarrhea (up to 30%), abdominal cramping

- take with food and reduce daily dose to minimize incidence of diarrhea

Caution: misoprostol is contraindicated in pregnancy.

- use in women in childbearing years requires negative serum pregnancy test and adequate contraception

Proton Pump Inhibitors (PPIs)

MOA:

- PPIs irreversibly bind to the proton pump and inhibit gastric acid secretion
- PPIs are the most potent inhibitors of gastric acid secretion (superior to H_2RAs)
- PPIs are indicated for patients who experienced tx failure with maximum doses of H_2RA
- Dosage reduction of PPIs is not required in renal insufficiency

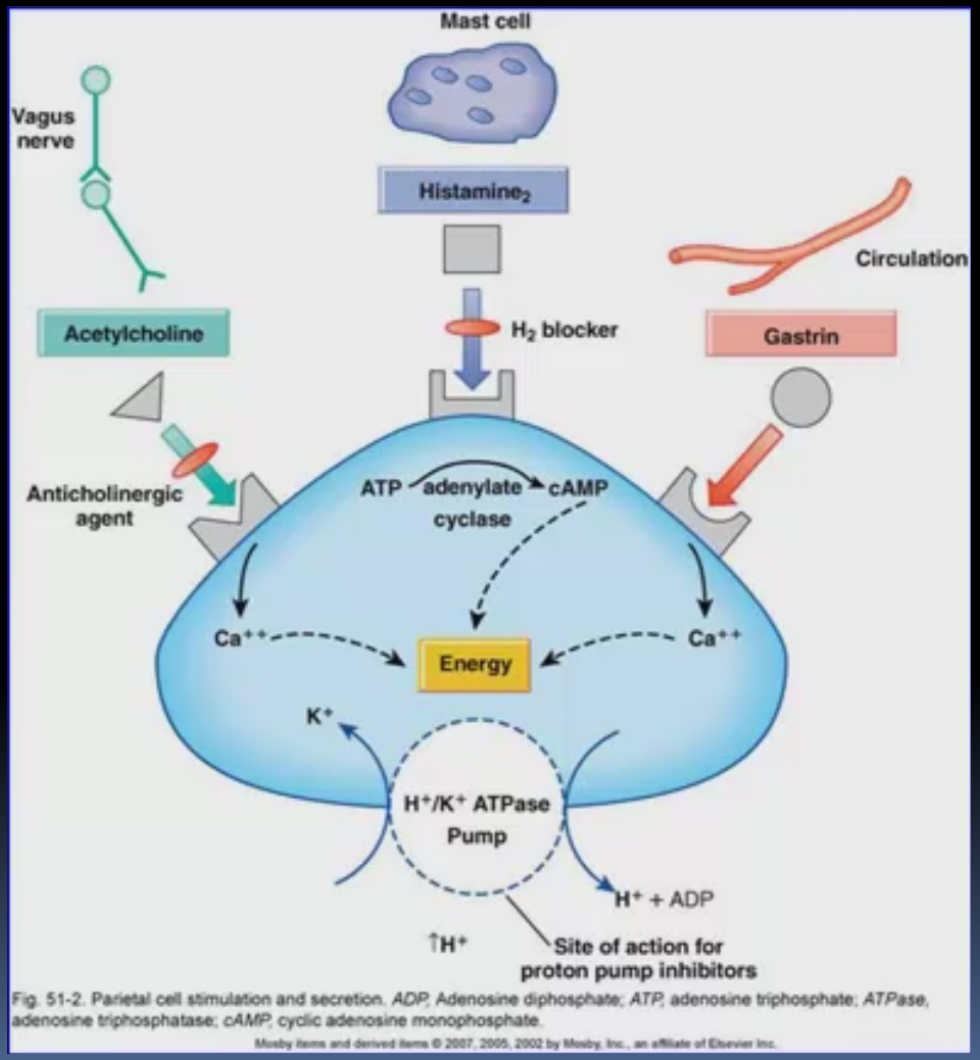


Fig. 51-2. Parietal cell stimulation and secretion. *ADP*, Adenosine diphosphate; *ATP*, adenosine triphosphate; *ATPase*, adenosine triphosphatase; *cAMP*, cyclic adenosine monophosphate.

Proton Pump Inhibitors (PPIs)

Table 1. Availability, Formulations, and Dosages for Proton Pump Inhibitors in Adults

Drug	*Availability*	*Route of administration*	*Starting dosage**	*Cost of generic (brand)†*
Dexlansoprazole (Dexilant)	Prescription	Oral	30 mg per day	NA ($153)
Esomeprazole (Nexium)	Prescription	Oral or IV	Oral: 20 mg per day IV: 20 mg per day for 10 days	Oral: NA ($201) IV: NA ($381)‡
Lansoprazole (Prevacid)	Prescription	Oral	15 mg per day	$106 ($196)
Lansoprazole (Prevacid 24H)	Over-the-counter	Oral	15 mg per day for 14 days§	NA ($13)
Omeprazole (Prilosec, Zegerid)	Prescription	Oral	20 mg per day	$33 ($196)
Omeprazole (Prilosec OTC, Zegerid OTC)	Over-the-counter	Oral	20.6 mg (Prilosec OTC) or 20 mg (Zegerid OTC) per day for 14 days§	$7 ($13)
Pantoprazole (Protonix)	Prescription	Oral or IV	Oral: 40 mg per day IV: 40 mg per day for 7 to 10 days	Oral: $16 ($186) IV: $42 ($42)‡
Rabeprazole (Aciphex)	Prescription	Oral	20 mg per day	NA ($250)

IV = intravenous; NA = not available.

**—Number of weeks of recommended treatment varies.*

†—*Estimated retail price of one month's treatment (unless otherwise specified) based on information obtained at http://www.drugstore.com (accessed January 31, 2012) or at a national retail chain.*

‡—*Estimated wholesale price based on information obtained at Red Book online. Micromedex 2.0. Micromedex Healthcare Series [Internet database]. Greenwood Village, Colo.: Thomson Reuters (accessed January 31, 2012).*

§—*Patients should not take more often than 14 days per month every four months.*

Proton Pump Inhibitors (PPIs)

Short-Term SEs of PPIs (infrequent and comparable to H_2RAs)

- GI discomfort: nausea, diarrhea, abdominal pain
- CNS: headache, dizziness

Long-Term SEs of PPIs (usually with high doses)

- Atrophic gastritis has been "rarely" associated with patients on long-term therapy PPIs for *Helicobacter pylori*.
- Risk of *C. difficile* and other enteric infections has been observed due to ability of pathogens to survive in a less acidic GI environment; however the overall risk is low.
- Vit B_{12} deficiency, since gastric acid is required to extract Vit B_{12} from dietary sources. Monitor Vit B12 levels in PPI patients.

Long-Term SEs of PPIs

- Hypomagnesemia may occur with long-term use of PPIs due to reduced intestinal absorption. Monitoring serum magnesium levels is recommended in patients on long-term PPI therapy.

- Hypocalcemia and increase risk of fractures is associated with reduced calcium absorption due to hypochlorhydria. Since calcium citrate does not require acid for absorption, it is the recommended calcium supplement in patients on long-term PPI therapy.

- Iron malabsorption secondary to long-term gastric acid suppression with PPIs, however this does not appear to be of clinical significance unless a patient requires oral iron supplementation. Higher doses and longer duration of iron supplementation are recommended in these patients.

Guidelines for Self-Medication

(1) read the label carefully

(2) follow the directions for use

(3) if symptoms persist, seek professional advice

(4) OTC drugs do not cure illness

(5) don't use expired or old medications

(6) store medications properly

(7) avoid OTC products with identical medications

Guidelines for Self-Medication (cont.)

(8) consult a pharmacist or a physician assistant for information on OTC drugs

(9) select an economical generic OTC product when available (generic vs brand)


PHARMACY
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