Classification.

1. Organism →
   - Antibacterial.
   - Antifungal.
   - Antimalarial
   - Antiviral.
2. chemical structure.
   - β lactams
   - Aminoglycosides.
   - Tetracyclines.
3. Source.
   - → from microorganism → Antibiotic.
   - → other - non-antibiotic.

All antibiotics are antimicrobials but all antimicrobials are not antibiotics. (plant source, lab synthesis are not antibiotics)

4. Activity.

Static	Cidal.
• we give it in normal patients.	• give it in immuno-supressed patients who doesn't have enough Immunity.
→ It works by decreasing/stopping multiplication.	↓ eg HIV patients on corticosteroids.
→ So our Immune system can work & kill the remaining.	• their immunity is not enough and cannot work so we can it entirely.

Static

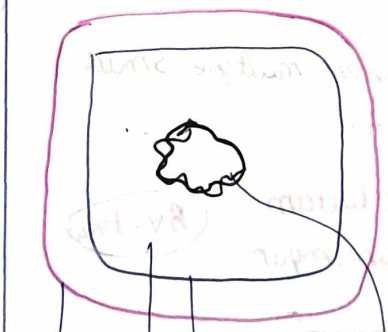
- protein synthesis (-) eg:
  - tetracycline
  - chloramphenicol
  - Macrolides
  - Linezolide.
- Sulfonamide.

Cidal

eg (BENAFA)

- β lactams:
  - penicillin, cephalosporin
  - Carbapenem
  - Monobactam
- Vancomycin.
- Fluoroquinolone.
- aminoglycosides (gentamycin).

(5) Based on mechanism of action.



1. drugs acting on cell wall.
2. on cell membrane.
3. on DNA
4. on protein.
5. on metabolism.

Antimicrobial resistance.

Natural -

mycoplasma - resistant to cell wall synth (-). due to lack of cell wall.

C. krusei - Fluconazole.

Acquired

Initially they were sensitive but later developed resistance.

reasons for resistance (5)

1. Enzymes → inactivating E.
   - A - aminoglycosides (A)
   - B - β lactams (B)
   - C - chloramphenicol (C)
2. Altered target.
   - MRSA
   - VRSA
   - FQ.

   } they alter the target of antibiotic.

MRSA → methicillin bind to transpeptidase & work so, they alter transpeptidase.

FQ → change DNA gyrase.

3. Development of efflux pumps for tetracyclines. (TT)
   - tetracycline.
   - tigecycline.
4. ↓ permeability for Aminoglycosides.
5. altered metabolism for sulfonamides.

- **Sulfonamides :**

- Inhibit folic acid synthesis of bacteria.

- In resistance, they change metabolism and instead of synthesising, they start to take up folic acid directly.

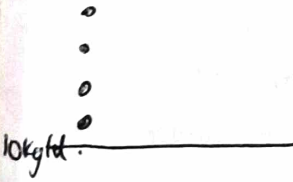
Transfer of Resistance

- Vertical → Mutation.
- Horizontal → Conjugation, transduction, transformation.

Special pharmacokinetics

Cidal drugs.

- Conc-dependent killing (CDK)
- time dependent killing.



CDK → as conc ↑, killing ↑ (number of).

→ AG & FQ.

- So we try to attain max plasma conc. within some limits.
- so we give high doses - less freq.

100 mg TDS ✗

300 mg OD ✓ preferred.

so that plasma conc. suddenly increased to max plasma conc.

TDK.

as plasma conc. increases, it doesn't increase more killing.

- It works as long as it is there.

so give multiple small doses.

- β lactam
- vancomycin

BV-time.

AUC dependent killing.

dependent on product of.

Conc x time → AUC

daptomycin
Newer FQ – Moxycycline.

post antibiotic effect

~~the time for which~~ persistent suppression of bacterial growth even when plasma concentration falls below MIC for a period of time.

GT

- all Long pae.

gm -ve

short-most.

long pae →

① DNA ⊣ FQ.

② protein sy (-) tetra macrolides AG.

③ Carbapenems.

Short pae.

β lactams except (carbapenem)

vancomycin.

(BV-short lumpen)

pseudomembranous colitis.

(URG reason @ 3d)

Organism	Not effective	DOC	Remarks
1. pseudomonas	VANCOMYCIN	ceftazidime + aminoglycoside	Vancomycin only → G(+)ve Pseudomonas - G(-)ve
2. MRSA	Any β lactams [Except 5th Gen cephalosporin]	Vancomycin. Ceftriaxone.	Not effective ans very Impt
3. Enteric fever	Aminoglycosides		
4. Anaerobes	Aminoglycosides	clindamycin - Lung, Brain abscess metronidazole - GIT infection.	as need O2 for entry, So for anaerobes, it won't work.
5. Mycoplasma.	cell wall synth. (-) ∴ β lactam Vancomycin	Azithromycin	

† food interferes with absorption of ciprofloxacin

* Levo – 100% B.A.

Ciprofloxacin

- most potent 1st gen.
- potent enzyme ⊖

D/I

- antacids, iron, Ca ↓ absorption of cipro. due to chelation
- ↑ theophylline, warfarin toxicity.

C/I

→ pregnancy & lactation.

→ children < 18 years. - cartilage damage.

Special

- only FQ with good activity against pseudomonas.
- DOC for anthrax ~~prophylaxis~~ exposure.

USES

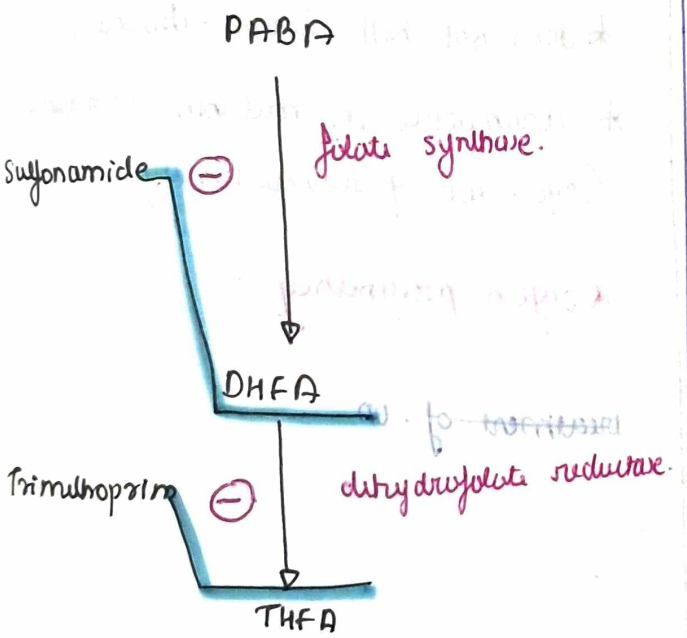
- UTI
- Prostatitis
- travelers diarrhea, Enteric fever.
- ☆ Anthrax (post exposure)
- ☆ pseudomonas infection.
- chlamydia, Gonorrhea, chancroid
- cystic fibrosis

COTRIMOXAZOLE {1:5 = 1:5}

The fixed dose combination of.

Sulfamethoxazole & Trimethoprim is called COTRIMOXAZOLE.

MOA



- Sulfamethoxazole MOA → earlier said.
- Trimethoprim. → ⊖ selectively bacterial DHF reductase.

USES

* UTI
* Respiratory tract infections.
* pneumocystis jirovecii
* chancroid.

ADR

- SJS, TEN.
- Megaloblastic anemia.
- Teratogenic - Kernicterus.
- ~~Rhabdomyolysis~~

Resistance

DHReductase enzyme having lower affinity.

C|I

pregnancy

Spectrum

.many g- & g+ organism

* Strep
* staph
* e.coli
* C. diphtheriae.
* E.coli
* pne. jirovecii

Sulfonamides

- short acting – Sulfadiazine.
- long – Sulfadoxine.
- special purpose – sulfasalazine.

Sulfa salazine.

DOC : Nocardia.

- Broad s. # Bacteriostatic.
- all sulfonamides have the same MOA

MOA

p-amino benzoic acid + Pteridine.

⊖ Sulfonamides

Dihydropteroic acid.

dihydrofolic acid (DHFA)

THFA.

purines

DNA.

Spectrum Broad.
- S. pyogenes.
- NOCARDIA – DOC
- chlamydia
- many g(+ve & g(-ve)
- Toxoplasma
- pn. jirovecii
- " static

" Sulfonamide are structurally similar to PABA"

Resistance
1. ↑ PABA synthesis
2. folate synthase enzyme having low affinity for sulfonamides
3. alternate pathway in folate metabolism.

MOA

Sulfonamides Competitively Inhibit

Union of PABA with pteridine residue.

prevents formation of Dihydropteroic acid.

⊖ DHFA, THFA formation.

PK
- oral absorption
- distribution – widely.
- metabolism – liver
- excretion – kidney.

USES
- DMARD in rheumatoid arthritis
- first line in ulcerative colitis

ADR
- SJS, TEN
- Kernicterus in neonates – displace Bb from albumin.

C/I pregnancy

- Hemolysis in G6PD deficient patients.