Classification and Mechanism of Action

NSAID

Non steroidal anti-inflammatory Drugs

*Classification- KD

Non-Selective COX Inhibitor	Preferential COX_2 Inhibitor	Selective COX_2 Inhibitor	Analgesic-Antipyretic with poor Anti-inflammatory Action
	Nimesulide, Diclofenac, Aceclofenac, Meloxicam, Etodolac	Celecoxib, Etoricoxib, Parecoxib	

Category	Example
Salicylates	Aspirin
Propionic acid derivative	Ibuproprofen, Naproxen, Ketoprofen, Flurbiprofen
Fenamate	Mephenrnic acid
Enolic acid derivative	Piroxicam, Tenoxicam
Acetic acid derivative	Ketorolac, Indomethacin, Nabumetone
Pyrazolone derivative	Phenylbutazone, Oxyphenbutazone

Example
Paracetamol (Acetaminophen)
Metamizol, Propiphenazone
Nefopam

*Constitutive = Constant Production

Key Point (Solution) - As name Indicate NSAIDs are those agents which are used to get relief from pain, inflammation and fever. And as per the COX pathway we understand that **COX-1** and **COX-2** ultimately form prostaglandin which initiates perception of pain and inflammation. So anyhow if we block or inhibit the synthesis of PG we may reduce pain and inflammation. Although COX-1 is constitutive in nature thus it always get secreted without induction of injury and called as a house keeper so it's better to inhibit COX-2 rather than COX-1.

* Tissue Injury
(Applicable for COX-2)

Breakdown of Membrane Phospholipids

↓

Arachidonic acid

Mechanism of Action

Cyclooxygenase Pathway

*Rough Idea- Lippincott

COX-1 gene transcription

COX-2 gene transcription

Induced by-
Oxidative Stress, Injury, Ischemia, Seizures

Glucocorticoids — Inhibit

COX-1 perform housekeeping function
Like neutralizing gastric acids

Inhibit

mRNA

Inhibit

Non-Selective
COX Inhibitor

COX- 1

Side Effect- Gastric Ulcer

mRNA

Non-Selective
COX Inhibitor

Inhibit

Selective
COX 2Inhibitor

Inhibit

COX- 2

PROSTAGLANDIN

*Diagrams and explainations are made by solution-pharmacy

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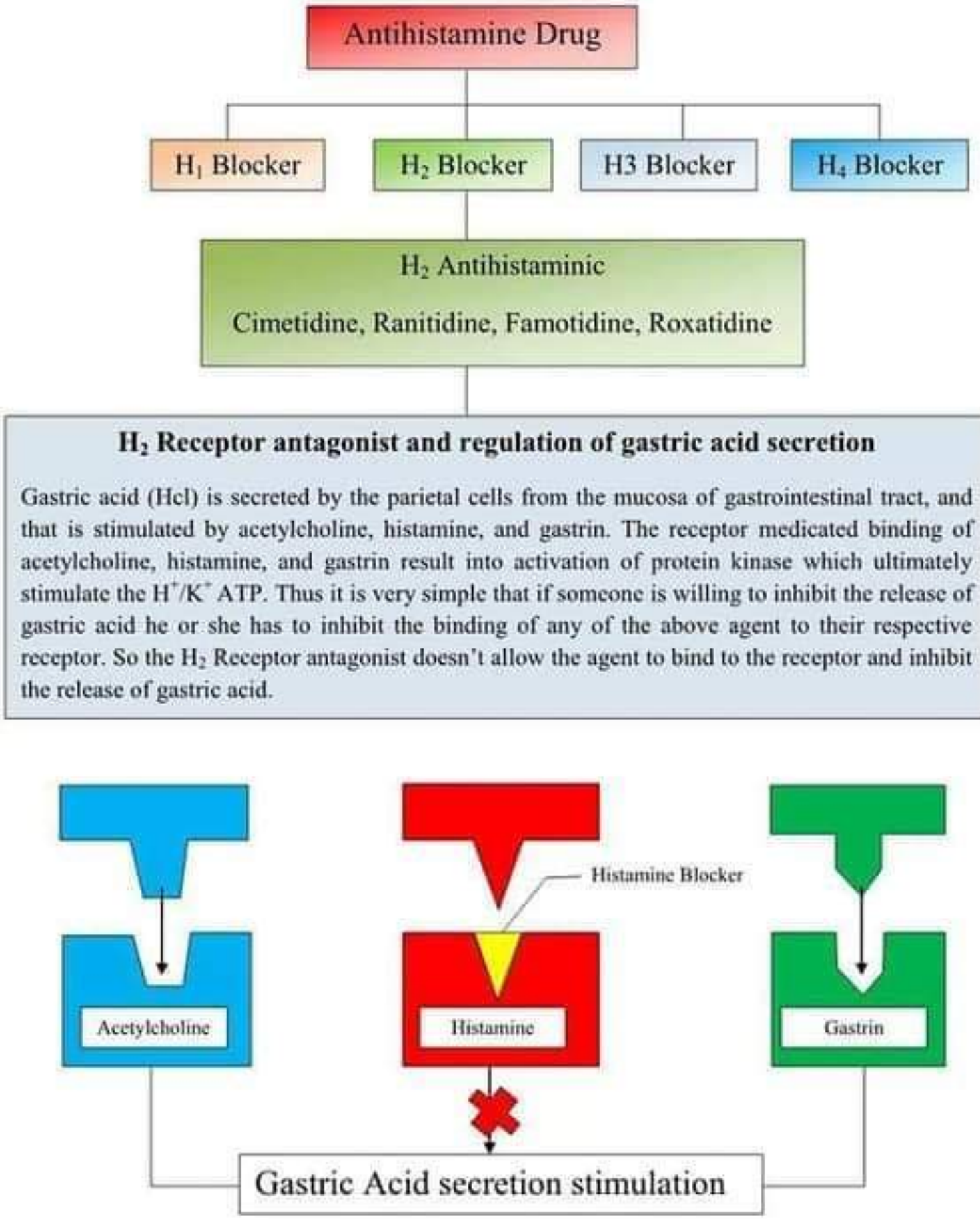
Classification and Mechanism of Action for Antihistamine Drug

Key Point to Understand- 'Histamine' is made up of two simple words- Histo (Tissue) + Amine. If we add them together the meaningful sentence will be- Amine released from tissue. Histamine is stored and release from mast cells. Other tissue like- Skin, gastric and intestinal mucosa, lungs, liver and placenta. Histamine receptors are of basically 02 types- (1) H1 and H2. H3 is also available. Histamine initiate allergic reaction thus antihistaminic drugs give relief from allergy by blocking any of the histamine receptor.



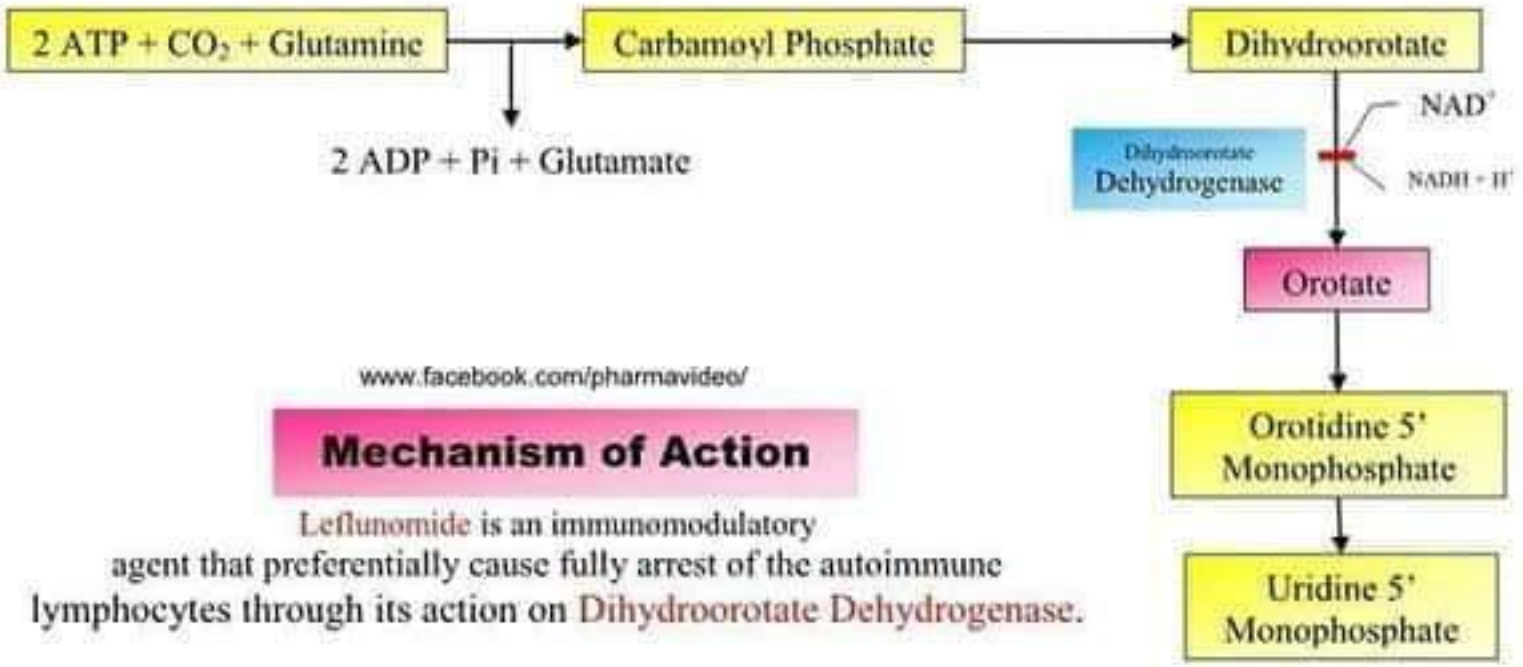
Diagrams and explainations are made by solution-pharmacy to make you better understand.

Antirheumatoid Drugs

Classification- KD Tripathi

- **Disease Modifying AR Drugs**
  Immunosuppressant-
  (Methotrexate, Azathioprine, Cyclosporine)
  Sulfasalazine, Chloroquine, Leflunomide, Gold sod. Thiomalate, Auranofin, d-Penicillamine
- **Biological Response Modifier**
  Etanercept
  Infliximab, Adalimumab,
  Anakinra
- **Adjuvant Drugs**
  Corticosteroids
  Prednisolone and others

Key point- Antirheumatoid arthritis (RA) is an autoimmune disease In RA there is joint inflammation, synovial proliferation and destruction of articular cartilage. These inflammatory cells secrete lysosomal enzyme which damage cartilage and erode bone, while PG produced in the process cause vasodilatation and pain.

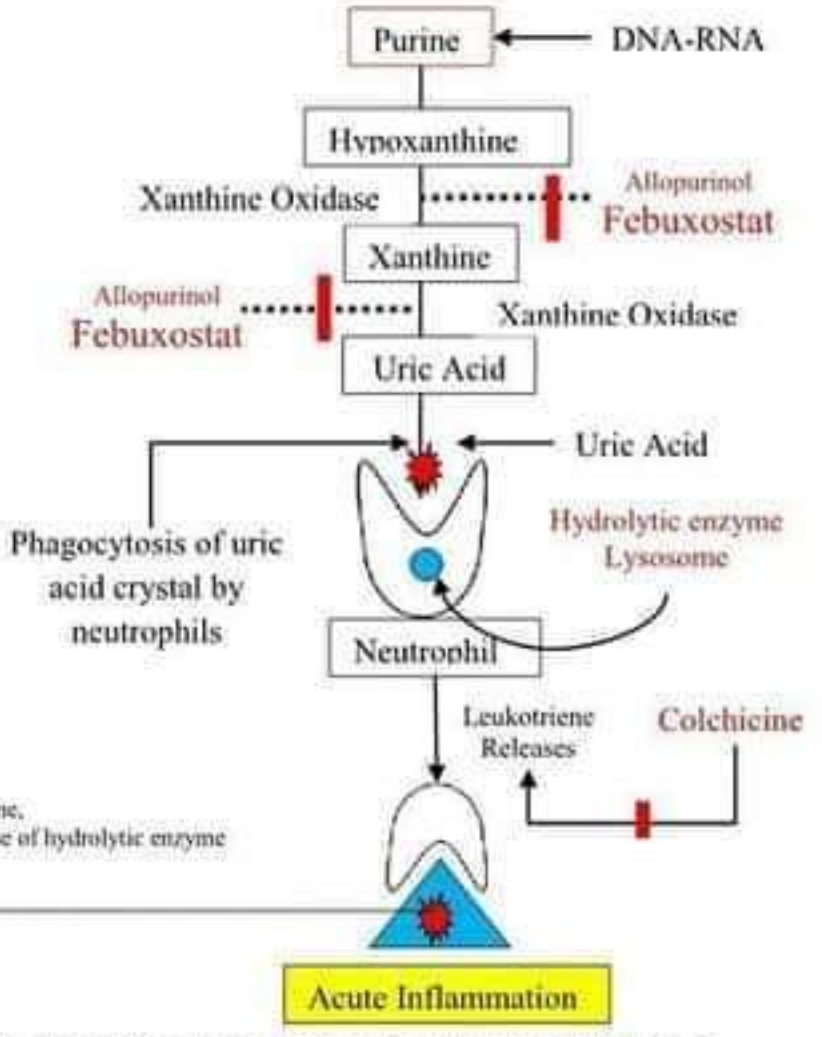


Mechanism of Action

Leflunomide is an immunomodulatory agent that preferentially cause fully arrest of the autoimmune lymphocytes through its action on Dihydroorotate Dehydrogenase.

Anti- Gout Drugs

- **For Acute Gout**
  - NSAID, Colchicine, Corticosteroids
- **For Chronic Gout**
  - Uricosurics Synthesis Inhibitor
    Probenecid, Sulfinpyrazone, Allopurinol, Febuxostat



Classification, Mechanism of Action and Tricks for

Drugs for Cough

Classification- KD Tripathi

A — Pharyngeal Demulcents
Lozenges, Cough Drops, Linctuses Syrup, Glycerin, Liquorice

B — Expectorants /Mucokinetics
Those which helps to Expel the cough. or which increase the kinetic movement of cough

C — Antitussive
Cough center suppressers
Drug which decrease sensitivity of cough center.

D — Adjuvant Antitussive
Bronchodilators, Salbutamol, terbutaline

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Mechanism of Action	Example of the Class
Bronchial secretion enhancer	Sodium citrate, Potassium citrate, Potassium Iodide, Guaiphenesin, Balsam of tolu, Vasaka, Ammonium Chloride. [illegible]
Mucolytics	Bromhexine, Ambroxole ,Acetyl cysteine Carbocisteine, [illegible]

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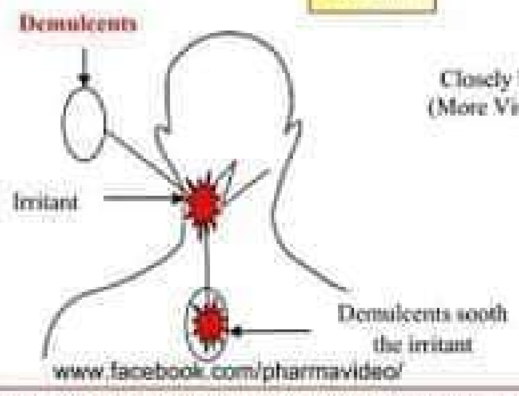
Mechanism of Action	Example of the Class
Opioids	Codeine, Pholcodeine [illegible]
Non Opioids	Noscapine, Dextromethorphan, Chlophedianol [illegible]
Antihistaminic	Chlorpheniramine, Diphenhydramine, Promethazine. [illegible] (Asthma is related with dust)

Key points- Coughing is a protective phenomenon until it does not hurt or create uneasiness. Cough is a protective reflex which tries to expel or eliminate the unwanted particles from our air passage along with mucus and other watery substances. The arising of cough is from stimulation of mechano or chemoreceptor present in throat, respiratory passage or in any other associated part of lungs. The main objectives of using anti cough drugs are to reduce the viscosity of cough so that they may be easy expelled. And this may achieve by breaking the bond between cough mucus which are used to bind them together.

A Mechanism



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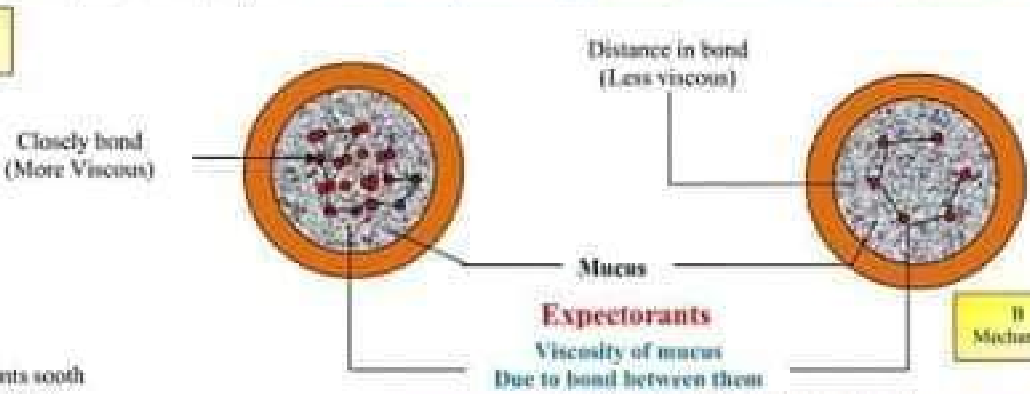
Demulcent sooth and cover the irritant and the stimulus released by irritant. This is not a treatment; this is just a temporary relief.



B Mechanism

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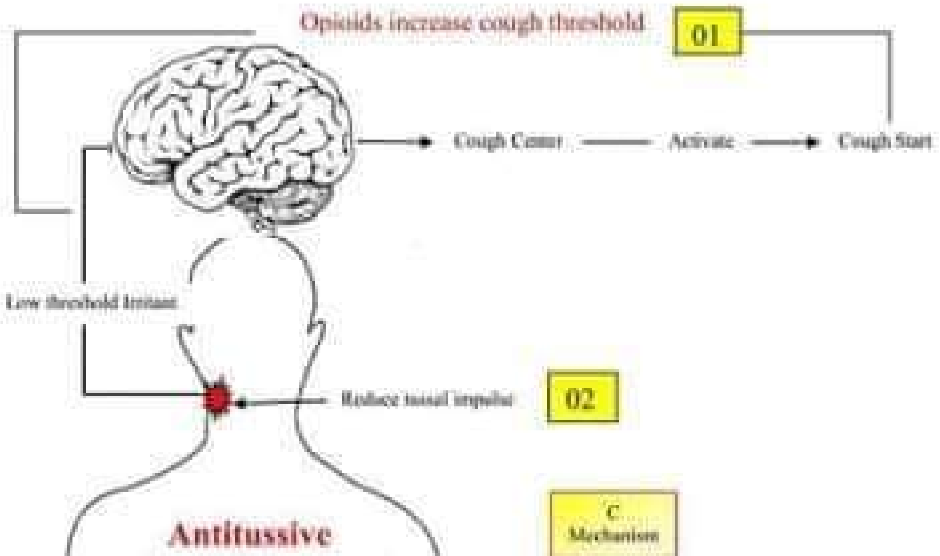
Expectorant or Mucokinetics are the drugs which produce their action either by increasing mucus production or by decreasing the viscosity of fluid so they become thin and easily expelled out.



C Mechanism

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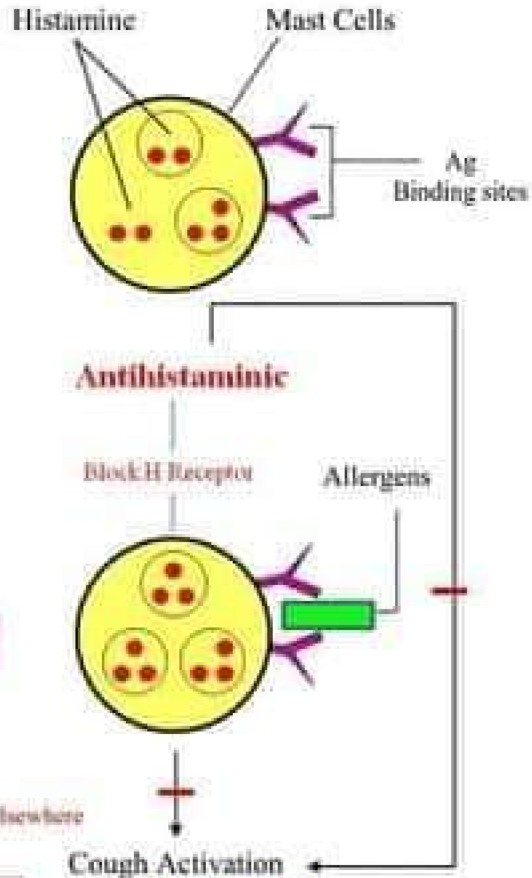
Antitussive are the agent or drug which generally increase the threshold of cough center, or reduce the tussal impulse or act by both mechanism.

Mechanism of Action



C Mechanism

* All Diagrams (Except Brain) and explanations are made by solution pharmacy

Note- All mechanisms of action are converted into diagram by Solution-Pharmacy. These are not available elsewhere

Many H_1 antihistaminic drugs showed their role as Antitussive agent. They produced antitussive action due to Their sedative and anticholinergic action, but lack selectivity for cough center.

* Diagrams Except brain) is made by solution to make you better understand
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SEDATIVE - HYPNOTICS

These drugs make exited patient calm and cool with and without causing sleep. They are differ only in concentration

Classification- KD Tripathi

SEDATIVE

Sedative are those drugs which make patient calm and relax without causing sleep, although patient may feel dizziness and may loss alertness or responsiveness.

D T F R A
दिमाग ठंडा रखने में फटाफट नींद आती है

L O D A C
लो डरावे आ गया क्लामिफिकेशन

C C D L
चलो चले दवाई लेने एंटी कनवलसन का

HYPNOTICS

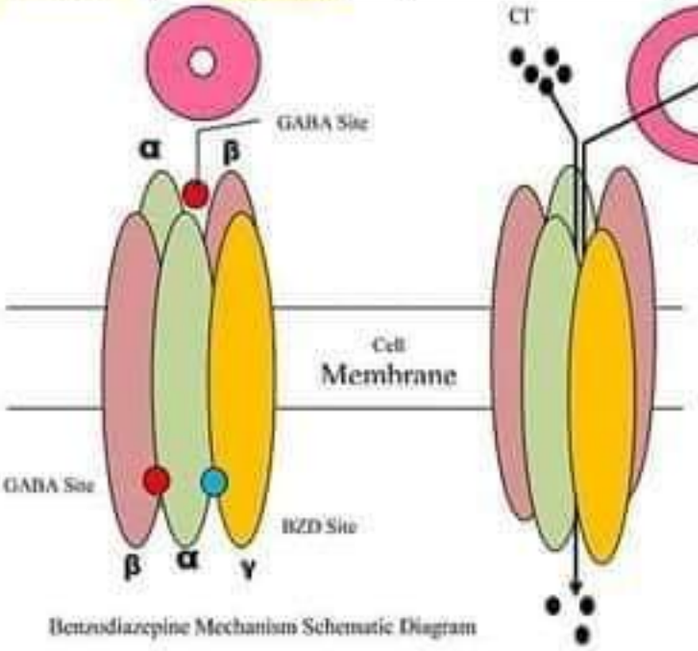
Hypnotics are the drugs which make person calm but also induce sleep. This is extension of sedative dose of any drugs. Sedative in large dose act as hypnotics

Barbiturate | **Benzodiazepine** | **Newer Non BZD Hypnotics** Zopiclone, Zolpidem, Zaleplon

Sedation – Sleep – Anaesthasia

- **Long Acting** Phenobarbitone
- **Short Acting** Butobarbitone, Phenobarbitone
- **Ultra Short Acting** Thiopentone, Methohexitone

Hypnotic	Antianxiety	Anticonvulsant
Diazepam	Lorazepam	Clonazepam
Temazepam	Oxazepam	Clobazam
Flurazepam	Diazepam	Diazepam
Nitrazepam	Alprazolam	Lorazepam
Alprazolam दिमाग ठंडा रखने में फटाफट नींद आती है	Chlordiazepoxide लो डरावे आ गया क्लामिफिकेशन	चलो चले दवाई लेने एंटी CONVULSION का



Benzodiazepine Mechanism Schematic Diagram

Different stages of Sleep pattern

Stage – 0
Awake
Constitute 1 – 2%

Stage – 01
Dozing
Constitute 3 – 6%

Stage – 02
Unequivocal Sleep
Constitute 40 – 50%

Stage – 03
Deep sleep transition
Constitute 5 – 8%

Stage – 04
Cerebral Sleep
Constitute 10 – 20%

REM sleep
Paradoxical Sleep
Constitute 20 – 30%

Mechanism of Action

Target of Benzodiazepine are on GABA receptor, because GABA is major inhibitory neurotransmitter in CNS. GABA is consisting of five- alpha, beta, gamma subunits that span the postsynaptic membrane. The influx of chloride Ions cause hyper polarization of the neuron and decrease neurotransmitter by inhibiting the formation of action potentials.

Empty receptor is inactive and coupled chloride channel is closed

→ Binding of GABA open the chloride channel cause hyper polarization

→ Binding of GABA is enhanced by benzodiazepine; result in more entry of negative chloride ions.

→ Entries of chloride hyper polarize cells, make them difficult to depolarize and reduce neural excitability

Mechanism of Action in various steps

Pharmacological Action

→ Reduction of Anxiety

→ Sedative-Hypnotics

→ Anterograde Amnesia

→ Muscle Relaxant

→ By Targeting GABA receptor

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Anti-Hypertensive Drugs

Prevention is always better than cure

Classification- KD Tripathi

1. Diuretics
2. ACE Inhibitor
3. Angiotensin Blocker
4. Calcium Channel Blocker
5. Beta Blocker
6. Beta+Alfa Blocker
7. Alfa Blocker
8. Central Sympatholytics
9. Vasodilator

Examples of above listed Class

- Thiazide- Hydrochlorothiazide, Chlorthiazide, Indapamide
  High Ceiling- Furosemide
  K+ Sparing- Spironolactone, Amiloride
- Captopril, Enalapril, Lisinopril, Perindopril, Ramipril, Fosinopril etc.
- Losartan, Candesartan, Irbesartan, Valsartan, Telmisartan
- Verapamil, Diltiazem, Nifedipin, Felodipine, Amlodipine, Lacidipine
- Propranolol, Metoprolol, Atenolol
- Labetalol, Carvedilol
- Prazocin, Terazocin, Doxazosin, Phentolamine, Phenoxybenzamine
- Clonidine, Methyldopa
- Arteriolar- Hydralazine, Minoxidil, Diazoxide
  Arteriolar+ Venous- Sodium Nitroprusside

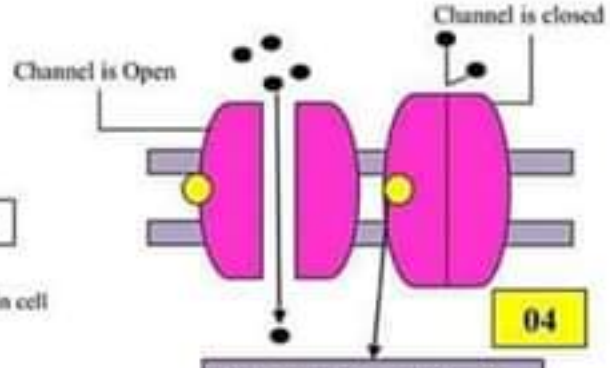
Thiazide Diuretics

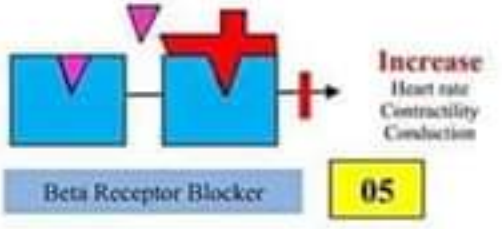
Increase Water & Sodium Excretion

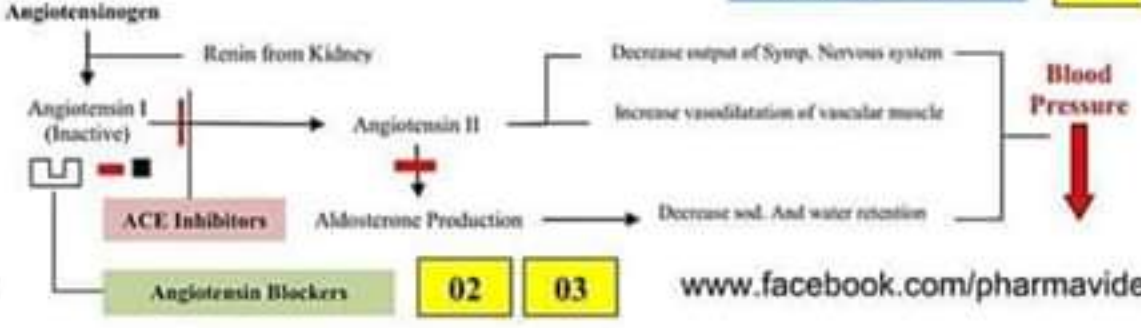
- **Short Term Effect**
  - **Decrease** Blood Volum
  - **Decrease** cardiac output
- **Long Term Effect**
  - Sodium content in cell
  - **Decrease** Muscles sensitivity to vasopressor
  - **Decrease** Peripheral Resistance

01

Decrease Blood Pressure







Antiparkinsonian Drugs

Classification- KD Tripathi

Drug affecting brain dopaminergic system

Dopamine Precursor
Levodopa

Peripheral decarboxylase inhibitor
Crbidopa, Benseraside

Dopaminergic Agonist
Bromocriptine, Ropinirole, Pramipexole

MAO- B Inhibitor
Selegiline

COMT Inhibitor
Entacapone, Tolcapone

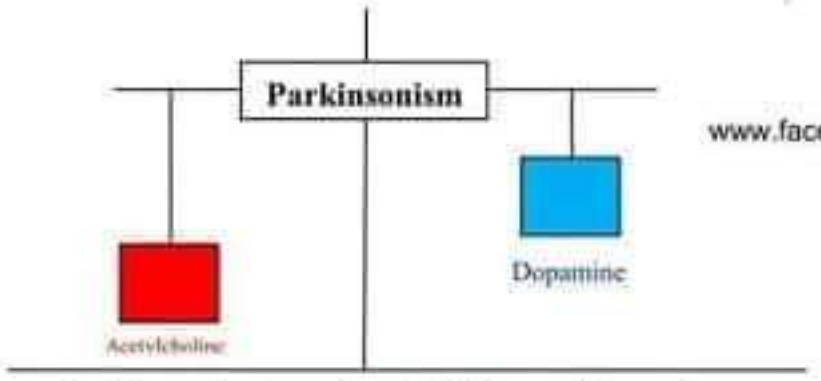
Dopamine Facilitators
Amantadine

Drug affecting brain cholinergic system

Central Anticholinergic
Trihexyphenidyl, Procyclidine, Biperiden

Antihistaminic
Orphenadrine, Promethazine

"ACLBSEBRPT" Acetylcholine Level Badalne Se Brain Rog Parkinson Taklif deta hai.
Acetylcholine लेवल बदलने से ब्रेन ब्रेन रोग पार्किन्सन तकलीफ देता है



Imbalance between Acetylcholine and Dopamine

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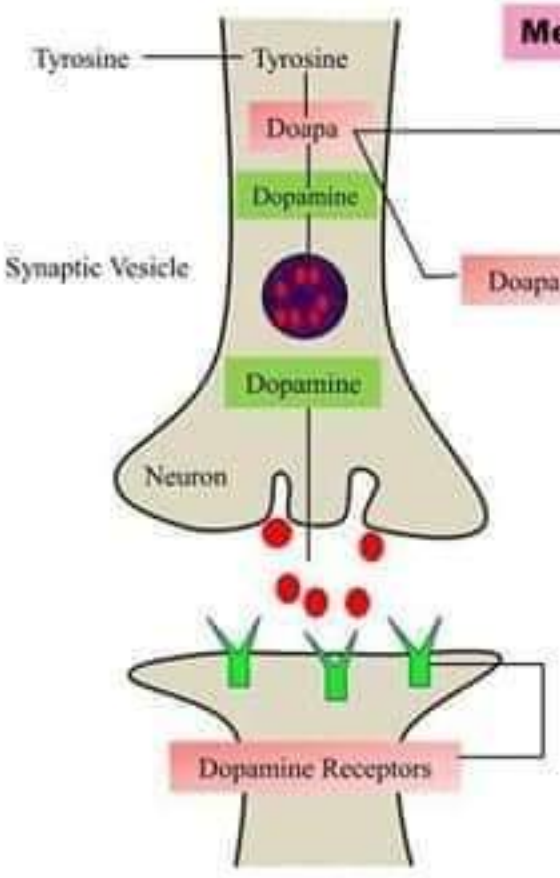
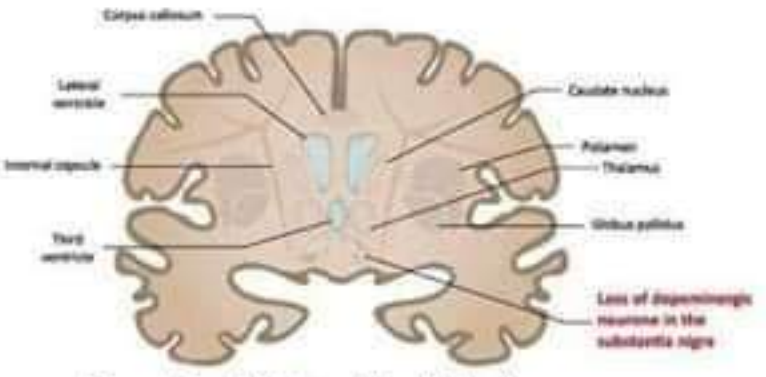
Mechanism of action of Levodopa



Parkinson's Disease Brain



* Diagram Except Brain is made by- Solution-Pharmacy

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Parkinsonism is an extra pyramidal motor disorder, symptoms includes- rigidity, tremors, with defective gesture and posture. Parkinsonism is a result of imbalance between acetylcholine and dopamine. When there is remarkably decrease in dopamine level or increase in acetylcholine level, Parkinsonism take place. As cause is clear their treatment is also very clear. Treatment goal is to restore the balance between above said neurotransmitters either by increasing dopamine by eternal source or by decreasing the level of acetylcholine.

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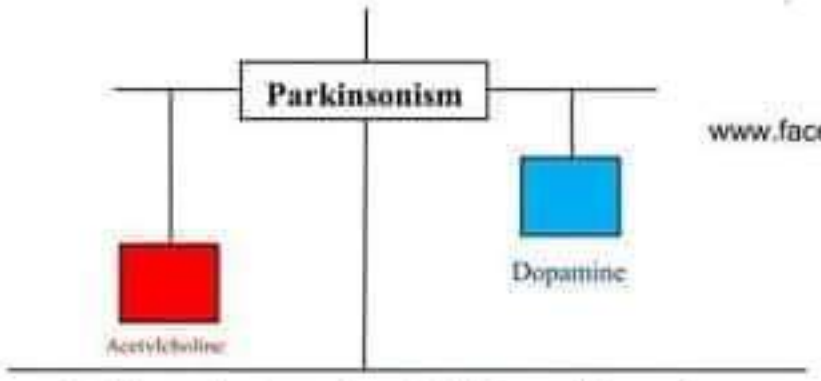

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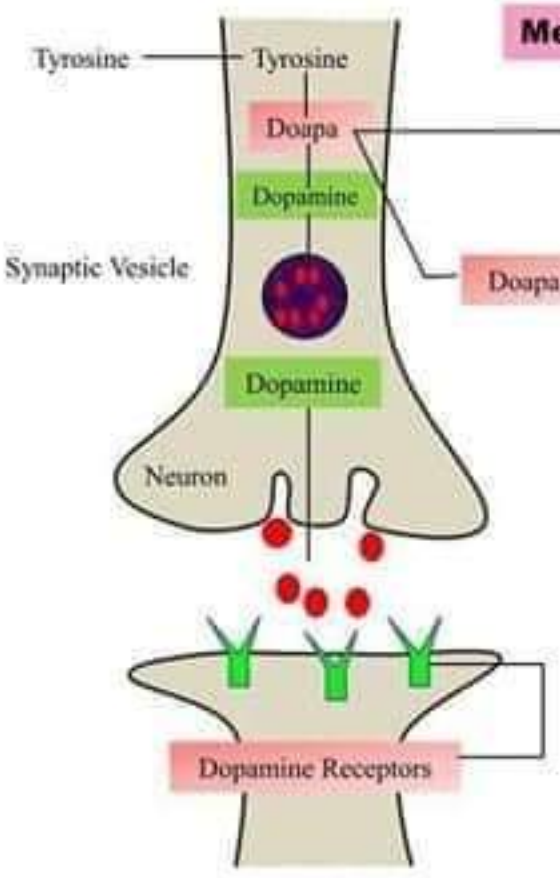

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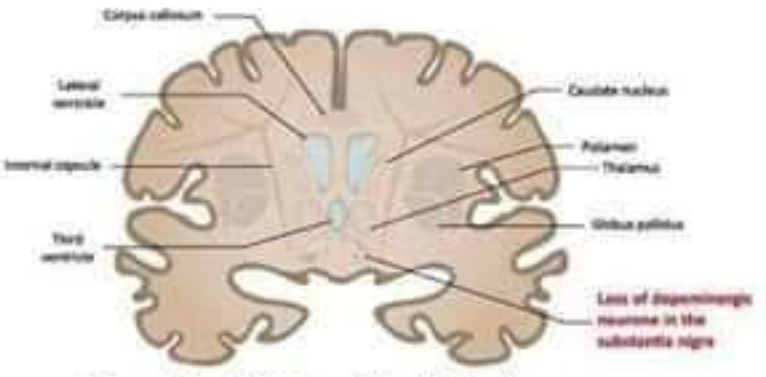

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Adverse Drug Effects

Introduction-Classification-Categorization

Concept & Idea by- Solution Pharmacy

Drug/ Medicine

Drug is a chemical moiety (either natural or synthetic) which is used for the prevention, diagnosis and treatment of any disease or disorder. Disease is caused by microorganism mainly and disorders And Disorders are result of imbalance of various biochemicals within body itself.

Adverse Effect

Any effect produced by the drug which is not expected or not desired and unintended is called adverse drug effects. It is one of the broad definitions which cover many subtypes from simple to serious effects.

Disease
Disorder

Diagnosis

Treatment

Drug/Medicine

Desirable Effects

Desirable effect means all those effect produced by drug which we are expecting and willing to get.

Example

Paracetamol, Nimesulide, Ibuprofen, Aspirin, Indomethacin Aspirin

NSAIDS
Non Steroidal Anti-inflammatory Drugs

Analgesic Effect

Reduce pain by inhibiting PG synthesis and increasing pain threshold potential.

Antipyretic Effect

Reduce fever by controlling thermoregulatory center in brain

Anti-Inflammatory Effects

Reduce inflammation by inhibiting COX-I and COX-

Undesirable Effects

All those effect given by drug which is not good for us and it may cause simple to dangerous effect.

Different Categories

Predicted
Type A or Augmented

Unpredicted
Type B or Bizarre

Side Effects
Rashes, Etching

Secondary Effects
Suspension of bacterial flora

Toxic Effects- Poisoning

Intolerance

Drug Allergy- Humoral & Cell Mediated

Photosensitivity- Phototoxic and Photo allergic

Drug Dependence
Physiological-Physical-Drug Abuse-Drug Addiction-Drug Habituation

Drug Withdrawal reaction- Alcohol and LSD

Teratogenicity- Thalidomide

Mutagenisity or Carcinogenicity

Drug Induced Disease- Peptic Ulcer by NSAIDS

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DRUG USED FOR PEPTIC ULCER

Based on KD Tripathi Classification

Reduction of Gastric acid Secretion

H_2 Antihistaminic
Cimetidine, Ranitidine, Famotidine, Roxatidine

Proton Pump Inhibitor
Omeprazole, Esomeprazole, Lansoprazole, Pantoprazole, Rabeprazole, Dexrabeprazole

Anticholinergic Drugs
Pirenzepine, Propantheline, Oxyphenonium

Prostaglandin Analogue
Misoprostol

Neutralization of Gastric acid

Systemic
Sodium bicarbonate, Sodium citrate

No systemic
MgOH, Magnesium trisilicate, Aluminum hydroxide gel, Magaldrate, Calcium carbonate

$NaHCO_3 + HCL \rightarrow NaCl + H_2O + CO_2$

Base

Acid + Base

Neutralization

Ulcer Protective

Example
Sucralfate, Colloidal bismuth sub citrate

Ulcer

Coating

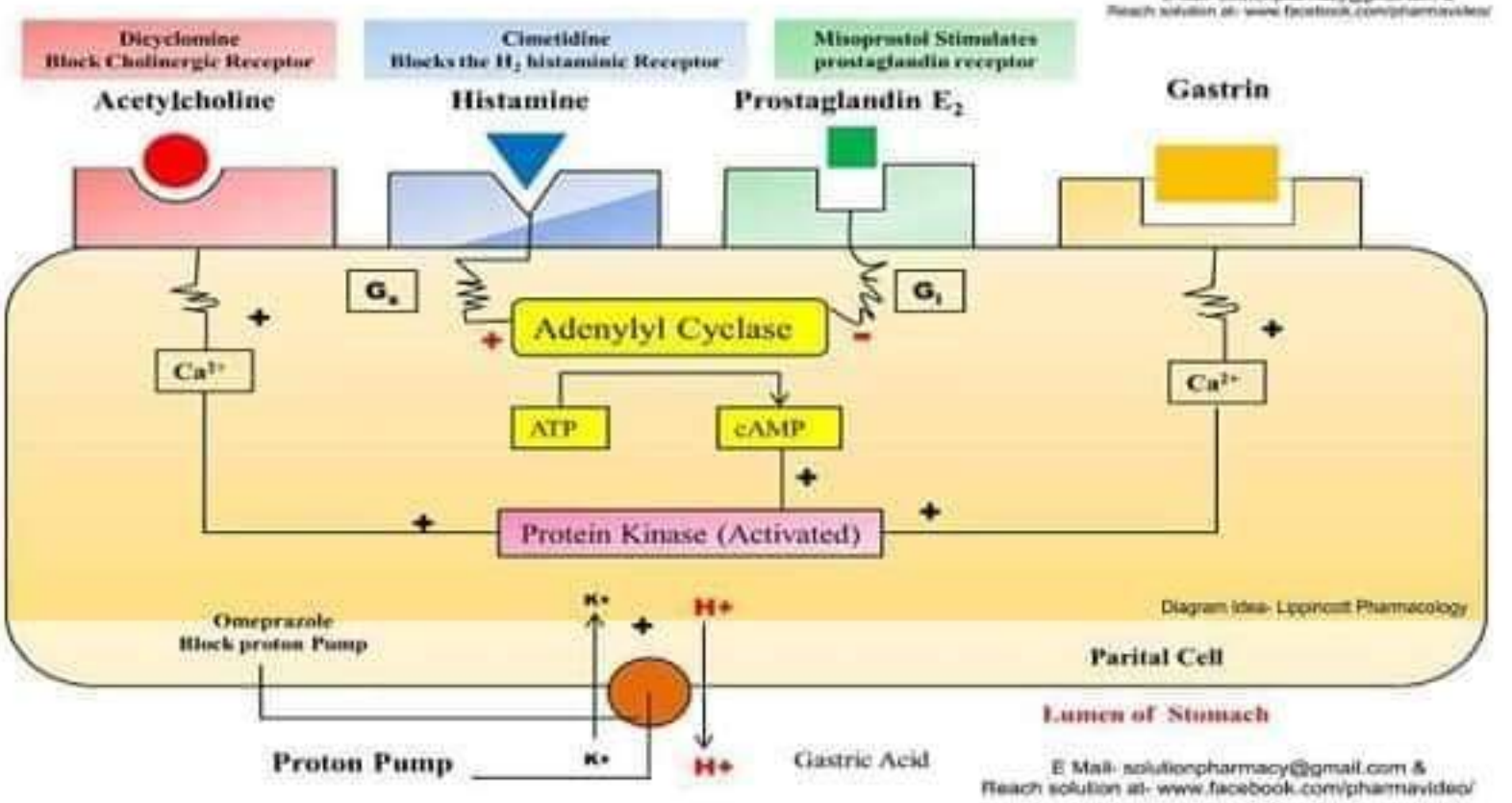
Anti H Pylori Drugs

Example
Amoxicillin, Clarithromycin Metronidazole, Tinidazole Tetracycline

Antibiotics

Pylori

Anti Pylori



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Key point- Peptic ulcer is result of imbalance between attacking gastric acid and protective bicarbonate system. Gastric acid secretion is regulated by cholinergic system, Histaminic system, stress, Hyperacidity, Microorganism and somehow smoking and spicy diet. The first attempt towards treatment is neutralization of hyper acidity by using antacids which are chemically base and they give their action by neutralizing acid. Protective drug are not the treatment they can mask the pain or irritation signal arising from the ulcer. Anti microbial drug may only be effective in case of infection.

Drugs for Constipation

Classification based on KD Tripathi (Pharmacology)

Bulk Forming Agents

Example

- Bran
- Psyllium (Plantago)
- Ispaghula
- Methyl Cellulose

Mechanism of Action

Stool Softener

Example

- Docusates (DOSS)
- Liquid Paraffin

Mechanism of Action

Stimulant Purgative

Diphenylmethanes
Phenolphthalein, Bisacodyl
Sodium picosulfate

Antraquinones
Senna, Cascara Sagrada

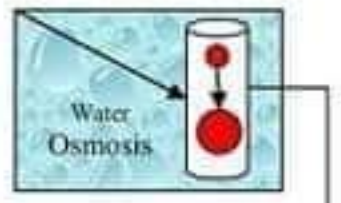
5HT$_4$ Agonist
Tegaseroid

Fixed Oil
Castor Oil

Osmotic Purgative

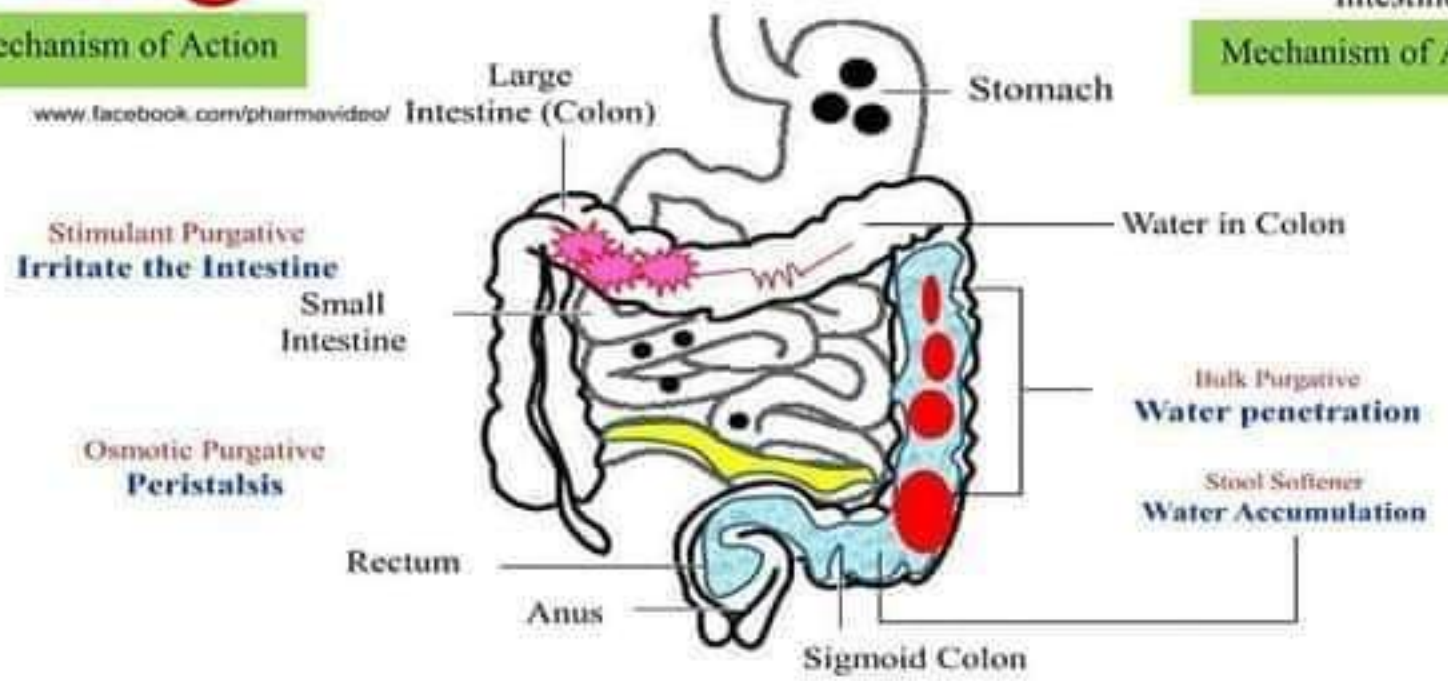
Magnesium Salt
Sulfate, Hydroxide

Sodium Salt
Sulfate, Phosphate, Sodium-Potassium tartrate, Lactose



Intestine

Mechanism of Action



*** Mechanism of Action- Representation by Solution Pharmacy**

Key point- Constipation is not a single disease or disorder; it is a root cause for several GIT related problems. When there is lack of water in large intestine and lack of fiber intake in diet, constipation take place. Constipation's treatment lies within the problem itself. Target for the treatment include-

1. Increase amount of water inside intestine
2. Increase fibers content in diet
3. Increase expel of stools by increasing peristaltic movement
4. Stimulating or irritating the colon to force the evacuation of stools
5. Increase retention of sufficient water inside intestine

*Based on our concept. Reference not available

Anti Microbial Drugs
Classification According to Mechanism of Action
Classification Reference- KD Tripathi (Pharmacology)

Inhibit Cell wall synthesis
- Penicillin
- Cephalosporins
- Cycloserine
- Bacitracin
- Vancomycin

Cause Leakage From cell membrane
- Polymyxines
- Colistin
- Nystatin
- Bacitracin polymers
- Amphotericin- B
- Hamycin

Inhibit Protein Synthesis
- Tetracycline
- Cloramphenicol
- Erythromycin
- Clindamycin
- Linezolis

Cause misreading or RNA and affect permeabilits
- Aminoglycosides
- Streptomycin
- Gentamycin

Inhibit DNA gyrase
- Fluoroquinolones
- Ciprofloxacin etc

Interfere with DNA function
- Rifampin
- Metronidazole

Interfere with DNA synthesis
- Acyclovir
- Zidovudine

Interfere with Intermediary Metabolism
- Sulfonamides, PAS, Sulfones, Trimethoprim, Ethambutol,

Inhibit Cell wall synthesis	PCCBV- [illegible]
Cause Leakage From cell membrane	PCNABH- [illegible]
Inhibit Protein Synthesis	TCECL- [illegible]
Cause misreading or RNA and affect permeability	SG- [illegible]
Inhibit DNA gyrase	C- [illegible]
Interfere with DNA function	RM- [illegible]
Interfere with DNA synthesis	A to Z [illegible]
Interfere with Intermediary Metabolism	SPSTE- [illegible]

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Note- Mnemonics are based on my thoughts; it may or may not useful to you. It's always better to create your own so you may memorize it.

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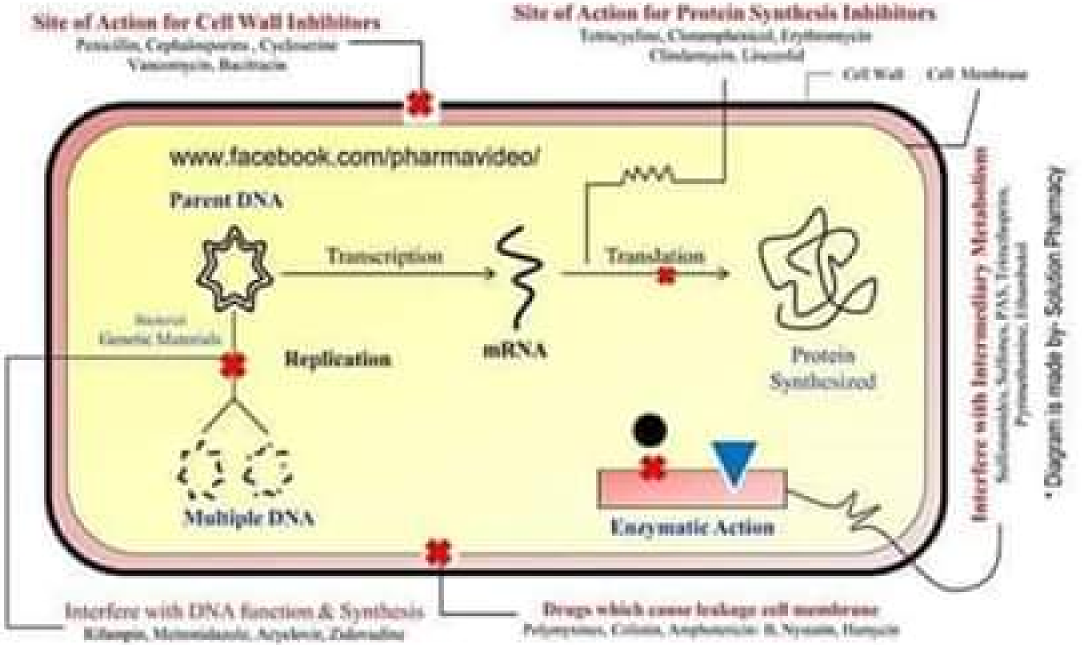

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Anti Microbial Drugs
Classification According to Various Parameters
Classification Reference- KD Tripathi (Pharmacology)

Types of Organism Against which primarily Active

Antibacterial
Penicillins, Aminoglycosides
Erythromycin etc

Antifungal
Griseofulvin, Amphotericin B,
Ketoconazole etc

Antiviral
Acyclovir, Amantadine,
Zidovudine etc

Antiprotozoal
Chloroquine, Pyrimethamine,
Metronidazole, Diloxanide etc

Anthalmentic
Mebendazole, Pyrantel,
Niclosamide, Diethyl

Spectrum of Activity

Narrow Spectrum
Penicillin G
Streptomycin
Erythromycin

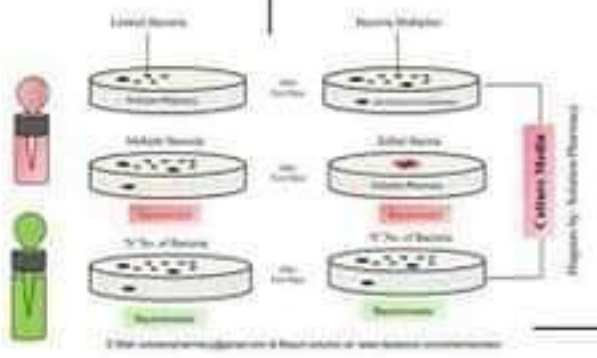
Broad Spectrum
Tetracycline
Chloramphenicol

Type of Activity

Bacteriostatic
Sulfonamide
Tetracycline
Chloramphenicol
Erythromycin
Ethambutol
Linezolid

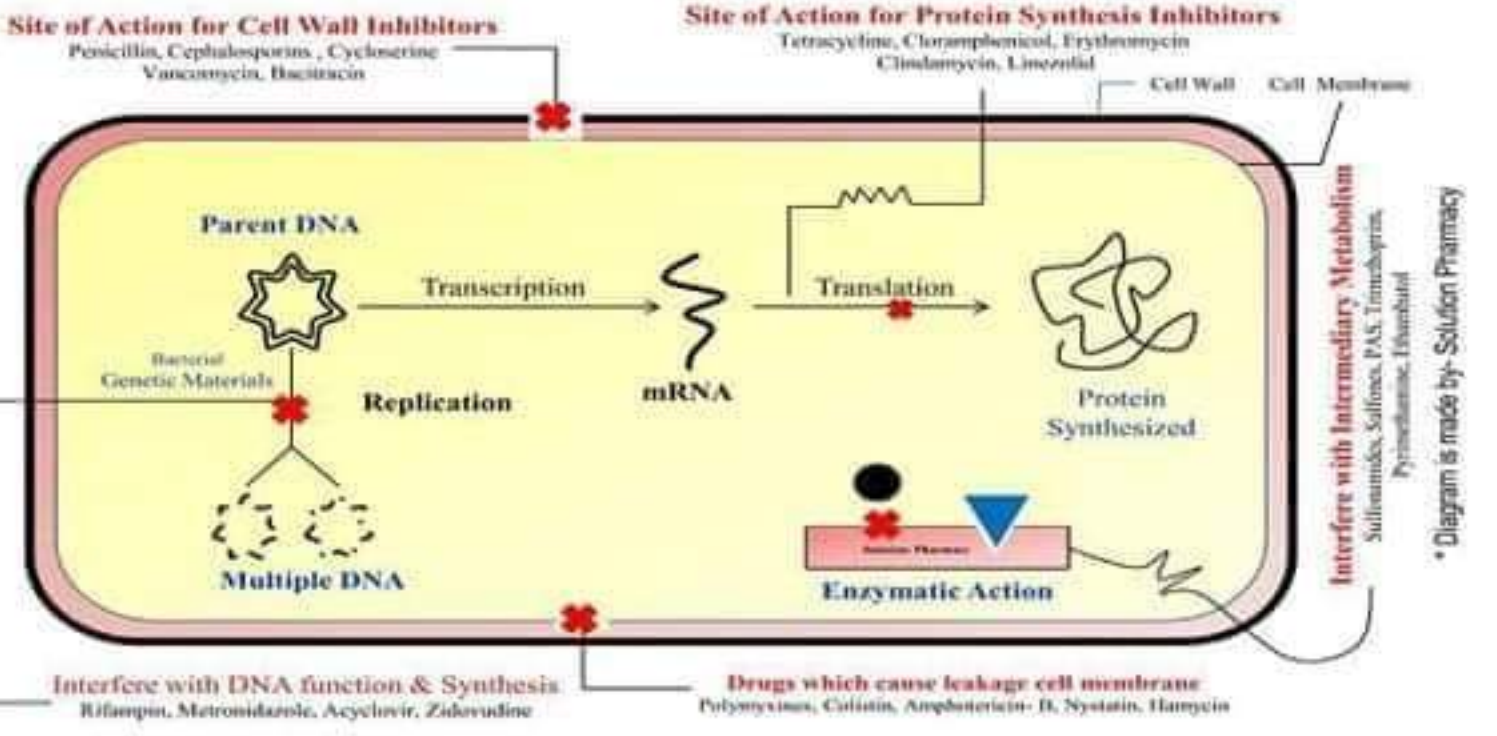
Bacteriosidal
Penicillin
Aminoglycosides
Polypeptides
Rifampin
Isoniazide
Pyrazinamide
Cephalosporin
Vancomycin
Nalidixic Acid
Ciprofloxacin
Metronidazole
Cotrimoxazole

Diagram is made by Solution-Pharmacy



Antibiotic according their sources

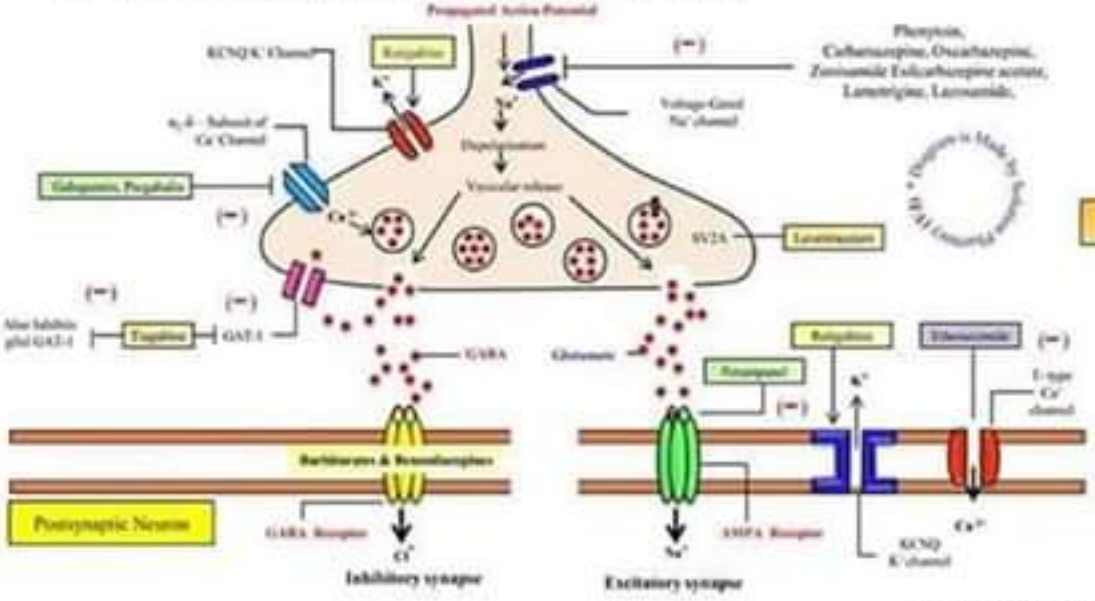
Sources	Example of Antibiotic
Fungi	Penicillin, Griseofulvin, Cephalosporin
Bacteria	Polimyxin B, Tyrothricin, Colistin, Azatreonam, Bacitracin
Actinomycetes	Aminoglycosides, Macrolides, Tetracyclines, Polyenes, Chloramphenicol



Drug Used in Treatment of Epilepsy

Classification and Mechanism of Action (Based on KD Tripathi)

- **Barbiturate**
  - Phenobarbitone
- **Deoxybarbiturate**
  - Primidone
- **Hydantoin**
  - Phenytoin
    - Fosphenytoin
- **Iminostilbene**
  - Carbamazepine
    - Oxacarbazepine
- **Succinimide**
  - Ethosuximide
- **Aliphatic Carboxylic Acid**
  - Valproic Acids
    - Divaproex
- **Benzodiazepines**
  - Clonazepam
    - Diazepam
      - Lorazepam
        - Clobazam
- **Phenyltriazine**
  - Lamotrigine
- **Newer Drugs**
  - Topiramate
    - Zonisamide
      - Levetiracetam
        - Vigabatrin
          - Tigabine
            - Lacosamid
- **Cyclic GABA Analog**
  - Gabapentin
    - Pregabalin



Types of Seizures

- **Generalized Seizers**
  - Generalized Tonic-clonic Seizers
    - Absence Seizers
      - Atonic Seizers
        - Myoclonic Seizers
          - Infantile Spasms
- **Partial Seizers**
  - Simple PARTIAL SEIZURES
    - Complex PARTIAL SEIZURES
      - Complex or Simple PARTIAL SEIZURES

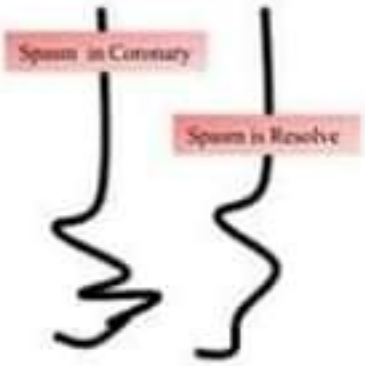
Antianginal Drugs

Classification & Mechanism of Action

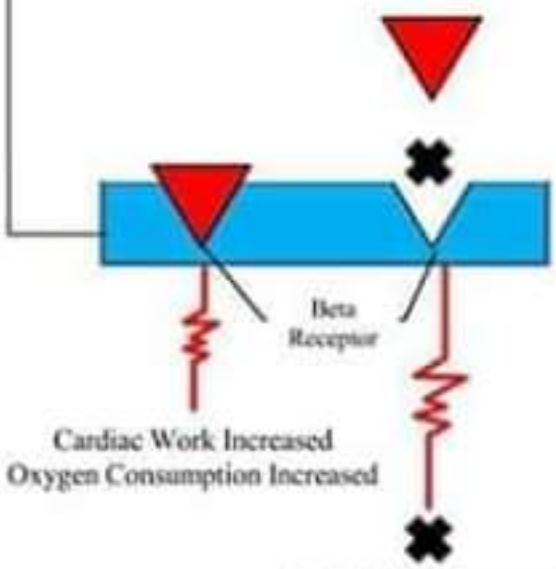
Based on KD Tripathi

Angina is a chest pain or we can say that it is a signal informing us that there is lack of oxygen supply to myocardium. This is generally occurring at the left side of the chest. It has 02 main types- (1) Classical Angina (Common form) - Those type of angina which may arise due to over work like- exercise, emotion etc. (2) Variant or prinzmetal's Angina (uncommon form) - Attack occurs at rest or during sleep and doesn't disappear after rest.

- **Nitrates**
  - **Short Acting**: Glyceryl Trinitrate (GTN, Nitro Glycerin)
  - **Long Acting**: Isosorbide dinitrate, Isosorbide mononitrate, Erythrityl tetranitrate, Pentaerythritol tetranitrate

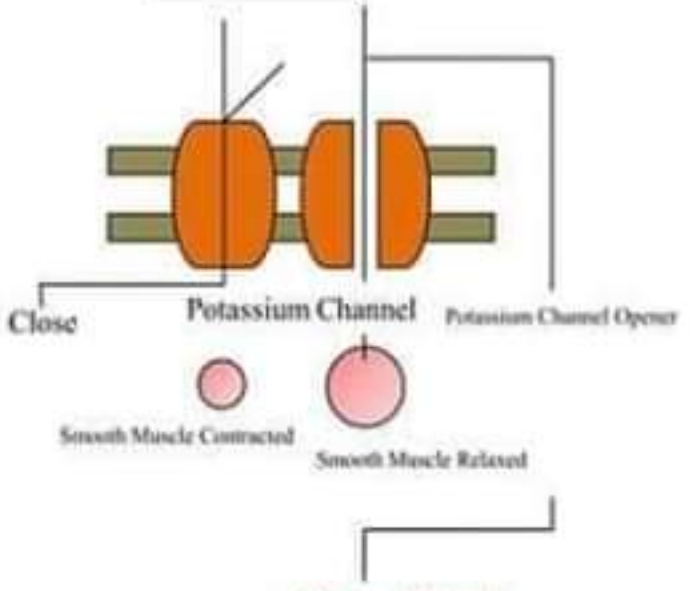


- **Beta Blocker**: Propranolol, Metoprolol, Atenolol, and others



- **Ca^{+} Channel Blocker**: Phenyl alkylamine- Verapamil, Benzothiazepine- Diltiazem, Dihydropyridines- Nifedipin, Felodipine, Amlodipine, Nitrendipine, Nimodipine, Lacidipine, Lercanidipine, Benidipine

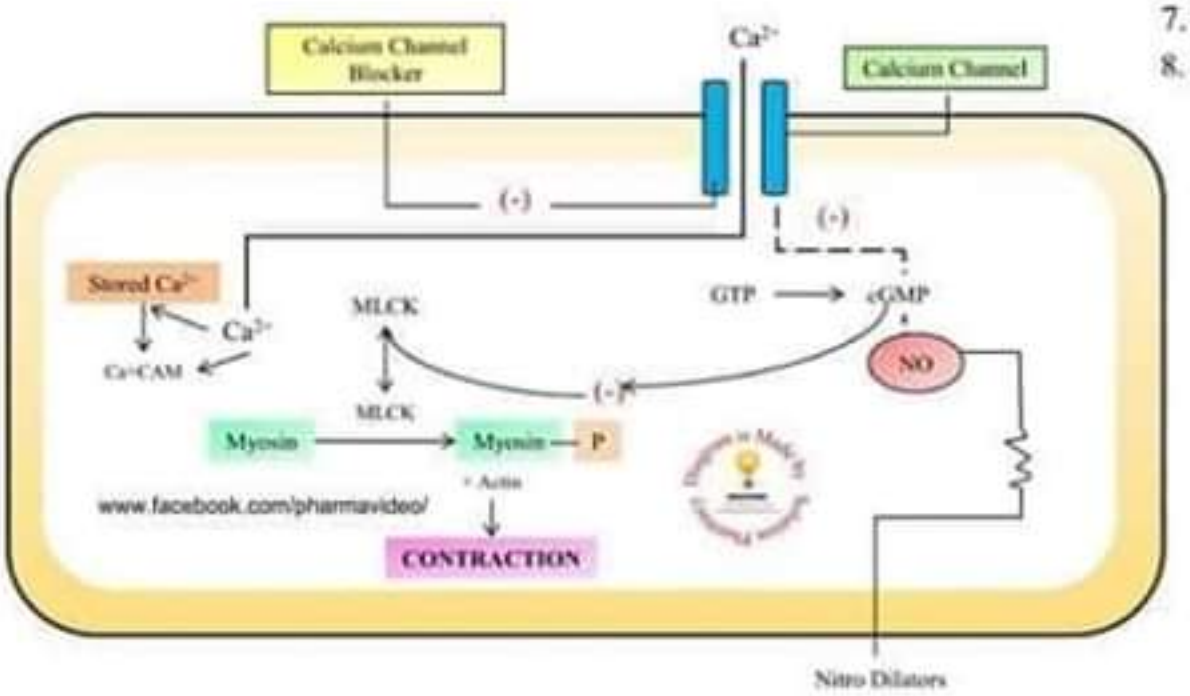
- **K^{+} Channel Opener**: Nicorandil



- **Others**: Dipyridamole, Trimetazidine, Ranolazine, Oxyphedrine

Other Clinical Application of K^{+} Channel Opener

1. Angina Pectoris
2. Hypertension
3. Congestive heart failure
4. Myocardial salvage in MI
5. Alopecia
6. Bronchial asthma
7. Urinary urge incontinence
8. Premature labour (Ref- KD Tripathi)



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Immunity

Immunology

Immunology is made of Immune + logy = Immunology. That means the complete study of Immune (Our body's bodyguard) system which majorly include antigen and antibody and their interaction, resulting in desirable or undesirable biological action. Reference- NK Jain (Microbiology)

- Natural Immunity
  - Species
  - Racial
  - Individual
- Acquired Immunity
  - Active
    - Natural/Clinical Subclinical
    - Artificial — Vaccine- Dead or Extract attenuated Toxoids
  - Passive
    - Natural — Congenital Colostrums
    - Artificial — Antiserum Antitoxin

Species

Man is susceptible for plague but not fowls. Mice are not affected by typhoid fever but man suffers.

Race

Negroes possess high resistance to yellow fever than white man. Negroes and white Indian are more susceptible to TB than Caucasian race.

Individuals

Children of age-2-5 are susceptible to diphtheria whereas most adult are immune to it.

Natural

It acquires when a person recover from- diphtheria, small pox and poliomyelitis. A person become immune because it's his antibody producing cell has received an adequate stimulus.

Artificial

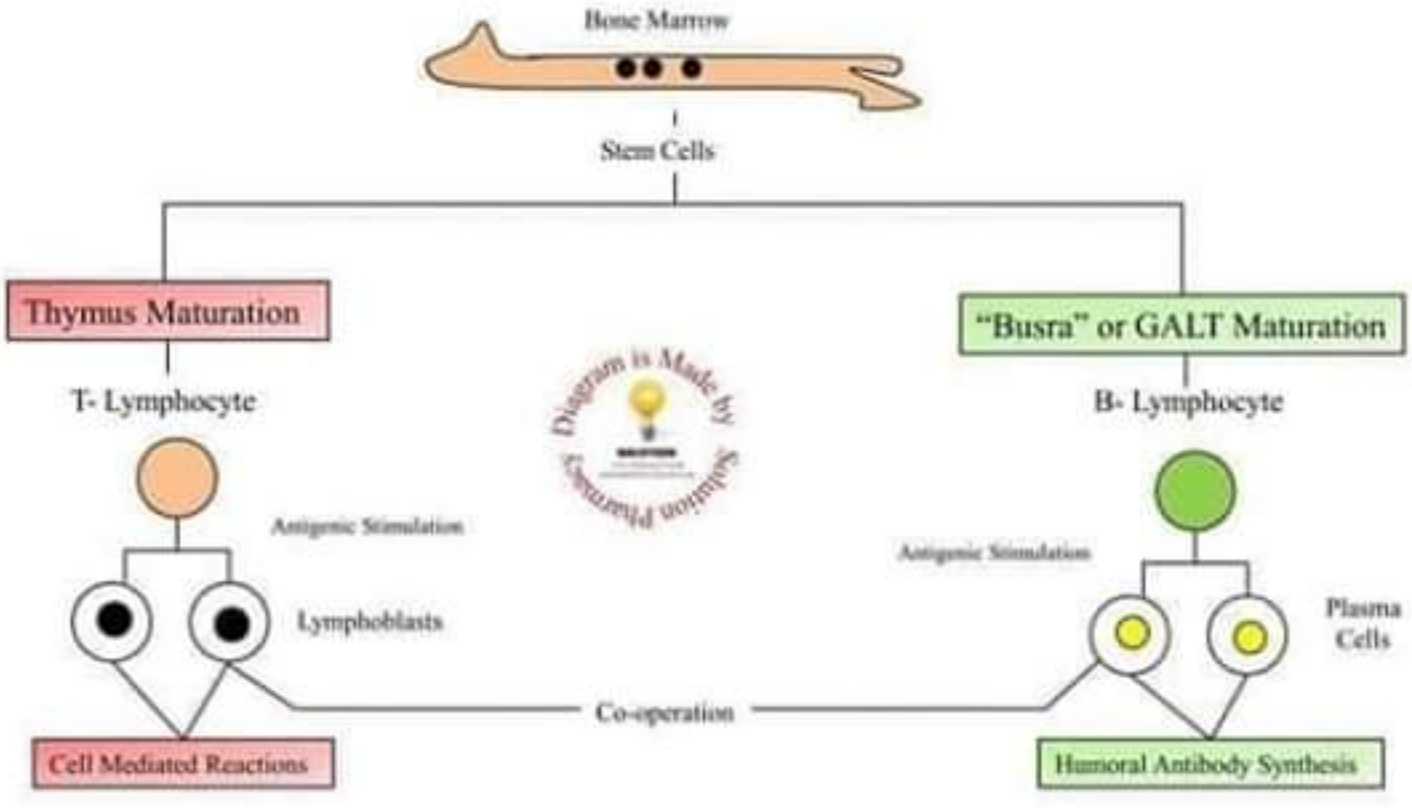
This is acquired by the administration of antigen usually by injection. Example- Vaccine either live or dead.

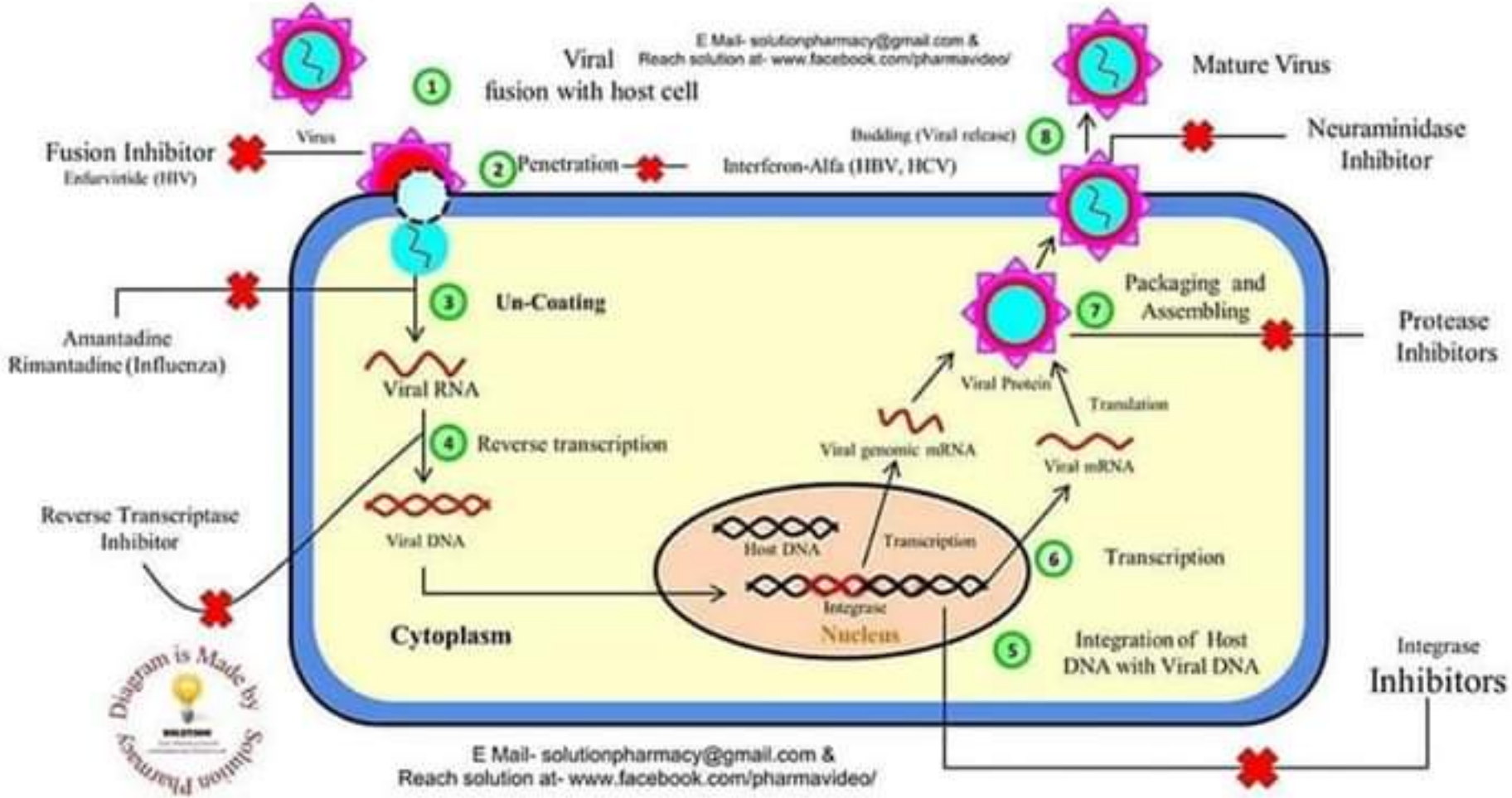
Natural Acquired Immunity

Antibody formed in a mother in response to disease may be transferred to fetus through the placental blood.

Artificially Acquired Immunity

This is acquired by injecting the preparation known s antiserum. These are the Antibodies produced in animal.

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Viral
1
fusion with host cell
Virus
Fusion Inhibitor
Enfurvirtide (HBV)
2
Penetration
Interferon-Alfa (HBV, HCV)
Budding (Viral release)
8
Mature Virus
Neuraminidase Inhibitor
3
Un-Coating
Amantadine
Rimantadine (Influenza)
Viral RNA
4
Reverse transcription
Reverse Transcriptase Inhibitor
Viral DNA
Cytoplasm
Host DNA
Transcription
Integrase
Nucleus
5
Integration of Host DNA with Viral DNA
6
Transcription
Viral genomic mRNA
Viral mRNA
Translation
Viral Protein
7
Packaging and Assembling
Protease Inhibitors
Integrase
Inhibitors
Diagram is Made by Solution Pharmacy
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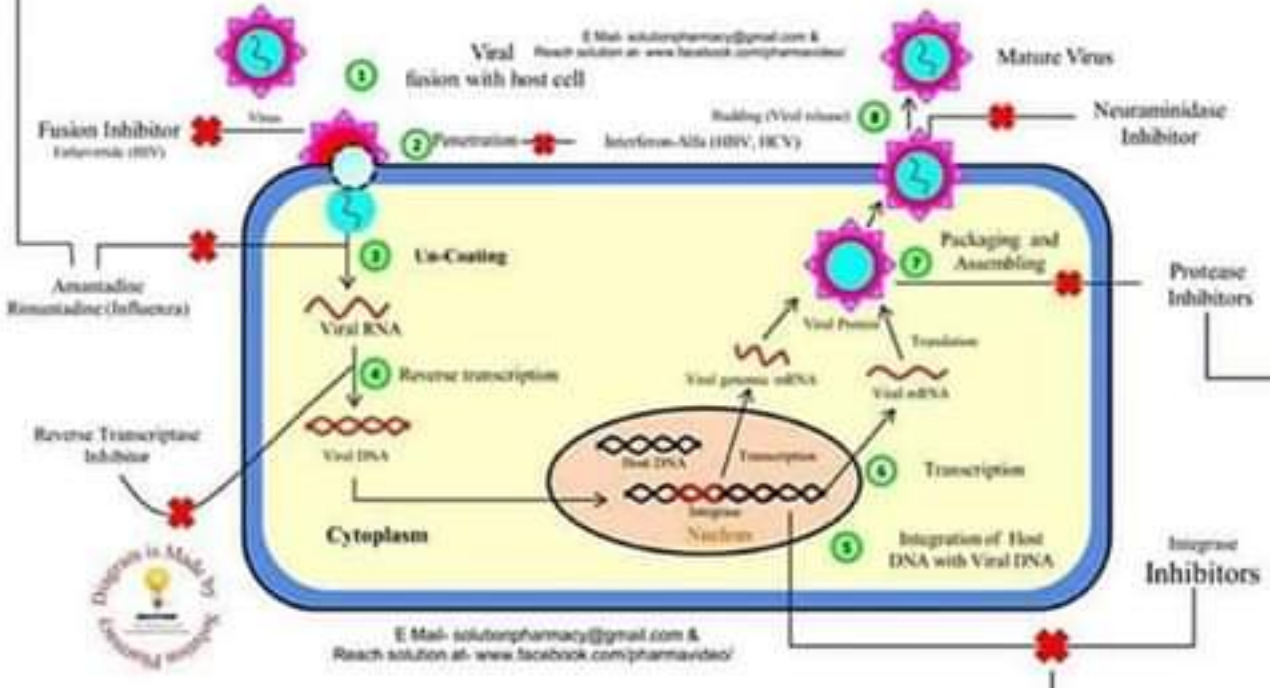
ANTIVIRAL DRUG
Classification and Mechanism of Action

Anti-Herpes Virus
Idoxuridine, Trifluridine, Acyclovir, Valacyclovir, Famciclovir, Ganciclovir, Valganciclovir, Cidofovir, Foscarnet,, Fomivirsen

Anti-Influenza Virus
Amantadine, Rimantadine, Oseltamivir, Zanamavir

Anti-Hepatitis virus or Nonselective antiviral drugs
Primarily for Hepatitis B
Lamivudine, Adefovir, Dipivoxil, Tenofovir
Primarily for Hepatitis C, Ribavirin, Interferon Alpha

Anti-Retrovirus



Nucleoside reverse transcriptase Inhibitors (NRTIs)
Zidovudine (AZT), Didanosine, Stavudine, Lamivudine, Abacavir, Emtricitabine, Tenofovir, (Nt RTI)

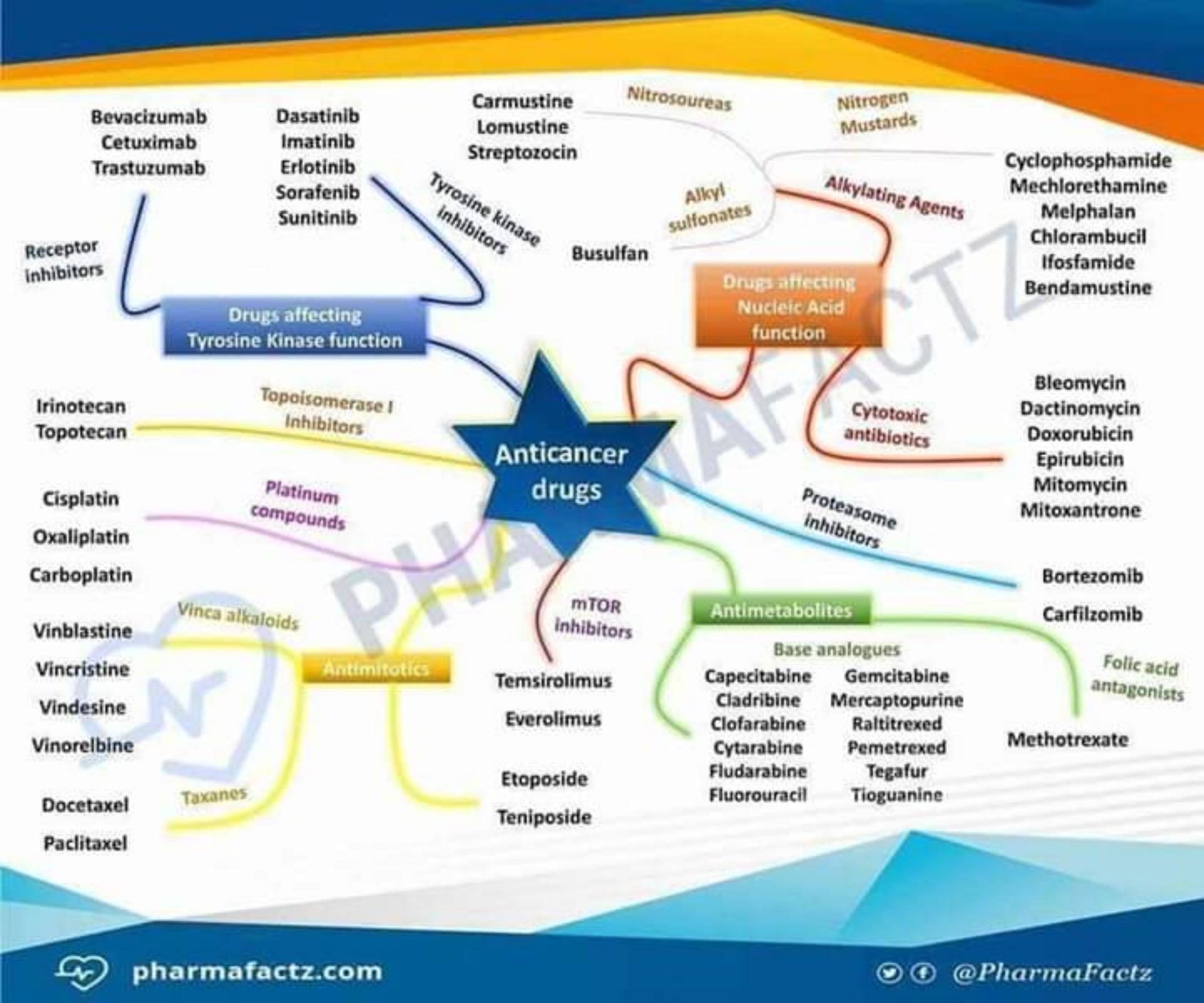
Non Nucleoside Reverse transcriptase Inhibitors (NNRTIs)

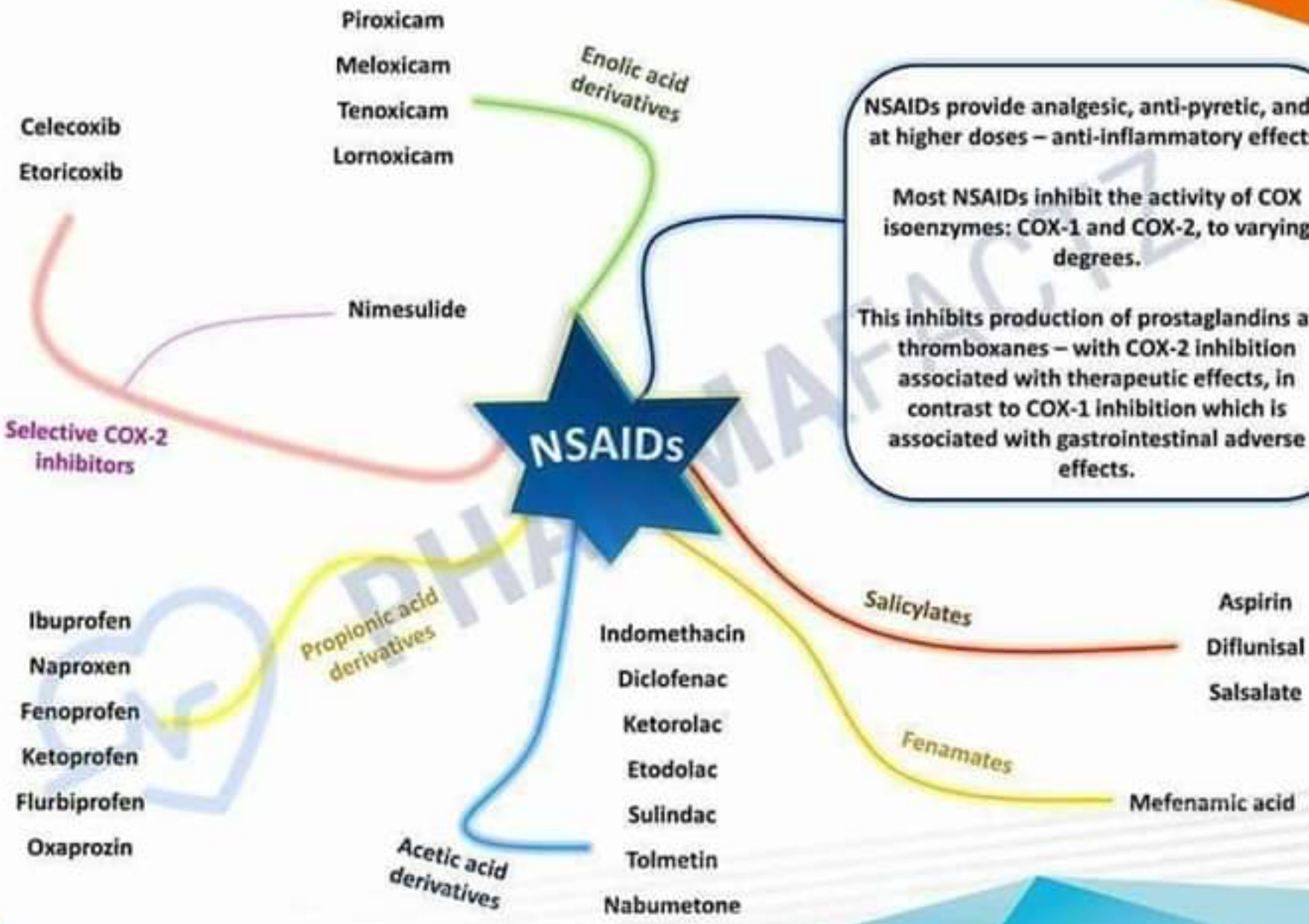
Protease Inhibitors
Ritonavir
Atazanavir, Indinavir, Nelfinavir, Saquinavir, Amprenavir, Lopinavir

Entry (Fusion) Inhibitor
Enfuvirtide

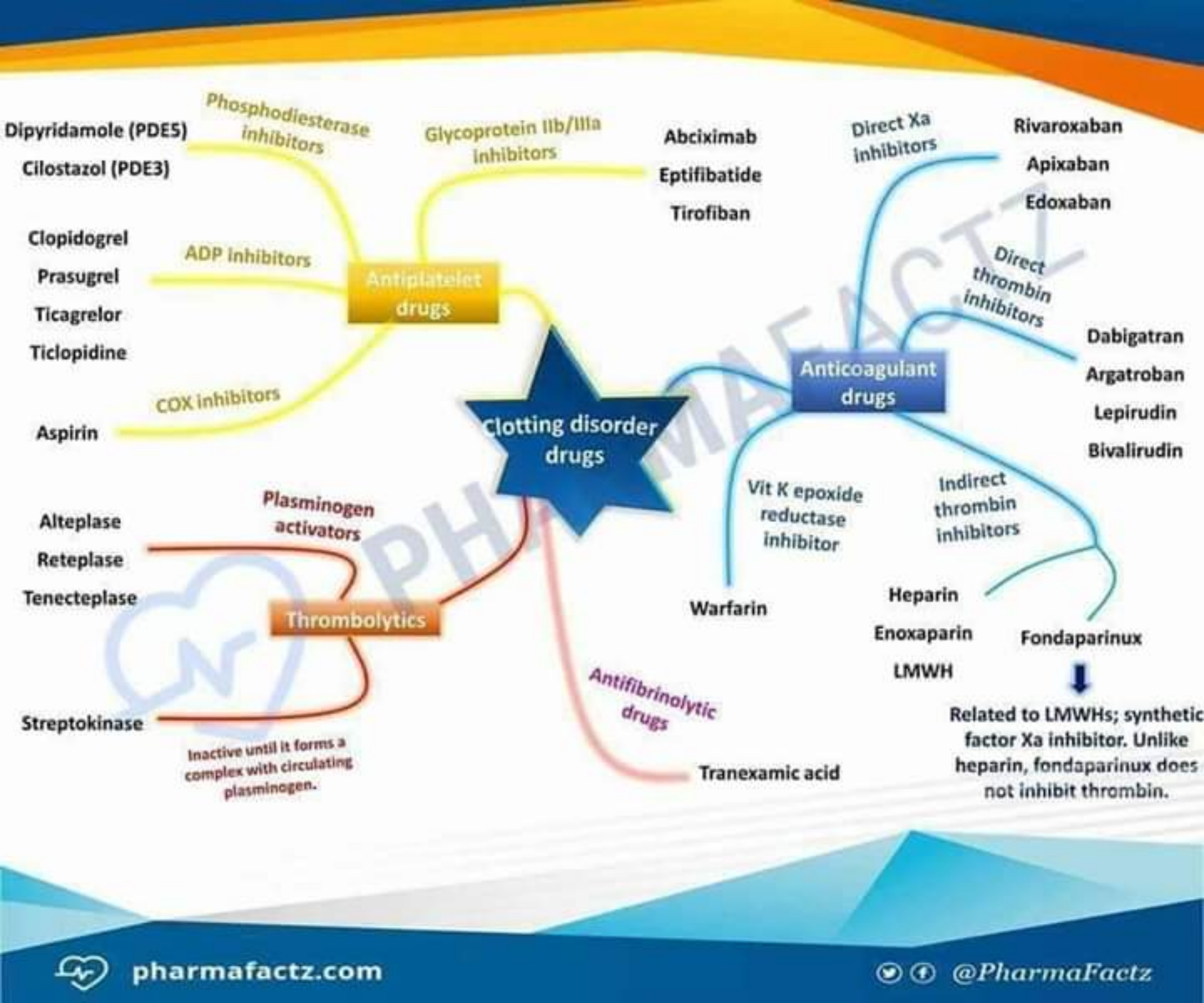
CCR5 Receptor Inhibitor
Maraviroc

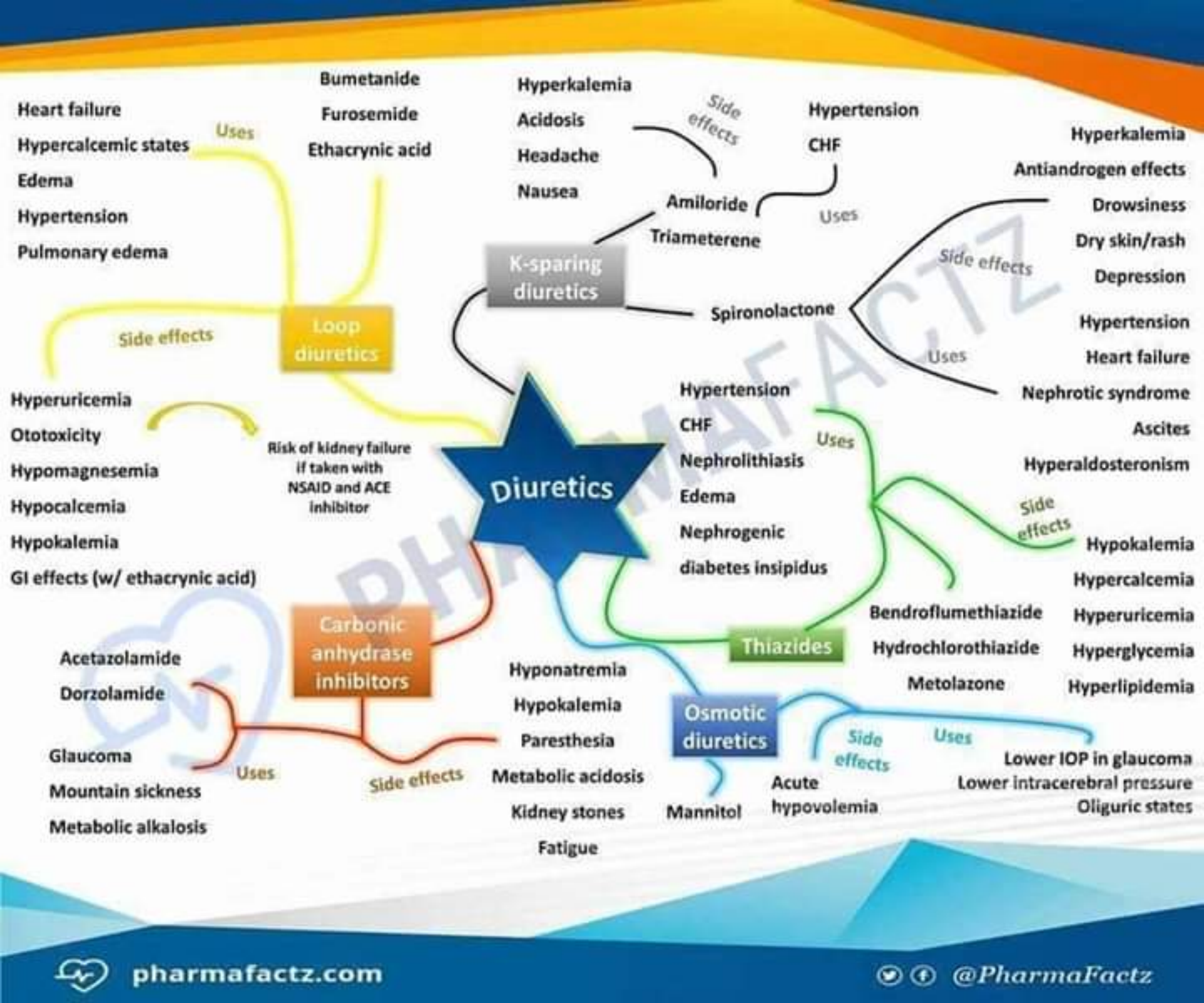
Integrase Inhibitor- Raltegravir

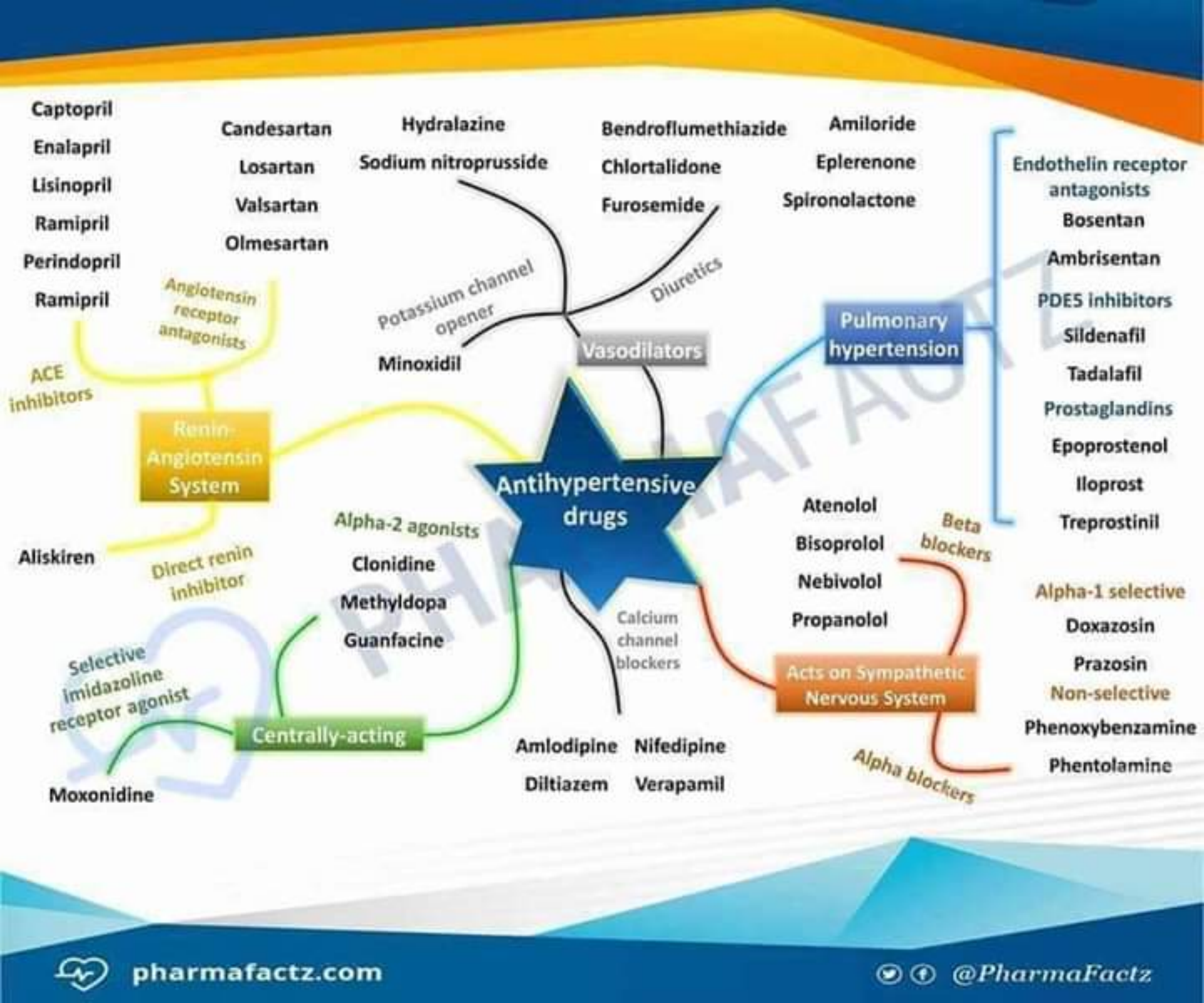

Anticancer drugs
Drugs affecting Tyrosine Kinase function
Receptor inhibitors
Bevacizumab
Cetuximab
Trastuzumab
Tyrosine kinase inhibitors
Dasatinib
Imatinib
Erlotinib
Sorafenib
Sunitinib
Drugs affecting Nucleic Acid function
Alkylating Agents
Nitrosoureas
Carmustine
Lomustine
Streptozocin
Alkyl sulfonates
Busulfan
Nitrogen Mustards
Cyclophosphamide
Mechlorethamine
Melphalan
Chlorambucil
Ifosfamide
Bendamustine
Cytotoxic antibiotics
Bleomycin
Dactinomycin
Doxorubicin
Epirubicin
Mitomycin
Mitoxantrone
Proteasome inhibitors
Bortezomib
Carfilzomib
Antimetabolites
Base analogues
Capecitabine
Cladribine
Clofarabine
Cytarabine
Fludarabine
Fluorouracil
Gemcitabine
Mercaptopurine
Raltitrexed
Pemetrexed
Tegafur
Tioguanine
Folic acid antagonists
Methotrexate
mTOR inhibitors
Temsirolimus
Everolimus
Antimitotics
Vinca alkaloids
Vinblastine
Vincristine
Vindesine
Vinorelbine
Taxanes
Docetaxel
Paclitaxel
Etoposide
Teniposide
Topoisomerase I Inhibitors
Irinotecan
Topotecan
Platinum compounds
Cisplatin
Oxaliplatin
Carboplatin


NSAIDs
NSAIDs provide analgesic, anti-pyretic, and – at higher doses – anti-inflammatory effects.
Most NSAIDs inhibit the activity of COX isoenzymes: COX-1 and COX-2, to varying degrees.
This inhibits production of prostaglandins and thromboxanes – with COX-2 inhibition associated with therapeutic effects, in contrast to COX-1 inhibition which is associated with gastrointestinal adverse effects.
Enolic acid derivatives
Piroxicam
Meloxicam
Tenoxicam
Lornoxicam
Selective COX-2 inhibitors
Celecoxib
Etoricoxib
Nimesulide
Propionic acid derivatives
Ibuprofen
Naproxen
Fenoprofen
Ketoprofen
Flurbiprofen
Oxaprozin
Acetic acid derivatives
Indomethacin
Diclofenac
Ketorolac
Etodolac
Sulindac
Tolmetin
Nabumetone
Salicylates
Aspirin
Diflunisal
Salsalate
Fenamates
Mefenamic acid

Metformin reduces hepatic glucose output and increases peripheral uptake of glucose. It does not cause weight gain. Glitazones bind to PPARγ – a receptor that influences insulin-sensitive genes.


Clotting disorder drugs
Antiplatelet drugs
Phosphodiesterase inhibitors
Dipyridamole (PDE5)
Cilostazol (PDE3)
Glycoprotein IIb/IIIa inhibitors
Abciximab
Eptifibatide
Tirofiban
ADP inhibitors
Clopidogrel
Prasugrel
Ticagrelor
Ticlopidine
COX inhibitors
Aspirin
Anticoagulant drugs
Direct Xa inhibitors
Rivaroxaban
Apixaban
Edoxaban
Direct thrombin inhibitors
Dabigatran
Argatroban
Lepirudin
Bivalirudin
Vit K epoxide reductase inhibitor
Warfarin
Indirect thrombin inhibitors
Heparin
Enoxaparin
LMWH
Fondaparinux
Related to LMWHs; synthetic factor Xa inhibitor. Unlike heparin, fondaparinux does not inhibit thrombin.
Thrombolytics
Plasminogen activators
Alteplase
Reteplase
Tenecteplase
Streptokinase
Inactive until it forms a complex with circulating plasminogen.
Antifibrinolytic drugs
Tranexamic acid


Diuretics
Loop diuretics
Uses
Heart failure
Hypercalcemic states
Edema
Hypertension
Pulmonary edema
Bumetanide
Furosemide
Ethacrynic acid
Side effects
Hyperuricemia
Ototoxicity
Hypomagnesemia
Hypocalcemia
Hypokalemia
GI effects (w/ ethacrynic acid)
Risk of kidney failure if taken with NSAID and ACE inhibitor
K-sparing diuretics
Side effects
Hyperkalemia
Acidosis
Headache
Nausea
Amiloride
Triameterene
Uses
Hypertension
CHF
Spironolactone
Side effects
Hyperkalemia
Antiandrogen effects
Drowsiness
Dry skin/rash
Depression
Uses
Hypertension
Heart failure
Nephrotic syndrome
Ascites
Hyperaldosteronism
Thiazides
Uses
Hypertension
CHF
Nephrolithiasis
Edema
Nephrogenic diabetes insipidus
Side effects
Hypokalemia
Hypercalcemia
Hyperuricemia
Hyperglycemia
Hyperlipidemia
Bendroflumethiazide
Hydrochlorothiazide
Metolazone
Carbonic anhydrase inhibitors
Acetazolamide
Dorzolamide
Uses
Glaucoma
Mountain sickness
Metabolic alkalosis
Side effects
Hyponatremia
Hypokalemia
Paresthesia
Metabolic acidosis
Kidney stones
Fatigue
Osmotic diuretics
Mannitol
Side effects
Acute hypovolemia
Uses
Lower IOP in glaucoma
Lower intracerebral pressure
Oliguric states


Antihypertensive drugs
Renin-Angiotensin System
ACE inhibitors
Captopril
Enalapril
Lisinopril
Ramipril
Perindopril
Ramipril
Angiotensin receptor antagonists
Candesartan
Losartan
Valsartan
Olmesartan
Direct renin inhibitor
Aliskiren
Vasodilators
Potassium channel opener
Hydralazine
Sodium nitroprusside
Minoxidil
Diuretics
Bendroflumethiazide
Chlortalidone
Furosemide
Amiloride
Eplerenone
Spironolactone
Pulmonary hypertension
Endothelin receptor antagonists
Bosentan
Ambrisentan
PDE5 inhibitors
Sildenafil
Tadalafil
Prostaglandins
Epoprostenol
Iloprost
Treprostinil
Centrally-acting
Alpha-2 agonists
Clonidine
Methyldopa
Guanfacine
Selective imidazoline receptor agonist
Moxonidine
Calcium channel blockers
Amlodipine
Nifedipine
Diltiazem
Verapamil
Acts on Sympathetic Nervous System
Beta blockers
Atenolol
Bisoprolol
Nebivolol
Propanolol
Alpha blockers
Alpha-1 selective
Doxazosin
Prazosin
Non-selective
Phenoxybenzamine
Phentolamine

Examples of Adverse Effects (1 of 2)

Drug	Adverse effect(s)
Thalidomide	Birth defects
Some sedatives/analgesics	Propensity to addiction
Aspirin	Internal bleeding
COX-2 inhibitors	Cardiovascular disease (eg. Vioxx)
Gentamicin	Deafness and kidney failure
Interferon	Depression and hepatic injury
Orlistat	Diarrhea
Antidepressants	Erectile dysfunction
Vaccinations	Fever
Chemotherapy	Hair loss

Antifungal Drugs

Antifungal drugs are medicines deployed in the treatment of mycoses. Fungal infections of the skin are, as of 2010, the fourth most common disease in the world – afflicting up to 1 billion people.

Imidazoles (COMET-K)

- Clotrimazole
- Oxiconazole
- Miconazole
- Econazole
- Tioconazole
- Ketoconazole

Triazoles (FIT VIP)

- Fluconazole
- Itraconazole
- Terconazole
- Voriconazole
- Isavuconazole
- Posaconazole

Azole antifungals

Inhibit lanosterol-14α-demethylase, the enzyme required to convert lanosterol into ergosterol.

Griseofulvin

Inhibits mitosis in dermatophytes. It is ineffective when applied topically.

Flucytosine

Pyrimidine analogue; converted into 5-fluorouracil by the fungal enzyme, cytosine deaminase. Active against yeast infections.

Polyenes (NAN)

- Natamycin
- Amphotericin B
- Nystatin

Echinocandins (CAM)

- Caspofungin
- Anidulafungin
- Micafungin

Inhibit cell wall synthesis by targeting glucans (1,3-β-glucan synthase).

Allylamines (ANT)

- Amorolfin
- Naftifine
- Terbinafine

→ Inhibit squalene epoxidase

Antiarrhythmic Drugs

Class Ia

1 Double Quarter Pounder with Lettuce, Mayo & Tomato and More Fries Please!

Disopyramide

Quinidine

Procainamide

Class Ib

Lidocaine

Mexeletine

Tocainide

Class Ic

Moricizine

Flecainide

Propefanone

Class II

Beta blockers? lol

Propanolol

Atenolol

Metoprolol

Class III

This is SAD

Sotalol

Amiodarone

Dofelitide

Class IV*

I and V in Class IV?

Diltiazem

Verapamil

* *Calcium channel blockers*

Antihypertensive Drugs and Pregnancy

Not every antihypertensive drug is safe for use in pregnancy. Think of the following mnemonic for those drugs which are:

'Her New Lab Method'

Hydralazine

Nifedipine

Labetalol

Methyldopa

Benzodiazepines

Benzodiazepines are often classified in terms of their elimination half-life – into short-acting, intermediate-acting and long-acting drugs.

Their half-life influences their clinical use and effects.

Short-acting	Intermediate-acting	Long-acting
'ATOM'	'FUN CLOTHES'	'CAMERON DIAZ HURRY!'
Alprazolam	Flunitrazepam	Clorazepate
Triazolam	Clonazepam	Diazepam
Oxazepam	Lorazepam	Chlordiazepoxide
Midazolam	Temazepam	Flurazepam

- ✓ **Short-acting benzos** have an average half-life of 1-12 hours. Few residual effects if taken before bedtime. May cause rebound anxiety with long-term use.
- ✓ **Intermediate-acting** drugs have an average half-life of 12-40 hours. Rebound insomnia more common on discontinuation than with long-acting compounds.
- ✓ **Long-acting** compounds have an average half-life of 40-250 hours. Risk of accumulation in the elderly and those with liver impairment.

Calcium Channel Blockers

L-type calcium channel blockers are used as antihypertensive drugs or antiarrhythmics; depending on whether the drug has a higher affinity for the heart (such as the phenylalkylamines, like verapamil), or for blood vessels (the dihydropyridines, nifedipine).

Dihydropyridine – '-dipine'	Non-dihydropyridine (DIVER)
Amlodipine	Diltiazem
Felodipine	Verapamil
Nicardipine	
Nifedipine	
Nimodipine	

Side effects		
Dizziness, headache	Fluid build-up	Facial redness
Altered heart rate	Constipation	Gingival overgrowth

CYP Inducers and Inhibitors

CYP enzymes are proteins that contain a heme cofactor – they are **hemoproteins.** They are also responsible for **75 percent** of total drug metabolism. Some drugs induce, whereas other drugs inhibit, the activity of these enzymes.

For example - St. Johns Wort *Induces* activity of CYP3A4 – meaning substrates of this enzyme (such as ketoconazole) are more rapidly metabolised.

Inducers

BULLSHIT CRAP GP'S

- **B**arbiturates
- **S**t. Johns Wort
- **C**arbamazepine
- **R**ifampin
- **A**lcohol (*chronic use*)
- **P**henytoin
- **G**riseofulvin
- **P**henobarbital
- **S**ulfonylureas

Inhibitors

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- **S**odium Valproate
- **I**soniazid
- **C**imetidine
- **K**etoconazole
- **F**luconazole
- **A**lcohol
- **C**hloramphenicol
- **E**rythromycin
- **S**ulfonamides
- **C**iprofloxacin
- **O**meprazole
- **M**etronidazole

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Grapefruit Juice Amiodarone Quinidine