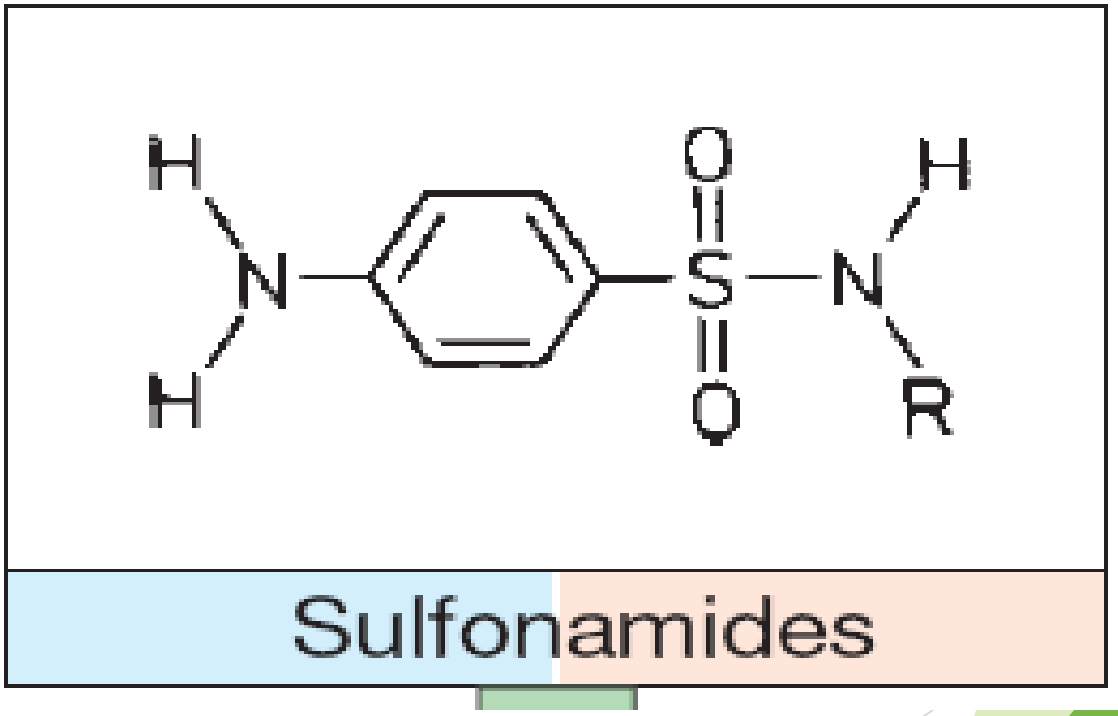
In the 1935 Domagk first demonstrated that prontosil could influence bacterial infection. It was the first sulfanilamide…

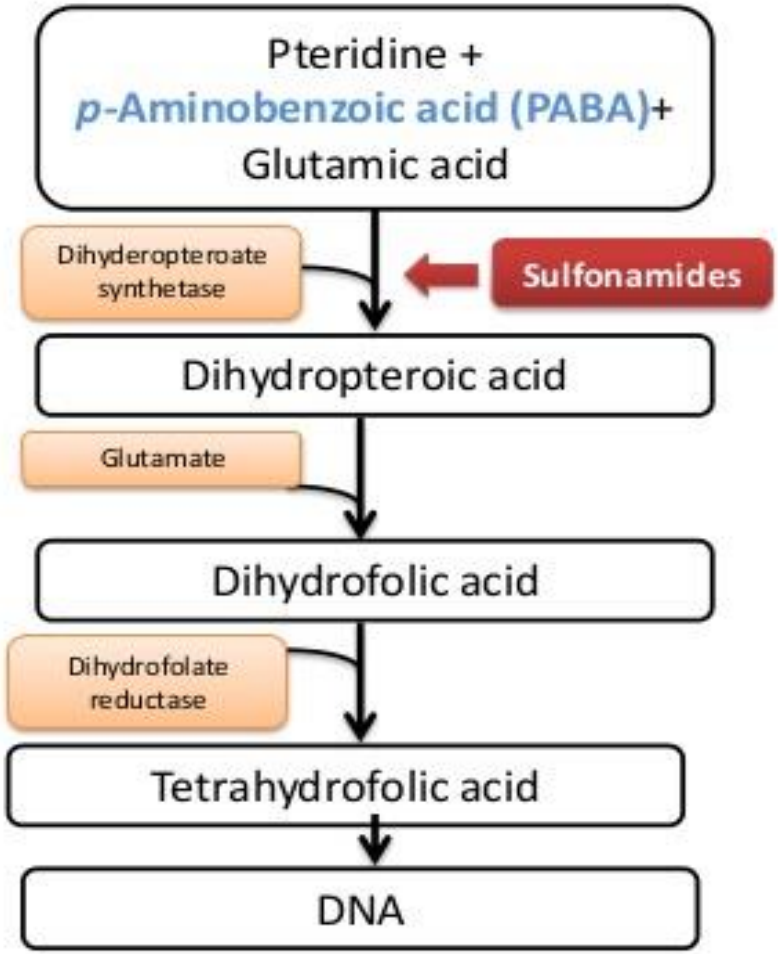
Sulfanilamides are effective against:

Bacteria
Protozoa
Chlamydia
Aktinomyces



Mechanism of action

- Bacteria synthesize their own folic acid (FA) of which *p*-aminobenzoic acid (PABA) is a constituent, and is taken up from the medium.
- Sulfonamides, are structural analogues of PABA, inhibit bacterial folate synthase and formation of folate get inhibited.
- Sulfonamides competitively inhibit the PABA with pteridine residue to form dihydropteroic acid which conjugates with glutamic acid to produce dihydrofolic acid.
- Sulfonamide altered folate an which is metabolically injurious



Sulfanilamides

I. Sulfanilamides with local action

Sulfasyl-sodium, Mafenid, Algimaf

II. Sulfanilamides with systemic action

а) Short duration action (t1/2 = 8 hours)

Streptoside, Norsulfazole, Sulfadimezine, Sulfazine, Etazole, Urosulfane

б) Long duration action (t1/2 = 24-48 hours)

Sulfapiridazine, Sulfamonometoxine, Sulfadimetoxine,

в) Very long duration action (t1/2 = 65 hours)

Sulfalene

III. Sulfanilamides poorly absorbed from the gut

Ftalazol, Sulgine, Ftazine

IY. **Combined sulfanilamides**

a) Combined with trimetoprim

Bactrim ***Sulfaton***

Lidaprim ***Poteseptil***

б) Combined with aminosalicylic acid

Salazosulfapiridazine

Salazodimetoxine

Pharmacokinetics

- Sulfonamides can be divided into three major groups
  - Oral absorbable;
  - Oral nonabsorbable;
  - topical.
- absorbed from the stomach and small intestine and distributed widely to tissues and body fluids, placenta, and fetus
- Protein binding varies from 20% to over 90%
- A portion of absorbed drug is acetylated or glucuronidated in the liver.
- Excreted into the urine, mainly by glomerular filtration.
- In significant renal failure, the dosage of sulfonamide must be reduced.

Basic characteristics (remember of **SULFA**):

Solubility low

Useful for Urinary tract infections

Large spectrum (active against gram positive and gram negative bacteria)

Folic acid synthesis blocker (DHPS inhibitor)

Antimetabolite /**A**nalog of PABA

Side effects of Sulfonamides include (remember of **SULFA**):

Steven-Johnson syndrome / **S**kin rashes

Urticaria / **U**rine precipitation (crystalluria)

Leucopenia

Folic acid deficency

Aplastic Anemia

Drug interactions

S.No	Interfering drug	Effect
1	Sulfonylureas	Displaced from protein binding site probable hypoglycemia
2	Phenytoin, warfarin	↑ action of both
3	Methotrexate	Displaced also ↓ excretion toxicity may occur
4	Procaine (Contain PABA)	Direct inhibition of sulfonamide action
5	Phenylbutazone , salicylate , probenecid	Sulfonamide displaced from protein binding ↑ activity of sulfonamides

Antibacterial drugs with different chemical structure

- ✓ Quinolone derivatives
- ✓ 8-quinolone derivatives
- ✓ Nitrofuran derivatives
- ✓ Quinoxaline derivatives

✓8-oxyquinolone derivatives

I. With systemic action

a) Low intestinal permeability

Enteroseptol, Mexaze, Mexaform, Intestopan

b) High intestinal permeability

Nitroxaline (5-NOC)

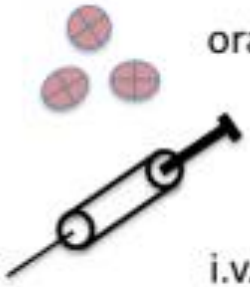
II. With local action

Dermozolone

Medicines	Route of administration	t ½ hour	Elimination	BBB permeability
Ciprofloxacin	Enteral, iv	3-4,5	Kidney	+
Norfloxacin	Enteral	4-5	Kidney	+/ -
Ofloxacin	Enteral	5-7	Kidney	+
Perfloxacine	Enteral, iv	9	Kidney	+
Lomefloxacine	Enteral	8	Kidney	+/ -
Enoxacine	Enteral	3-6	Kidney	
Rufloxacine	Enteral	33	Kidney	

Fluoroquinolone Uses

Routes:



oral

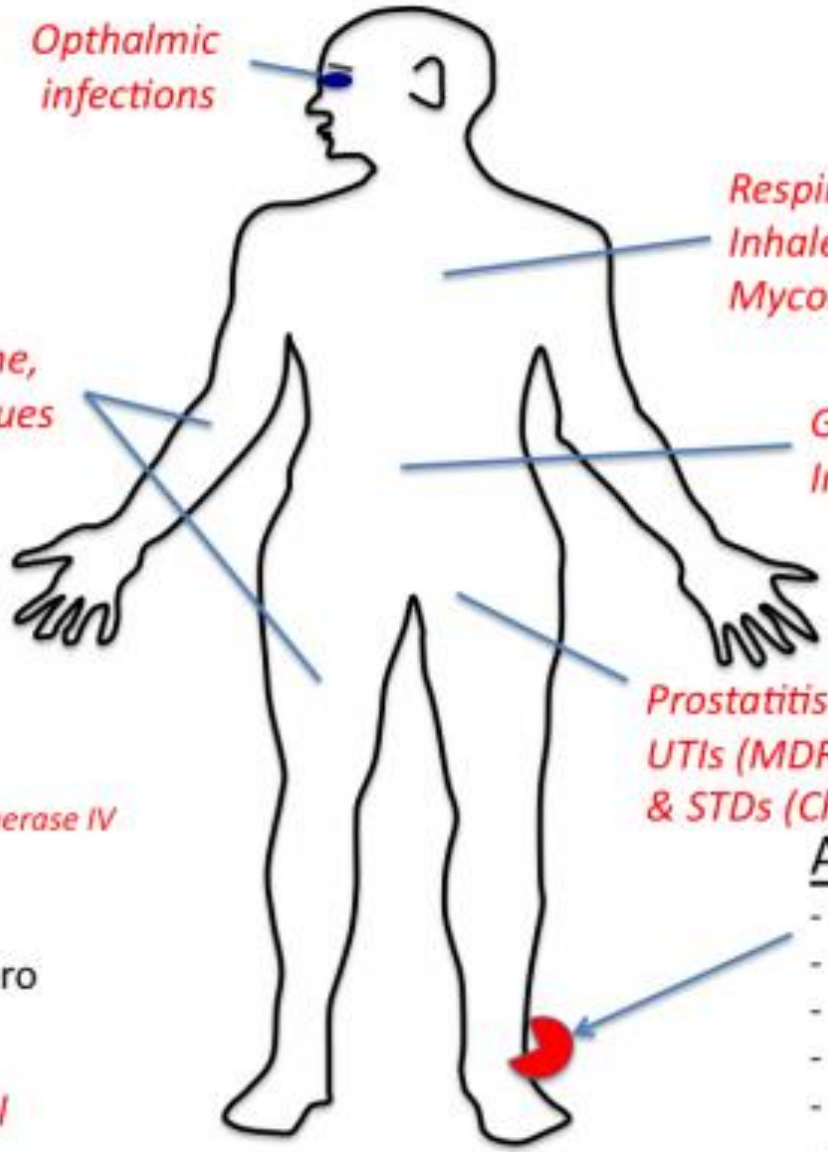
i.v.

Mechanism:

Inhibits DNA Gyrase & Topoisomerase IV

Cipro

bactericidal



Opthalmic infections

Respiratory Infections, Inhaled Anthrax, Mycobacteria (TB)

Infections of bone, joints & soft tissues

GI & Abdominal Infections

Prostatitis
UTIs (MDR strains)
& STDs (Chlamydia)

Adverse Effects:

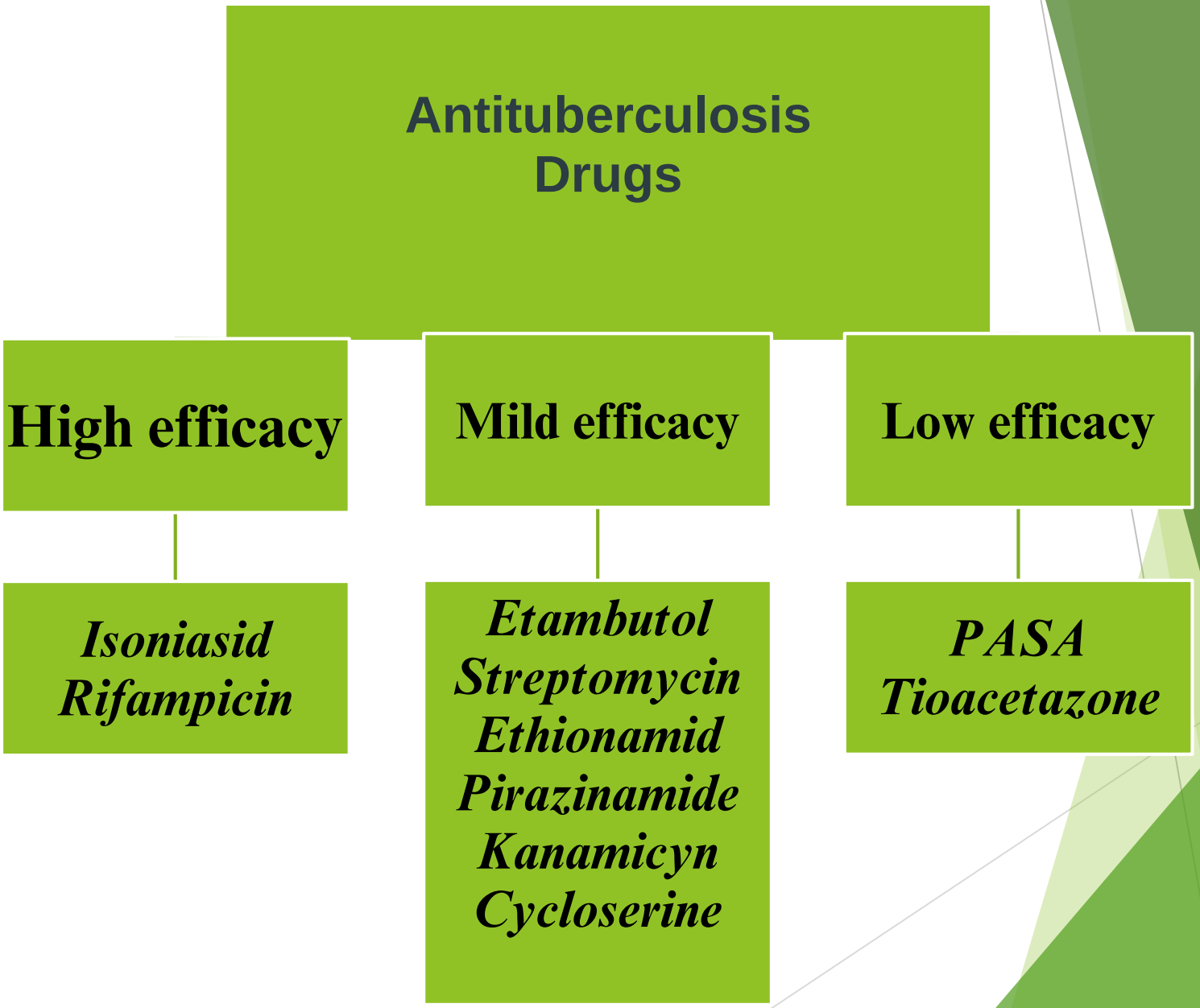
- Tendon rupture
- Children <18 yo (cartilage)
- Pregnancy Category C
- Seizures, prolong QT
- Dizziness, Confusion
- Photosensitivity

Antimycobacterial Drugs

Mycobacteria are intrinsically resistant to most antibiotics. Because they grow more slowly than other bacteria, antibiotics that are most active against rapidly growing cells are relatively ineffective. Mycobacterial cells can also be dormant and thus completely resistant to many drugs or killed only very slowly. The lipid-rich mycobacterial cell wall is impermeable to many agents. Mycobacterial species are intracellular pathogens, and organisms residing within macrophages are inaccessible to drugs that penetrate these cells poorly. Finally, mycobacteria are notorious for their ability to develop resistance. Combinations of two or more drugs are required to overcome these obstacles and to prevent emergence of resistance during the course of therapy. The response of mycobacterial infections to chemotherapy is slow, and treatment must be administered for months to years, depending on which drugs are used.

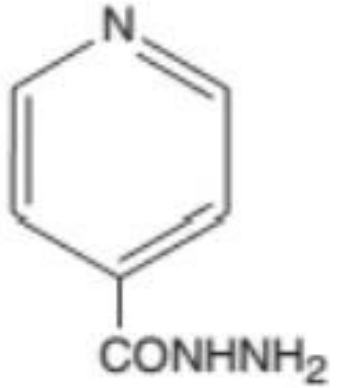
Classification

- **Synthetic drugs:**
  Isoniazid Ethambutol Sodium paraaminosalicylateEtionamide Protionamide Pyrazinamide
- **Antibiotics**
  Rifampicin, Streptomycin, Cycloserin, Kanamycin


Antituberculosis Drugs
High efficacy
Isoniasid
Rifampicin
Mild efficacy
Etambutol
Streptomycin
Ethionamid
Pirazinamide
Kanamicyn
Cycloserine
Low efficacy
PASA
Tioacetazone

ISONIAZID

- Isonicotinic acid hydrazide
- Most active drug for the treatment of tuberculosis
- freely soluble in water
- bactericidal for actively growing tubercle bacilli
- less effective against atypical mycobacterial species
- penetrates into macrophages and is active against both extracellular and intracellular organisms



Isoniazid

Mechanism of Action & Basis of Resistance

- inhibits synthesis of mycolic acids - essential components of mycobacterial cell walls
- Higly selective for mycobacterium
- Resistance
  - Its prodrug – activated by enzyme catalase-peroxidase
  - Mutation causes inhibition of this enzyme
  - No cross resistance occurs with other antitubercular drug
  - Always given in combination

Pharmacokinetics

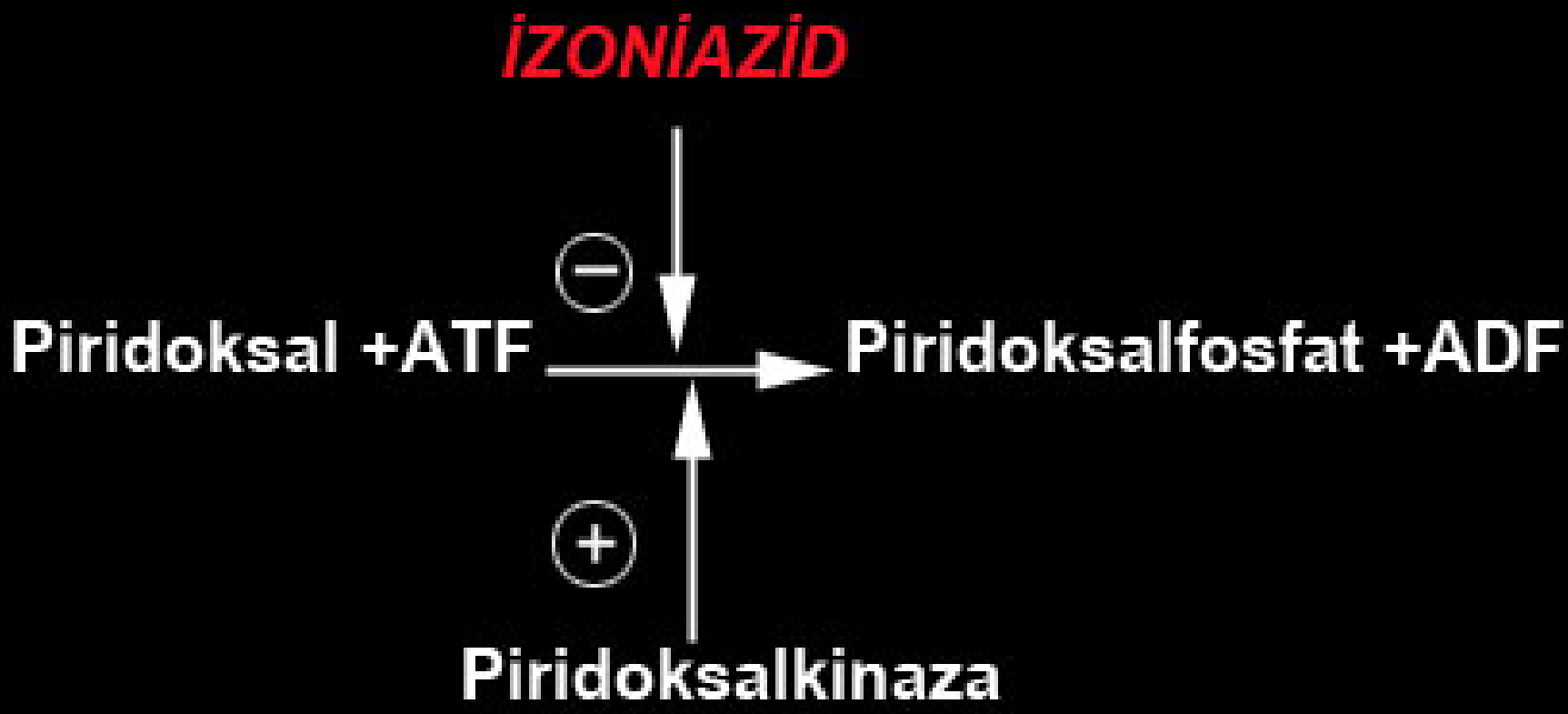
- readily absorbed from the gastrointestinal tract - diffuses readily into all body fluids and tissues.
- acetylation by liver *N* -acetyltransferase, is genetically determined
- half-lives :1 hour(fast acetylators) and 3 hours (slow acetylators)
- Excreted, mainly in the urine - need not be adjusted in renal failure
- Contraindicated - severe preexisting hepatic insufficiency

Adverse Reactions

- Depends on dosage and duration of administration
- **Immunologic Reactions**
  - Fever and skin rashes are occasionally seen.
  - Drug-induced **systemic lupus erythematosus** has been reported.
- **Direct Toxicity**
  - Clinical hepatitis with loss of appetite, nausea, vomiting, jaundice – promptly discontinued
  - The risk of hepatitis is greater in individuals
    - Alcohol dependence
    - Possibly during pregnancy and the postpartum period

Adverse Reactions : Direct

- Peripheral neuropathy is observed in 10–20% - occur in slow acetylators and patients with predisposing conditions
  - malnutrition,
  - alcoholism,
  - diabetes,
  - AIDS, and uremia
- Relative pyridoxine deficiency - promotes excretion of pyridoxine
- readily reversed by administration of pyridoxine in a dosage as low as 10 mg/d
- Central nervous system toxicity : less common, includes memory loss, psychosis, and seizures.


İZONİAZİD
Piridoksal +ATF
Piridoksalfosfat +ADF
Piridoksalkinaza

RIFAMPIN

- Semisynthetic derivative of rifamycin -produced by ***Streptomyces mediterranei***
- Active in vitro against gram-positive and gram-negative cocci, some enteric bacteria, mycobacteria, and chlamydiae.
- Resistant mutants - approximately 1 in 10^6 organisms
- Rapidly selected out if rifampin is used as a single drug – must be used in combination
- no cross-resistance to other classes of antimicrobial drugs

Mechanism of Action & Resistance

- Binds to the bacterial DNA-dependent RNA polymerase - inhibits RNA synthesis
- Bactericidal for mycobacteria
- Readily penetrates most tissues and penetrates into phagocytic cells
- Can kill organisms that are poorly accessible to many other drugs
  - Intracellular organisms
  - sequestered in abscesses and lung cavities
- **Resistance:** mutations result in reduced binding of rifampin to RNA polymerase

Ethambutol

Ethambutol inhibits mycobacterial arabinosyl transferases, which are encoded by the embCAB operon. Arabinosyl transferases are involved in the polymerization reaction of arabinoglycan, an essential component of the mycobacterial cell wall. Resistance to ethambutol is due to mutations resulting in overexpression of emb gene products or within the embB structural gene

Ethambutol is given orally. It is generally well tolerated, but may cause dose dependent damage to the optic nerve with disturbances of vision (red/green blindness, visual field defects).

- ***Pyrazinamide***
- It is given orally. Pyrazinamide may impair liver function; hyperuricemia results from inhibition of renal urate elimination.

- ***Streptomycin*** must be given i.v. i.m. like other aminoglycoside antibiotics. It damages the inner ear and the labyrinth. Its nephrotoxicity is comparatively minor.

DRUGS USED IN LEPROSY

Dapsone is a sulfone that, like sulfonamides, inhibits dihydrofolate synthesis**.** It is bactericidal against susceptible strains of *M. leprae.* Dapsone is given orally. The most frequent adverse effect is methemoglobinemia with accelerated erythrocyte degradation (hemolysis).

Clofazimine is a dye with bactericidal activity against *M. leprae* and anti-inflammatory properties. It is given orally, but is incompletely absorbed. Because of its high lipophilicity, it accumulates in adipose and other tissues and leaves the body only rather slowly (t1/2 ~ 70 d). Red-brown skin pigmentation is an unwanted effect, particularly in fair-skinned patients.

Antisyphilitic drugs

Antibiotics: Penicillins

Tetracyclines

Azytromycine

Ceftriaxone

Bismuth drugs :

Bismoverol

Biyochinol

Iodine drugs:

Potassium iodide

Antifungal Agents

- Human fungal infections have increased dramatically in incidence and severity in recent years, owing mainly to advances in surgery, cancer treatment, treatment of patients with solid organ and bone marrow transplantation, the HIV epidemic, and increasing use of broad-spectrum antimicrobial therapy in critically ill patients. These changes have resulted in increased numbers of patients at risk for fungal infections.

Chemical Classification of antifungal drugs

1. Antibiotics

   a)Polyenes: Amphotericin B(AMB),Nystatin, Hamycin, Natamycin (Pimaricin)

   b) Heterocyclic benzofuran: Griseofulvin

2. Antimetabolite: Flucytosine(5-FC)

3. Azoles:

   a) Imidazoles (topical):Econazole, Miconazole,Clotrimazole,Oxiconazole, Ketonazole(Systemic)

   b)Triazoles (systemic):Fluconazole, Itraconazole, Voriconazole

4. Allylamine: Terbinafine

5. Other topical agent:Tolnaftate, Undecylenic acid, Benzoic acid, Quiniodochlor, Ciclopirox olamine, Butenafine, Sod. thiosulfate.

Classification of antifungal drugs based on treatment

- Drugs used to treat systemic fungal infection

1. Triazoles
2. Amphotericin-B
3. Ketoconazole(Imidazole)
4. Echinocandis
5. Flucytosine(5-FC)

- Drugs given systemically for treating Superficial infections

1. Griseofulvin
2. Terbinafine

- Topically used Antifungal drugs

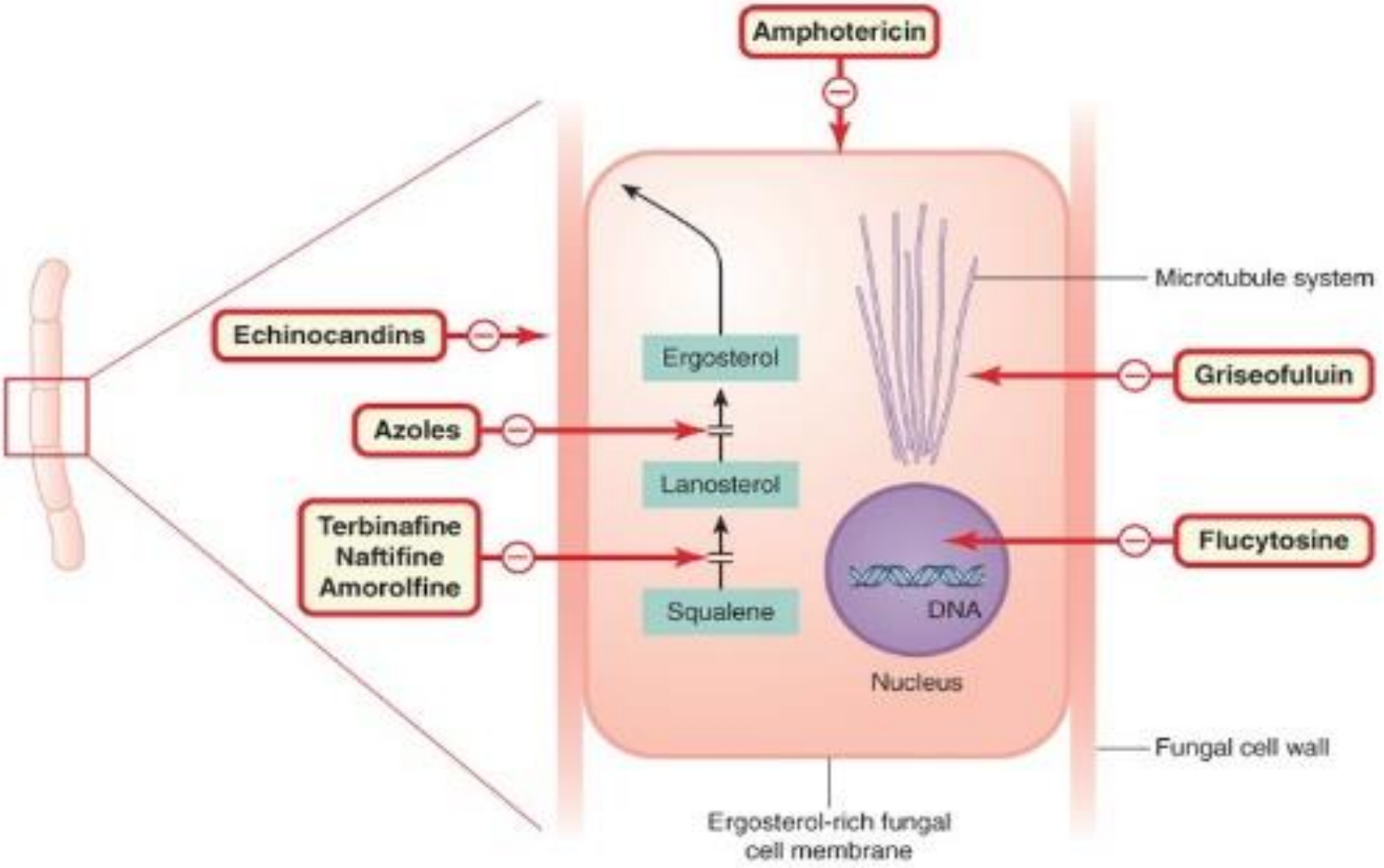
-Nystatin

-Clotrimazole,Miconazole,Butaconazole,Sertaconazole,Oxiconazole

-Ciclopirox

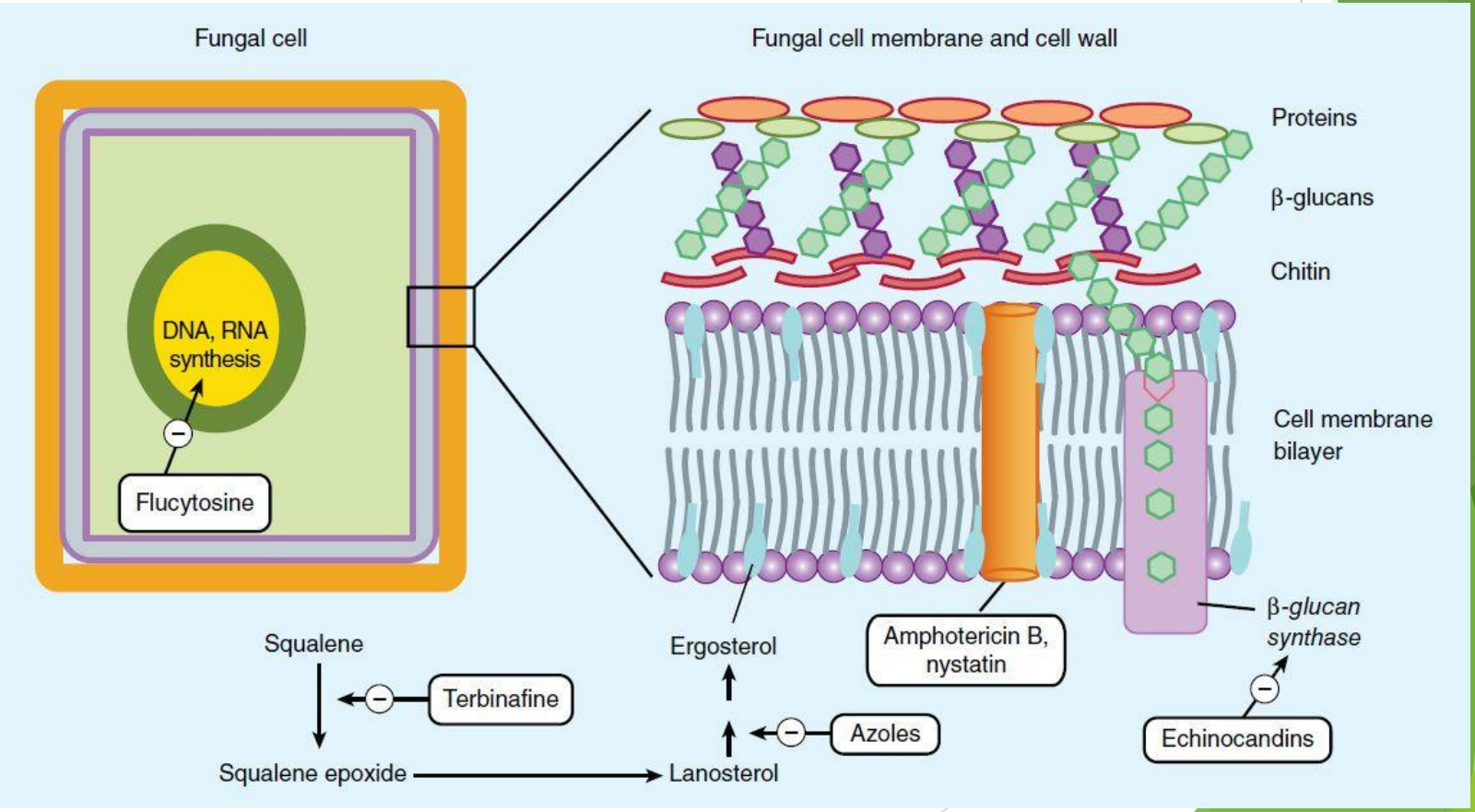
-Benzoic acid & Sodium Thiosulphate

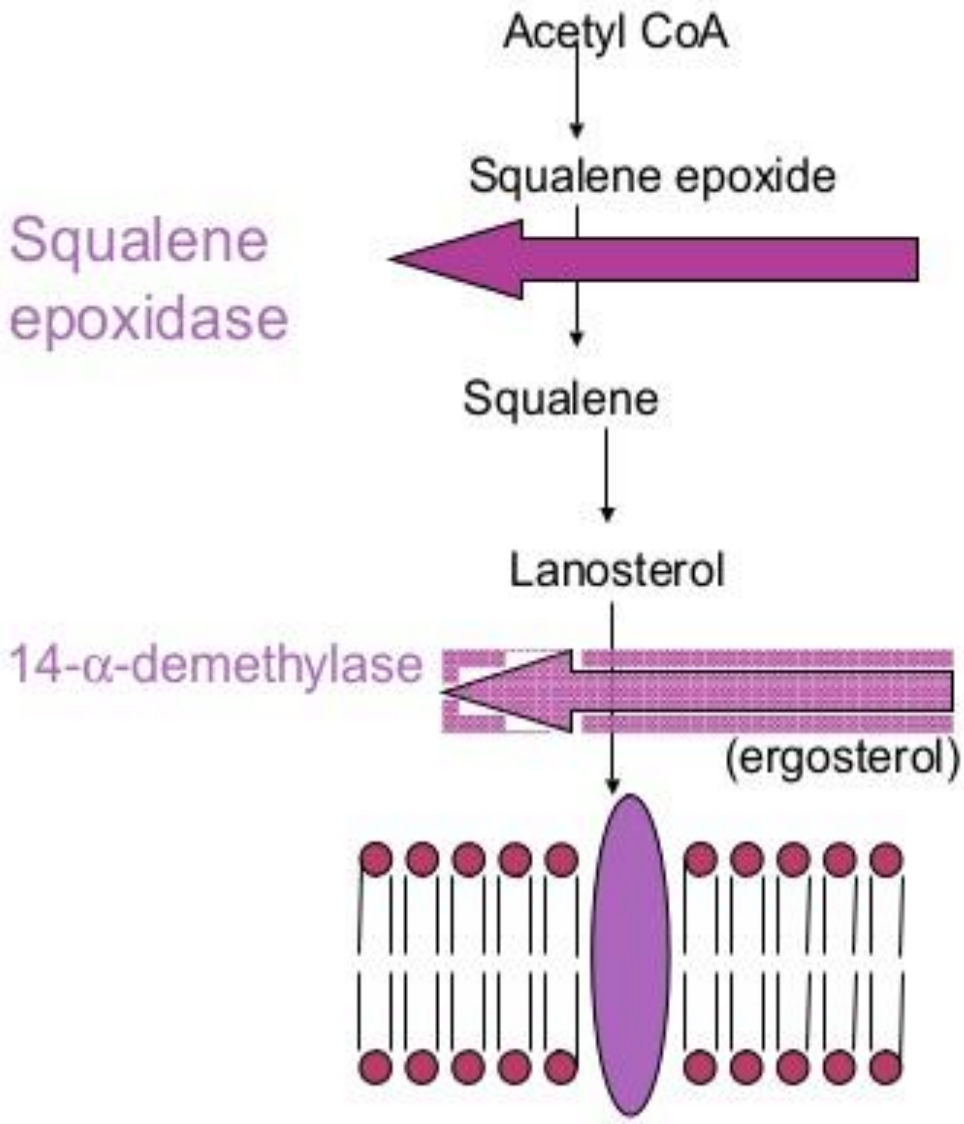
M/A of Antifungal drugs



Amphotericin B

- an amphoteric polyene macrolid, nearly insoluble in water and is therefore prepared as a colloidal suspension for intravenous injection
- **Mechanisms of Action** Amphotericin B binds to ergosterol and alters the permeability of the cell by forming amphotericin B-associated pores in the cell membrane





Allylamine drugs

Azoles

Ketoconazole

- Pharmacokinetics:

-Acidic pH required for the absorption of Ketoconazole.

- bioavailability is reduced in achlorhydria. Such patients should be given acidifying agents (like orange juice) before ketoconazole administration.

-Plasma half-life is 8-10hours

- penetration into CSF is negligible; hence it is ineffective in the treatment of fungal meningitis

- Therapeutic Uses:

-Histoplasmosis

- coccidioidomycosis

- non -CNS blastomycosis

Adverse Effects:

-Nausea, vomiting and anorexia occur commonly but these adverse effects can be minimized by taking ketoconazole with food

-Allergic dermatitis

-Reversible elevations in liver enzymes

- inhibits the synthesis of testosterone and estradiol which may lead to gynaecomastia and irregular menstrual cycles

Drug Interactions:

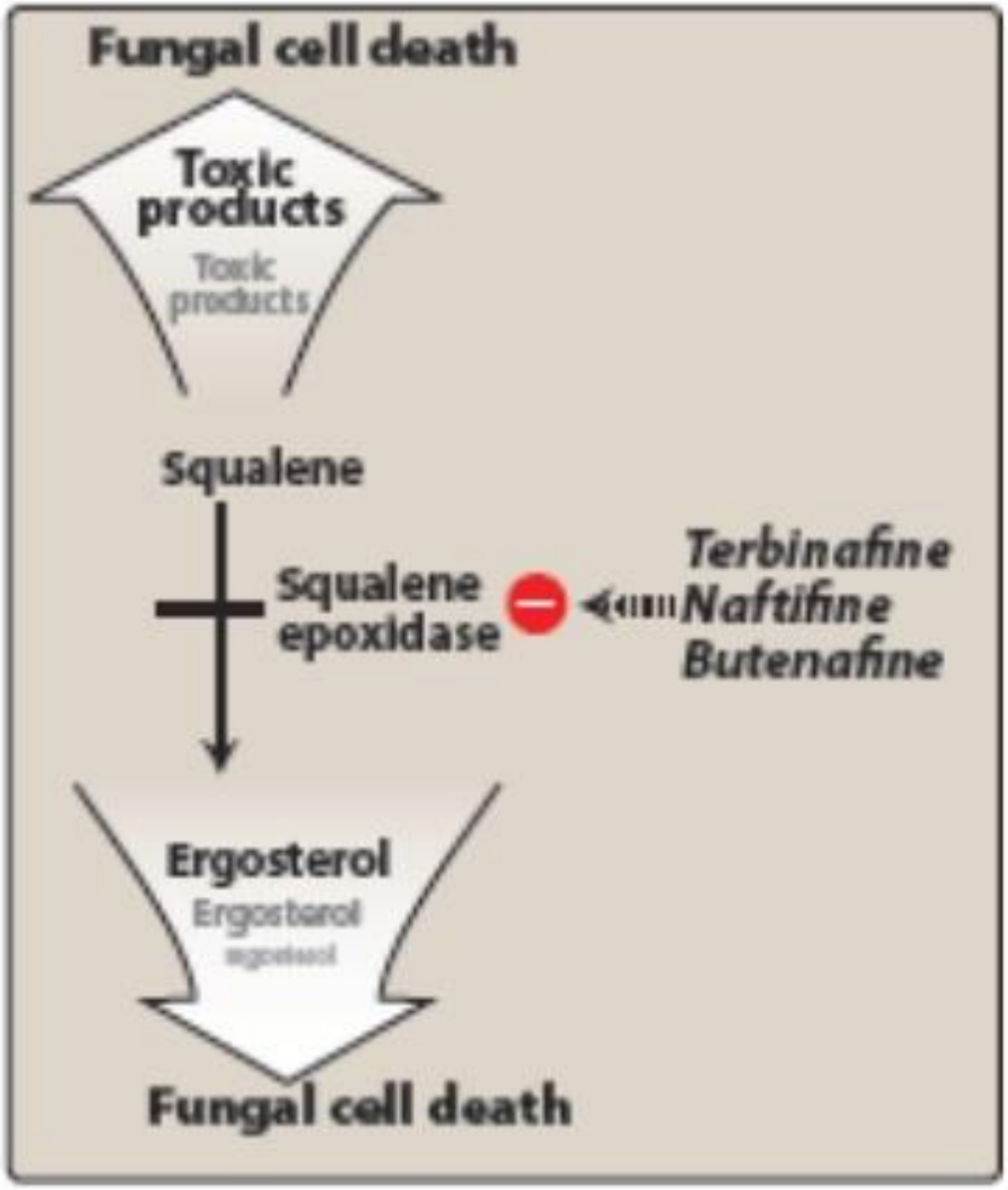
-Ketoconazole inhibit mammalian cytochrome P450 (CYP3A4) more than fungal cytochrome P450.

1. Increases the serum concentrations of cisapride, terfenadine, astemizole and quinidine, warfarin, cyclosporine, tacrolimus,

HMG-CoA reductase inhibitors

2. Rifampicin and phenytoin accelerate ketoconazole metabolism and reduce its efficacy

-H_2 receptor blockers, proton pump inhibitors and antacids decrease ketoconazole absorption by decreasing gastric acidity


Fungal cell death
Toxic products
Toxic products
Squalene
Squalene epoxidase
Terbinafine
Naftifine
Butenafine
Ergosterol
Ergosterol
Fungal cell death

Antiviral Drugs

1. Biogen agents

Interferones

2. Synthetic agents

a) Nucleozid derivatives

Zidovudin, Stavudin, Asiklovir, Valasiklovir, Qansiklovir, Trifluridin, İdoxuridin,

b) Peptid derivatives

Sakvinavir, Nelfinavir, İndinavir, Ritonavir

v) Adamantan derivatives

Amantadin, Remantadin

q) İndolkarbon acid derivatives

Arbidol

d) Fosfon acid derivatives

Foskarnet

e) Tiosemikarbazon derivatives

Metisazon

3. Herbal origin agents

Flakozid, Alpirazin, Xelepin, Meqosin, Qossipol

Understanding Viruses

Viruses are difficult to kill because they live inside the cells

- Any drug that kills a virus may also kill cells

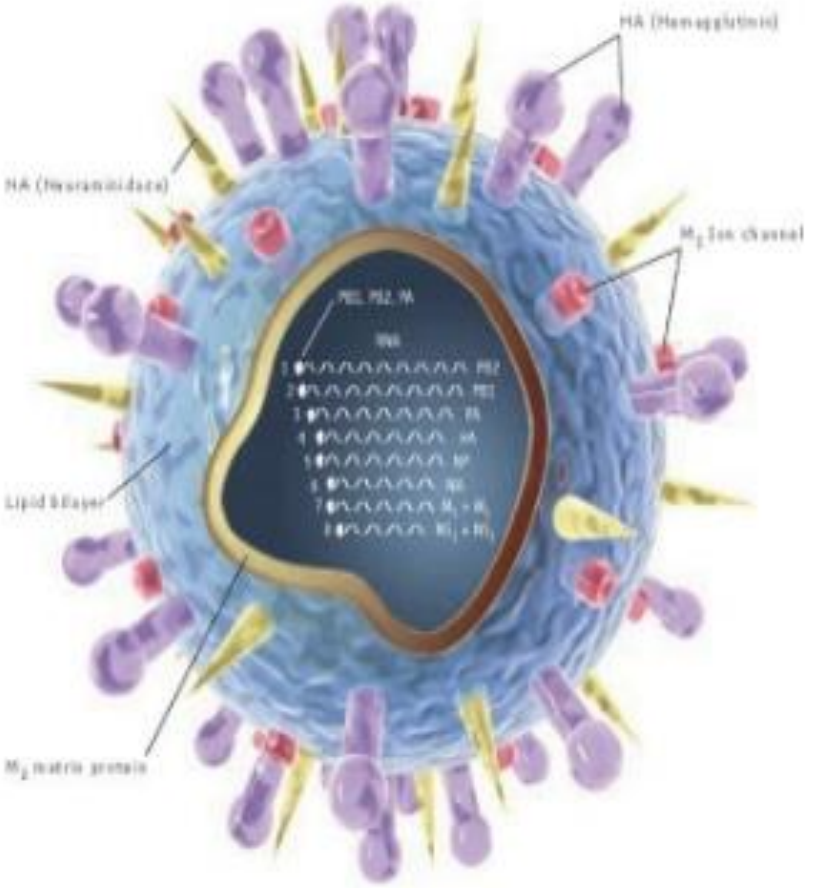


Illustration: Chris Bickel/Science. Reprinted with permission from Science Vol. 312, page 380 (21 April 2006) © 2006 by AAAS

Antivirals

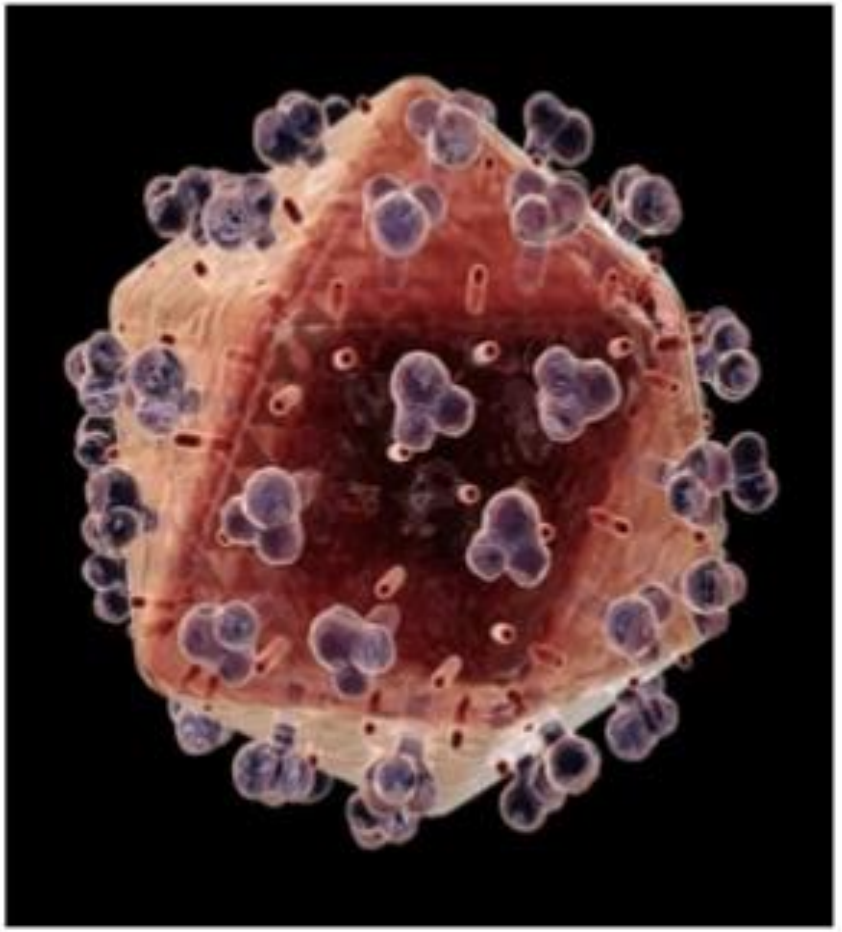
available for many viral infections

Viruses controlled by current antiviral therapy

- Cytomegalovirus (CMV)
- Hepatitis viruses
- Herpes viruses
- Human immunodeficiency virus (HIV)
- Influenza viruses (the "flu")
- Respiratory syncytial virus (RSV)

Anti-viral drugs

- Certain viruses multiply in the cytoplasm but others do in the nucleus
- Most multiplication take place before diagnosis is made



Anti-Viral drugs

- Many antiviral drugs are *Purine or Pyrimidine analogs.*
- Many antiviral drugs are Prodrugs. They must be phosphorylated by viral or cellular enzymes in order to become active.
- Anti-viral agents inhibits active replication so the viral growth resumes after drug removal.

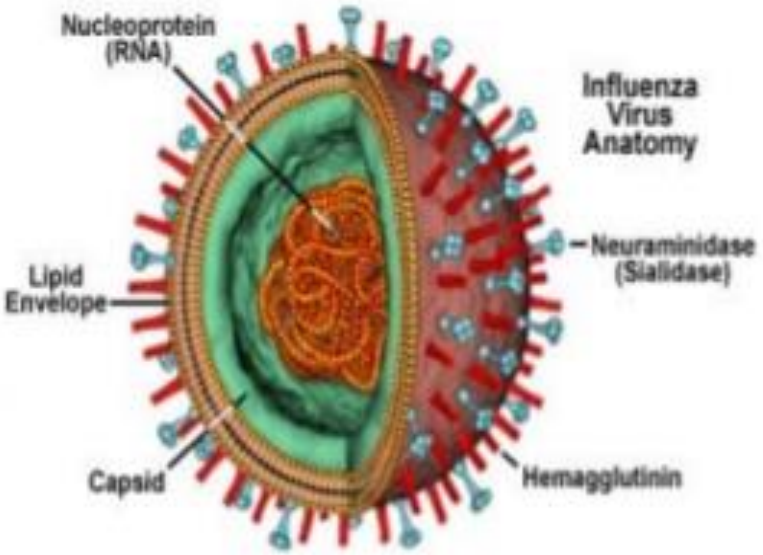
Antivirals how they act

Key characteristics of antiviral drugs

- ☐ **Able to enter the cells infected with virus**
- ☐ **Interfere with** viral nucleic acid **synthesis and/or regulation**
- ☐ Some drugs **interfere with ability of virus to bind to cells**
- ☐ Some drugs **stimulate the body's immune system**
- ☐ **Best responses** to antiviral drugs are in **patients with competent immune systems**
- ☐ A **healthy immune system works synergistically with the drug** to eliminate or suppress viral activity

Antiviral Medications

- **Antiviral drugs**
  - Used to treat infections caused by viruses other than HIV
- **Antiretroviral drugs**
  - Used to treat infections caused by HIV, the virus that causes AIDS
- **Herpes-Simplex Viruses**
  - HSV-1 (oral herpes)
  - HSV-2 (genital herpes)
- **Varicella Zoster Virus**
  - Chickenpox
  - Shingles

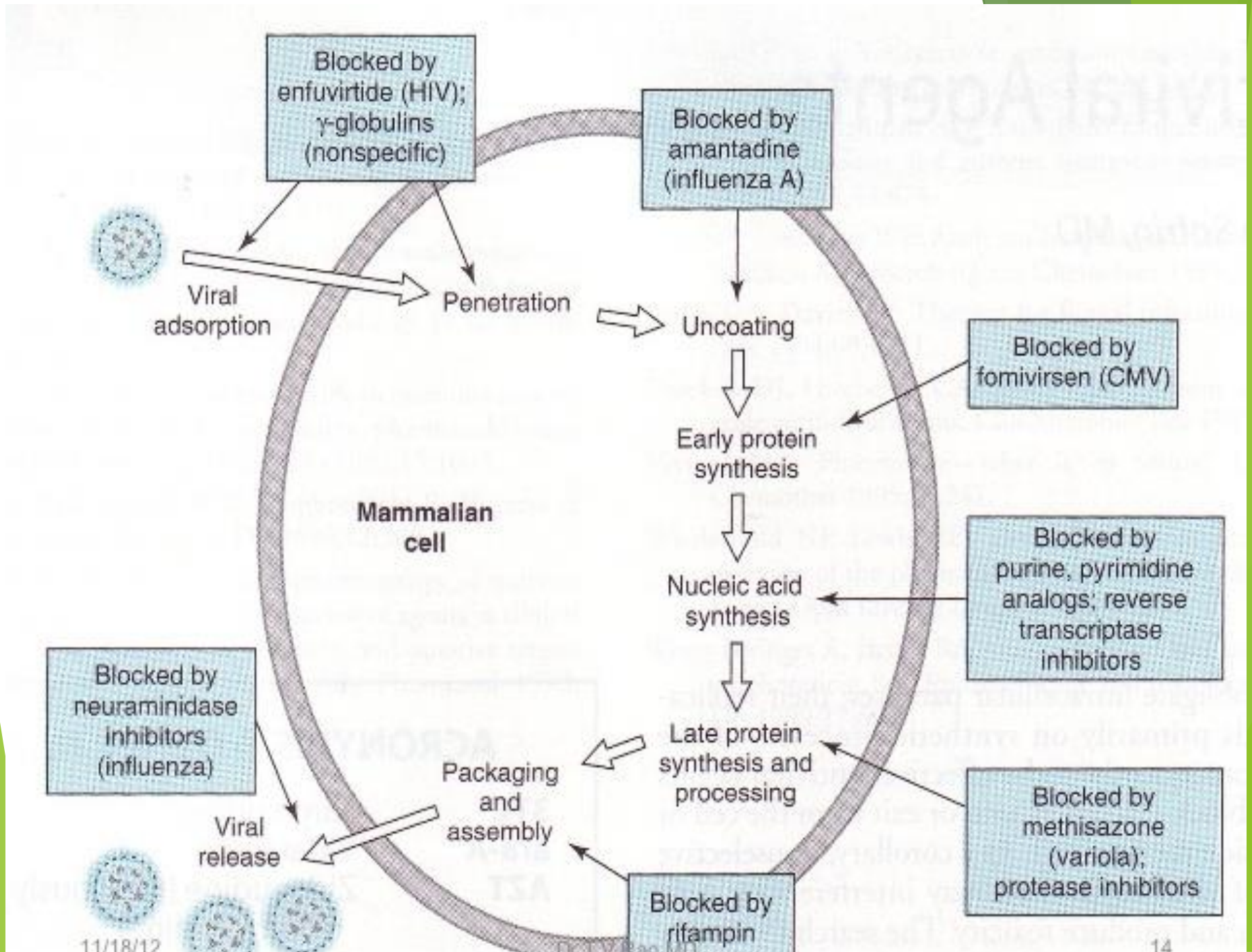


Anti-viral drugs

Stages of viral replication

- Cell entry – attachment

  - penetration
- Uncoating
- Transcription of viral genome
- Translation
- Assembly of virion components
- Release


Blocked by enfuvirtide (HIV); γ-globulins (nonspecific)
Blocked by amantadine (influenza A)
Viral adsorption
Penetration
Uncoating
Blocked by fomivirsen (CMV)
Early protein synthesis
Mammalian cell
Blocked by purine, pyrimidine analogs; reverse transcriptase inhibitors
Nucleic acid synthesis
Blocked by neuraminidase inhibitors (influenza)
Late protein synthesis and processing
Packaging and assembly
Viral release
Blocked by methisazone (variola); protease inhibitors
Blocked by rifampin

Anti-viral drugs

Anti-herpes virus agents

- Acyclovir / Valacyclovir
- Famciclovir / Penciclovir
- Ganciclovir / Cidofovir
- Foscarnet
- Trifluridine / Idoxuridine / Vidarabine

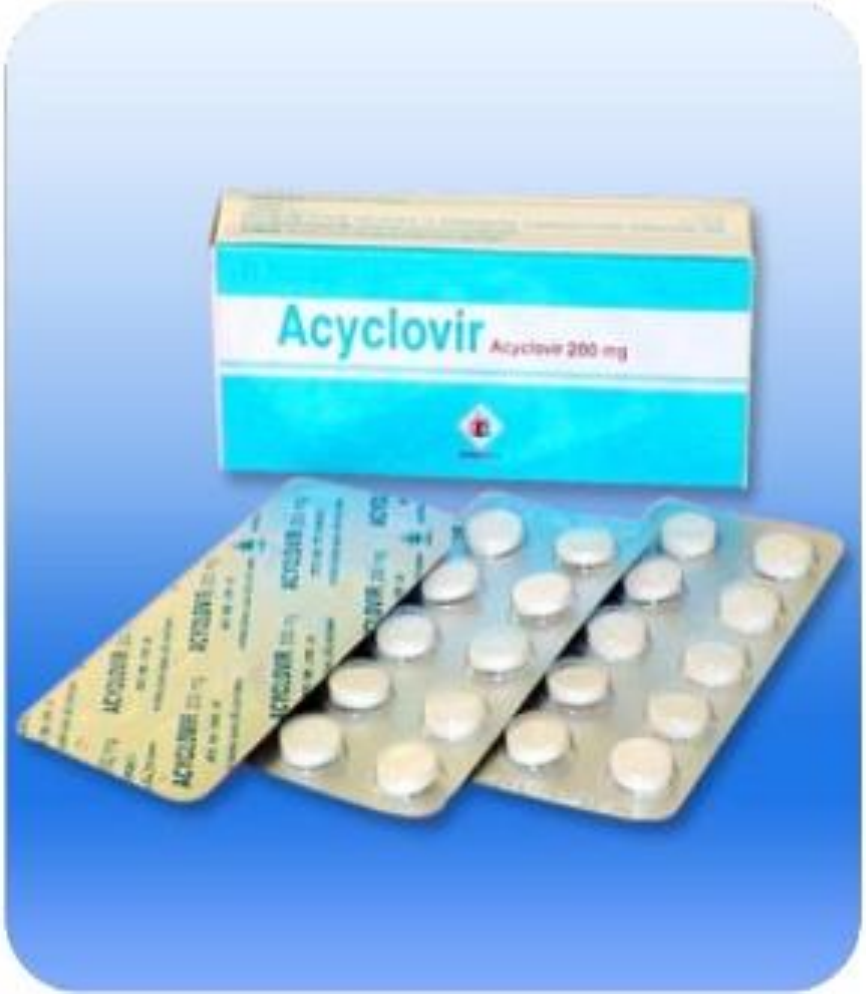
Anti-viral drugs

Mechanism of action of Acyclovir and congeners :

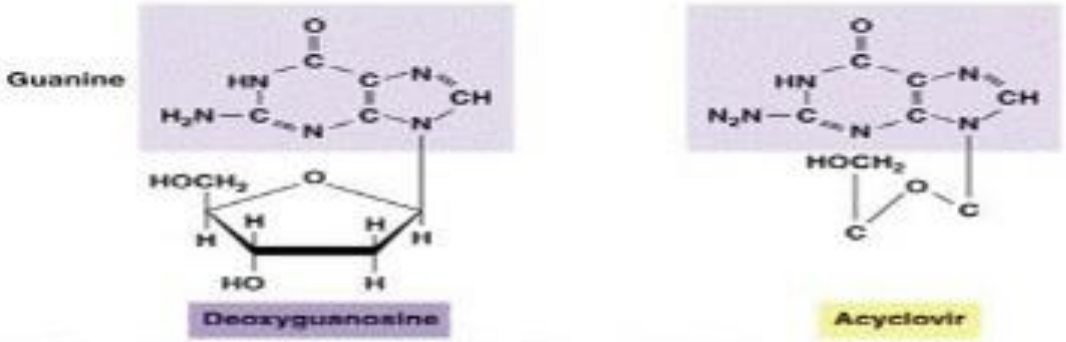
- All drugs are phosphorylated by a viral thymidine-kinase, then metabolized by host cell kinases to nucleotide analogs.
- The analog inhibits viral DNA-polymerase
- Only actively replicating viruses are inhibited

Anti-viral drugs

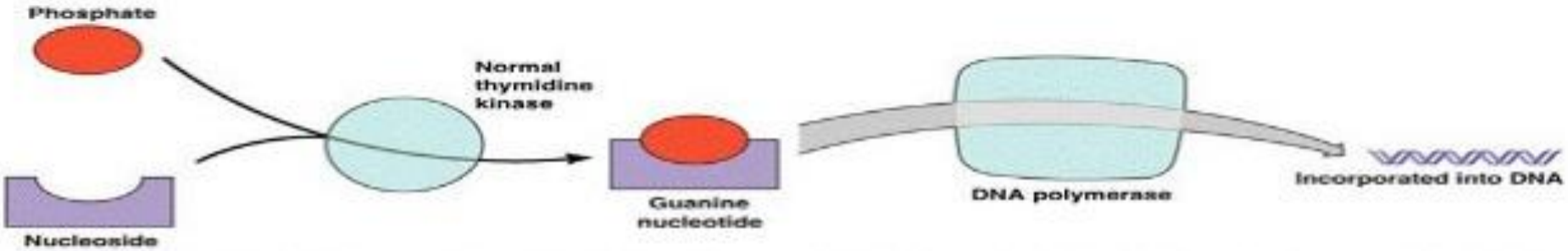
- Acyclovir is thus selectively activated in cells infected with herpes virus.
- Uninfected cells do not phosphorylate acyclovir.



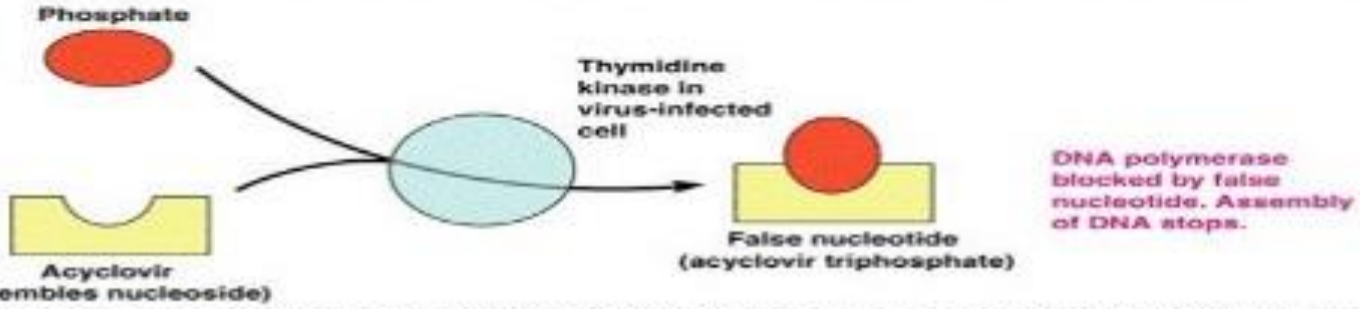
Mechanism of Action of Acyclovir



(a) Acyclovir structurally resembles the nucleoside deoxyguanosine.



(b) The enzyme thymidine kinase combines phosphates with nucleosides to form nucleotides, which are then incorporated into DNA.



(c) Acyclovir has no effect on a cell not infected by a virus, that is, with normal thymidine kinase. In a virally infected cell, the thymidine kinase is altered and converts the acyclovir (which resembles the nucleoside deoxyguanosine) into a false nucleotide—which blocks DNA synthesis by DNA polymerase.

Anti-Viral drugs

Pharmacokinetics of Acyclovir :

- Oral bioavailability ~ 20-30%
- Distribution in all body tissues including CNS
- Renal excretion: > 80%
- Half lives: 2-5 hours
- Administration: Topical, Oral , IV

Anti-viral drugs

Adverse effects of Acyclovir / Ganciclovir

- Nausea, vomiting and diarrhea
- Nephrotoxicity - crystalluria, haematuria, renal insufficiency
- Myelosuppression – Neutropenia and thrombocytopenia – Ganciclovir

Anti-viral drugs

Respiratory viral infections

Influenza –

- Amantadine / Rimantadine
- Oseltamivir / Zanamavir

(Neuraminidase inhibitors)

RSV bronchiolitis –

- Ribavirin

Anti-viral drugs

Amantadine and Rimantadine : Influenza

- Prevention & Treatment of influenza A
- **Inhibition of viral uncoating** by inhibiting the viral membrane protein M2
- Influenza A virus
- Amantadine has anti-parkinsonian effects.

Anti-viral drugs

Neuraminidase inhibitors : Influenza
Oseltamivir / Zanamavir

- **Influenza** contains an enzyme *neuraminidase* which is essential for the replication of the virus.
- ***Neuraminidase inhibitors*** prevent the release of new virions and their spread from cell to cell.

Anti-viral drugs

PHARMACOLOGY OF RIBAVIRIN

- **Ribavirin** is a guanosine analog.
- ***Inhibition of RNA polymerase***

Antiviral spectrum : DNA and RNA viruses are susceptible, including influenza, parainfluenza viruses, **RSV,** Lassa virus

Anti-viral drugs

Interferons

Interferons (IFNs) are natural proteins produced by the cells of the immune systems in response to challenges by foreign agents such as viruses, bacteria, parasites and tumor cells.

- Antiviral, immune modulating and anti-proliferative actions
- Three classes of interferons – **α , β, γ**

Antiretroviral Drugs

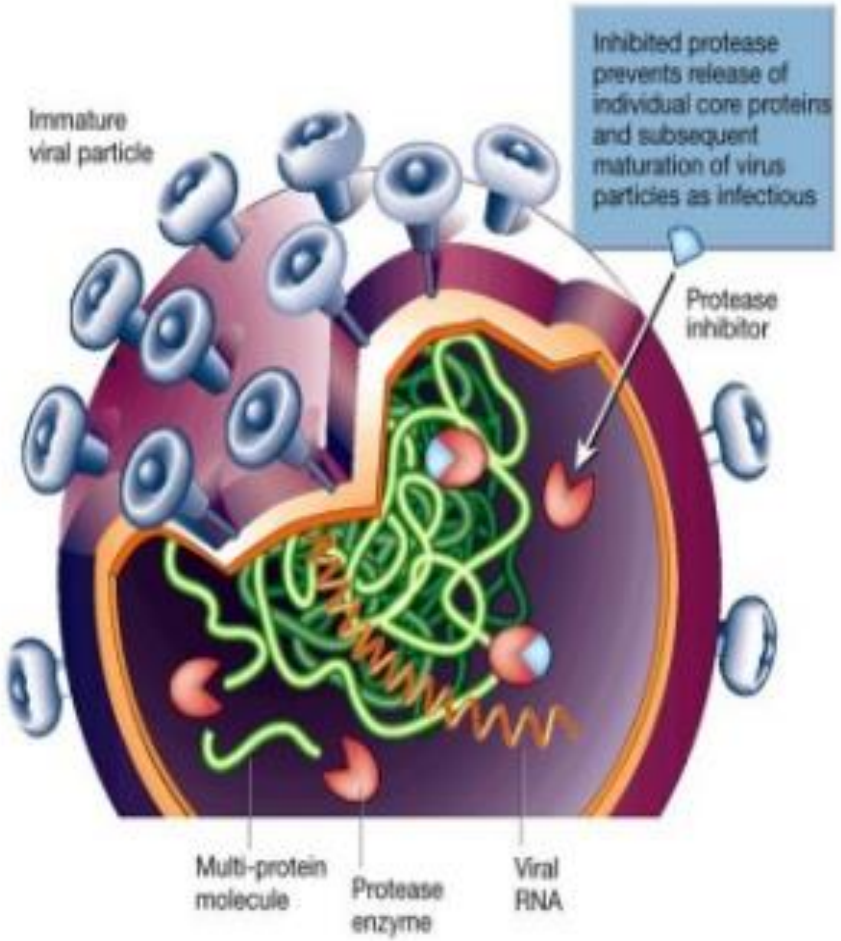
- **Reverse transcriptase inhibitors (RTIs)**
  - Block activity of the enzyme reverse transcriptase, preventing production of new viral DNA
- **Reverse transcriptase inhibitors (RTIs)**
  - Nucleoside RTIs (NRTIs)
  - Nonnucleoside RTIs (NNRTIs)
  - Nucleotide RTIs (NTRTIs)
- **Examples**

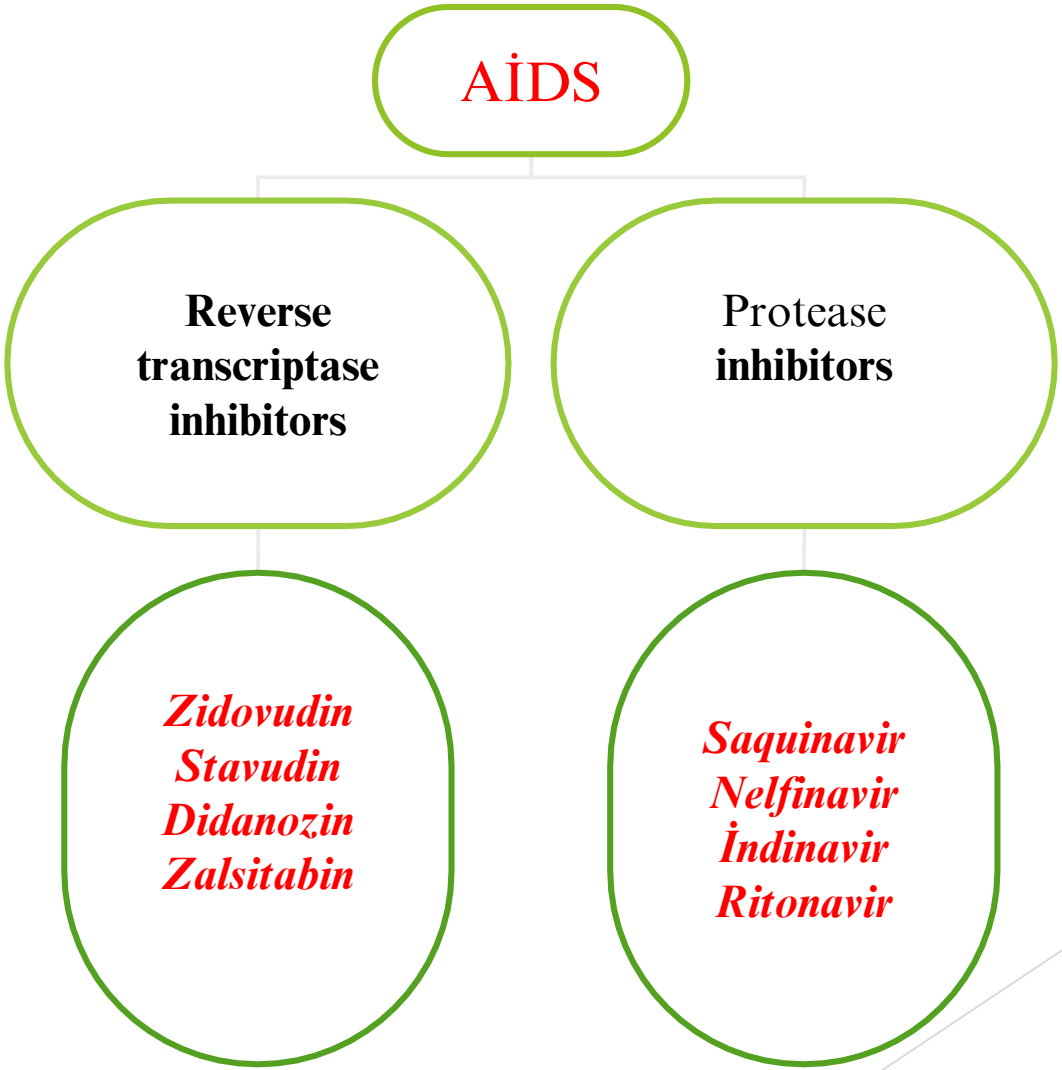
abacavir (Ziagen)
didanosine (Videx)
stavudine (Zerit)
delavirdine (Rescriptor)
lamivudine (Epivir)
tenofovir (Viread)

Antiretroviral Drugs

- **Protease inhibitors (PIs)**
  - Inhibit the protease retroviral enzyme, preventing viral replication
  - Examples:

amprenavir (Agenerase)
indinavir (Crixivan)
nelfinavir (Viracept)
ritonavir (Norvir)
saquinavir (Invirase)



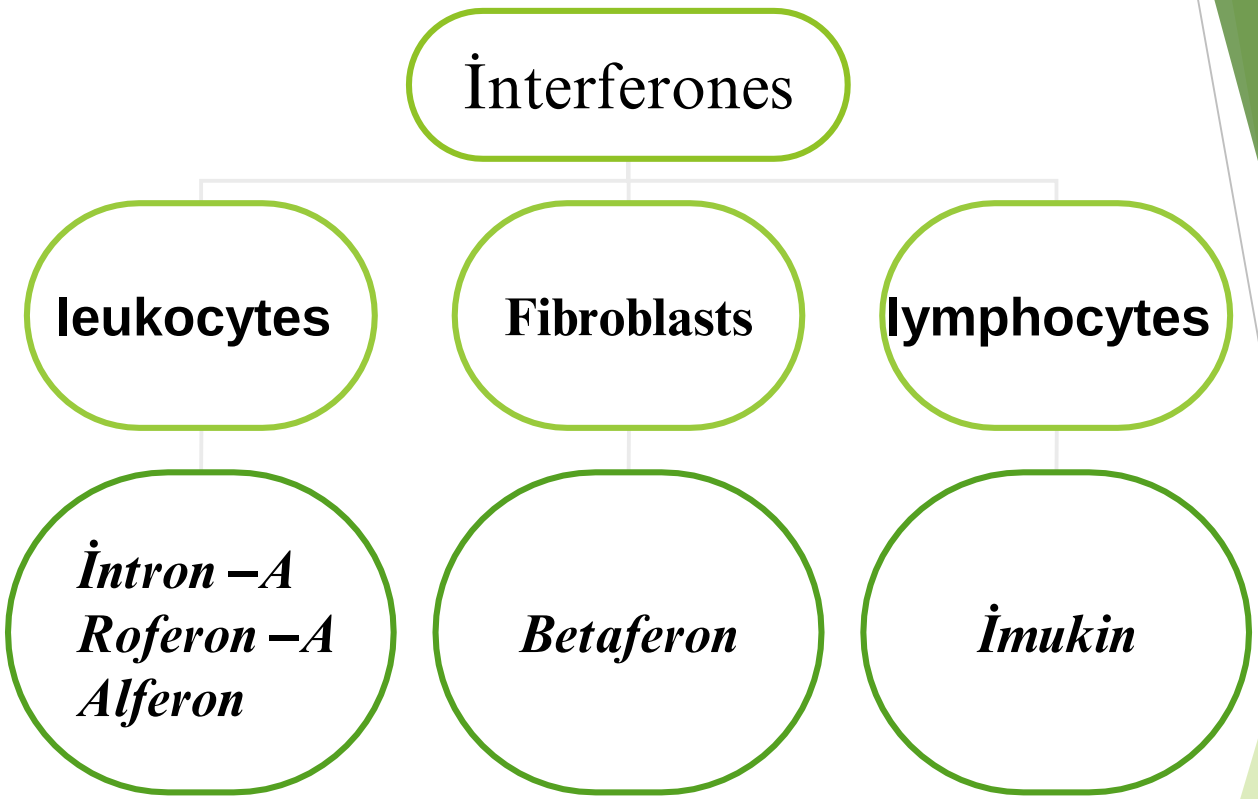

AİDS
Reverse transcriptase inhibitors
Protease inhibitors
Zidovudin
Stavudin
Didanozin
Zalsitabin
Saquinavir
Nelfinavir
İndinavir
Ritonavir

Herpes simplex viruses

Asiklovir
Valasiklavir
Vidarabin
Trifluridin
İdoxuridin

Influenze

Midantan
Remantadin
Arbidol
Oxolin


İnterferones
leukocytes
Fibroblasts
lymphocytes
İntron –A
Roferon –A
Alferon
Betaferon
İmukin

Antimalarials

1. Quinolin derivatives

A. 4-aminoquinolin derivatives

Quinin, Chinqamin, Meflochin

B. 8-aminoquinolin derivatives

Primachin, Chinosid

2. Diaminopirimidin derivatives

Chloridin

3. 9-aminoakridin derivatives

Akrichin

4. İzopropilbiquanin derivatives

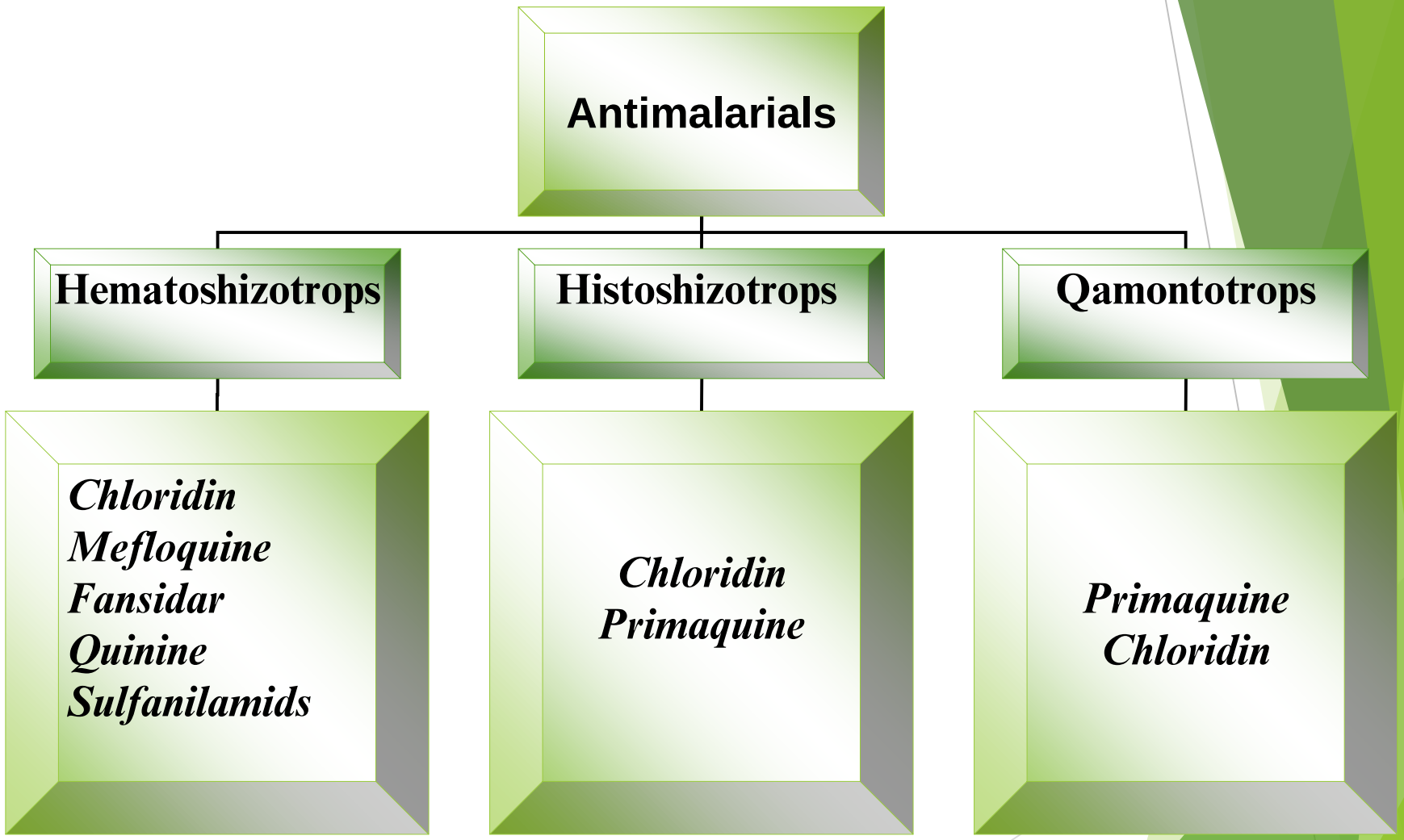
Biqumal

5. Sulfanilamids and sulfon

Sulfazin, Sulfapiridazin, Sulfadimetoxin, Sulfalen, Diafenilsulfon

6. Combined drugs

Metakelfin, Fansidar


Antimalarials
Hematoshizotrops
Histoshizotrops
Qamontotrops
Chloridin
Mefloquine
Fansidar
Quinine
Sulfanilamids
Chloridin
Primaquine
Primaquine
Chloridin

Treatment of amebic infection

- **1. Drugs acting in the intestine**
- **Qu*iniofon, Vioform, Diyodoxin***
- **2. Drugs acting in the intestine and intestine wall**
- ***Tetracyclines***
- **3. Drugs acting in the liver**
- ***Quinqamin***
- 4. **Drugs acting in the liver and in the intestine wall**

  Emetine hydrochloride
- **4. Drugs acting in all localization of amebic infection**

  Metronidazol

Treatment of trichomonadoz

- *Metronidazol*
- *Tinidazol*
- *Trichomonasid*
- *Furazolidon*

Treatment of leishmaniasis

- *Solusurmin*
- *Sodium stiboqlukonat*
- *Akrichin*
- *Monomisin*
- *Metronidazol*

Treatment of tripanosomoz

Melarsoprol
Pentamidin
Suramin
Primachin

Treatment of toxoplazmoz

Chloridin
Sulfanilamides

Treatment of lyamblioz

Metronidazol
Aminochinol
Furazolidon

Mycoses are most commonly due to *dermatophytes,* which affect the skin, hair, and nails following external infection. *Candida albicans,* a yeast organism normally found on body surfaces, may cause infections of mucous membranes.

Imidazole derivatives inhibit ergosterol synthesis. This steroid forms cytoplasmic membranes of fungal cells, analogous to cholesterol in animal plasma membranes. Fungi exposed to imidazole derivatives stop growing (fungistatic effect) or die (fungicidal effect).

Miconazole is given locally, or systemically by short-term infusion.

Ketoconazole is well absorbed and available for oral administration. Adverse effects are rare; however, the possibility of fatal liver damage should be noted. Remarkably, ketoconazole may inhibit steroidogenesis (gonadal and adrenocortical hormones**).**

Fluconazole and **itraconazole** are newer, orally effective **triazole** derivatives.

The **polyene antibiotics,** amphotericin B and nystatin, are of bacterial origin. They insert themselves into fungal cell membranes and cause formation of hydrophilic channels. The result increase in membrane permeability, accounts for the fungicidal effect.

Amphotericin B is active against most organisms responsible for systemic mycoses. Because of its poor absorbability, it must be given by infusion, which is, however, poorly tolerated (chills, fever, CNS disturbances, impaired renal function, phlebitis at the infusion site). Applied topically to skin or mucous membranes, amphotericin B is useful in the treatment of candidal mycosis. Because of the low rate of enteral absorption, oral administration in intestinal candidiasis can be considered a topical treatment.

Nystatin is used only for topical therapy

Anthelmintics

NEMATODOZ

intraenteric

- Askaridoz
- Enterobioz
- Trichosefalyoz
- Ankilostamidoz
- Stronqiloidoz
- Trichostronqiloidoz

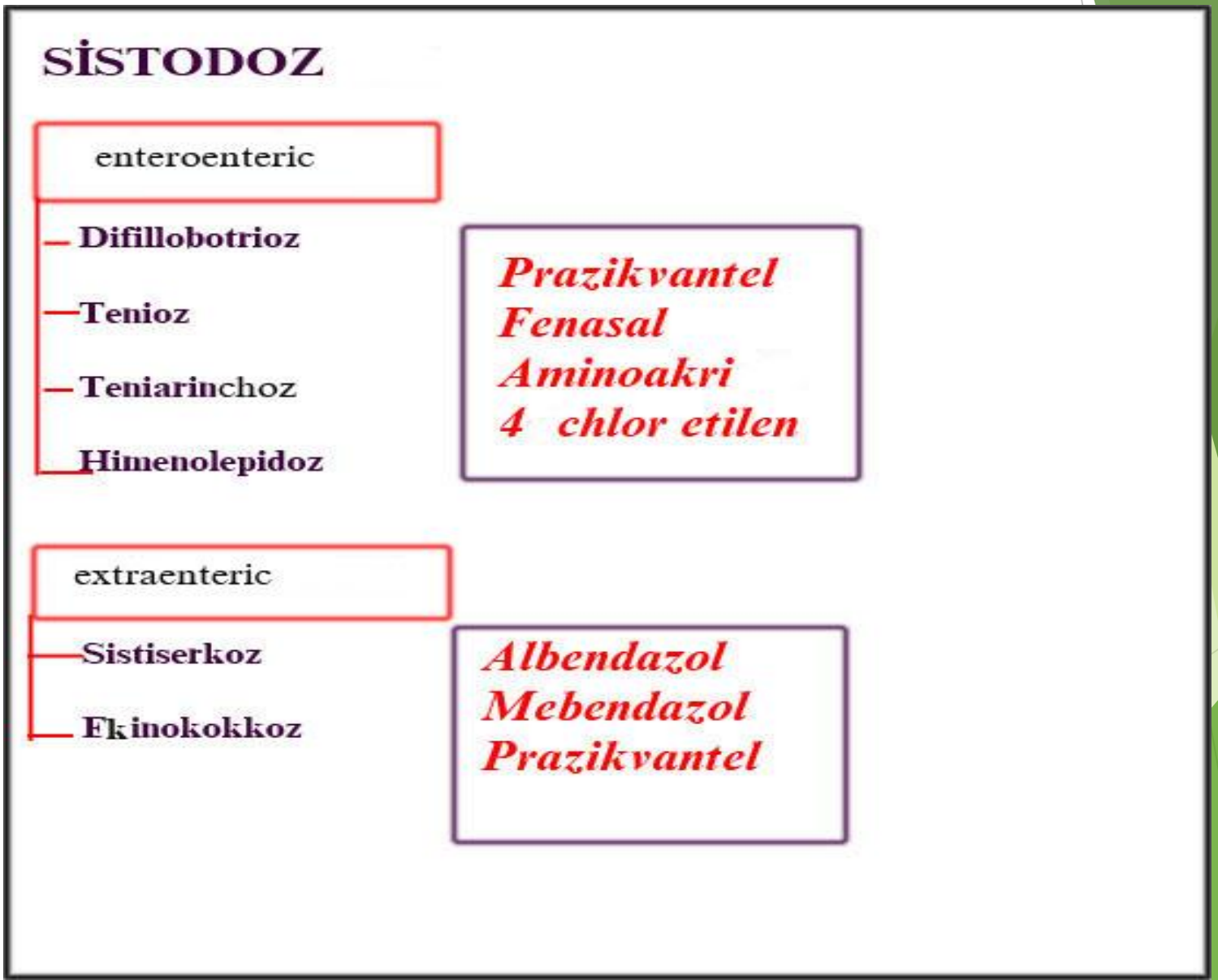
Albendazol
Mebendazol
Levamizol
Pirantel pamoat
Naftamon
Prazikvantel
Piperazin pamoat
Pirvinium pamoat

extraenteric

- Filyaritoz
- Trichinelez

İvermektin
Ditrazin sitrat
Mebendazol

Anthelmintics



Anthelmintics