* Antimicrobial Drugs main modes of Action:

1] Inhibitors of cell wall synthesis:

a) Penicillins:

6 aminopenicillanic acid. + Differ in the Side chain Attached to the amino group.
* the main active ingredient and the Bioactive material ⇒ β. lactam Ring.
+ Penicillin Resistante Bacteria Produce an enzyme called β. lactamase → Hydrolise the Bond in the Ring of β. lactam.
→ what is the mode of Action for penicillin?
↳ Blocking the enzyme that catalyzes Transpeptidation → (Peptide Bonds in the cell wall) ⇒ No Peptide Bonds will be formed.
↳ Prevent the complete synthesis of cell wall.
↳ Binds to Periplasmic Proteins.
* Naturally occuring penicillin → Produced by microorganisms.
* Penicillin G, V → gram Positive Resistance → Narrow spectrum
* Semisynthetic penicillins → Broad spectrum + it has Bulky Side → makes it more difficult to β-lactamase to destroy it.
* Aminopenicillin (Ampicillin) → Gram neg Resistance.

b) Cephalosporins:
Has the Same Structure and Function of Penicillin.
→ used for Patients that are allergitic to Penicillin. + for species that are Resistance to Peni

c) Vancomycine:
MRSA → Multicyclic Resistance Agent → Resistance to most of Antibiotics. + used as side + last Drug choice.
glycopeptide antibiotic + Important for the treatment of antibiotic Resistance Staphylococcus + enterococcal.

2] Protein Synthesis Inhibitors:
the main thing for protein is the Ribosome, So this type of antibiotics interferes with the Ribosomal function and units.
→ Bind specifically to the Bacterial Ribosome and target different steps: i) Translocation ii) Peptide Bond formation iii) mRNA Reading iv) Aminoacyl-tRNA Binding

a) Aminoglycosides:
→ Cyclohexane Ring + aminosugars.
→ Bind to 30s Ribosomal Subunit + interferes with Proteins synthesis by directly inhibiting the Proccess → Misreading of the mRNA.

b) Tetracyclines:
4 Rings and it may has Side chains.
Broad spectrum + Bacteriostatic (inhibition) + target 30s Ribosomal unit.

c) Macrolides:
12 - 22 Carbon lacton ring linked to one or more Sugars (Erythromycin)
Broad Spectrum + Bacteriostatic + 50s Ribosomal unit (inhibit Protein elongation) + for Patients that are allergic to Penicillin

d) Chloramphenicol:
Chemically synthesised + 50s Ribosomal Subunit
Inhibition of Bacterial Protein synthesis + So toxic

3] Metabolic Antagonistic → Produce Metabolite that are structurally the same in eukaryotics.
→ Acts As Antimetabolite → Antagonize / Block the function of metabolic Pathway (inhibit the use of metabolites)

a) Sulfonamids OR Sulfadrugs:
looks similar to compounds used for the synthesis of Folic Acid. + Para aminobenzoic acid Analog.
PABA → used for synthesis of folic Acid + made by many pathogens
* Selectivly toxic → Competitive inhibition of folic acid synthesis enzymes.

b) Trimethoprim:
interfers with Folic acid Production. + Broad
can be combined to Sulfa drugs → increase efficacy of treatment. (the combination Blocks 2 pathways for the Production of Folic acid)

4] Nucleic acid synthesis Inhibition:
+ Inhibit DNA Polymerase + topoisomerase. + RNA Polymerase. (Not as effective as others).
Because Bacteria + eukarya do not differ. greatly in the way of synthesis nucleic acid.

a) Fluoroquinolones:
Effect DNA syntheses (unwinding of DNA Strands)
Contains the 4 quinolone + Inhibit Bacterial DNA Gyrase + topoisomerase.
→ Broad spectrum → Cidal.

* Antiviral Drugs:

- Once viruses enter a Host cell they Start Replicating and making their own structures, so these Antiviral Drugs Are challenging Because viruses are Nonliving organisms.
→ So the antiviral drugs only limit the duration of illness. (Inhibit viruses specific enzymes + life cycle Process).
* they are not Preferred Because they also effect the Host cell.

1] Antiviral Drugs for influenza:
a) Tamiflu Anti: influenza agent / neuraminidase Inhibitor _ Shorten course of illness.
b) Acyclovir + Vidarabin: Herp Infection + shingels.
c) Ganciclovir: treat systemic cytomegalovirus illness.
d) Foscarnet: for the Resistance of acyclovir + ganciclovir. → DNA Genome.
↳ Herps + cytomegaloviruses.

* Broad Spectrum Antiviral Drugs:
_ Cidofovir: (inhibits viral DNA Polymerase)

* Anti HIV Drugs:

1) Nucleoside Reverse trascriptase inhibitors:
Interfere with Critical steps in the Replication Process.
2) Protease inhibitors (PIs).
Block activity of HIV Protase for Producing the viral Protein.
3) Nonnucleoside Reverse trascriptase Inhibitors:
Prevent HIV DNA synthesis → by Binding to and inhibiting the viral Reverse Trascriptase enzyme.
4) Integrase Inhibitors:
Prevent cooperation of HIV Genome to the Host's cell Genome
5) Fusion inhibitors:
Prevent the entry of HIV into the Host cell.

* Antifungal Drugs: least effective Because the similarity of the eukaryot + fungal cells
Used on outside + we may use combination of Drugs

Treating Mycoses:

1) Superficial mycoses (External)
Candida (Gastro intestal tract.
_ topical + oral.
Disturb Permeability + inhibit Sterol synthesis + Disturbs mitotic Spindle + may inhibit Protein-DNA synthesis

2) Systemic mycoses (internal)
Difficult to be controled
a) Amphotericin B: Bind to Sterol membrane
b) 5- flucytosin: RNA function disturbes.
c) fluconazol: low sideeffects

* Drug Resistance by Microbs types:

1] Intrinsic:
Mycoplasma lack the cell wall So it's Resistance to the B. lactam antibiotic.

2] Aquired:
when there is change in the Genome that Converts it from one that is sensitive to one that is Resistace.

3] Drug Tolerant Bacteria:
Ignore the Presence of Antibiotic → embeded in Biofilm So cannot Pentrate the Organisms.

* what are the mechanisms for Drugs Resistance:
- Modify the target of antibiotic
- inactivation
- Minimizing Cor.
- Increase Produce of metabolites.

* How the Resistace is detected
1) color change → when B. lactam is cats open Chromosore
2) PCR.

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