ANTIMICROBIAL AGENTS

Tishk International University
Faculty of Applied Science
Medical Analysis Department

Lecture – 5
Second Semester
05-04-2026

Dr. Sami Mamand

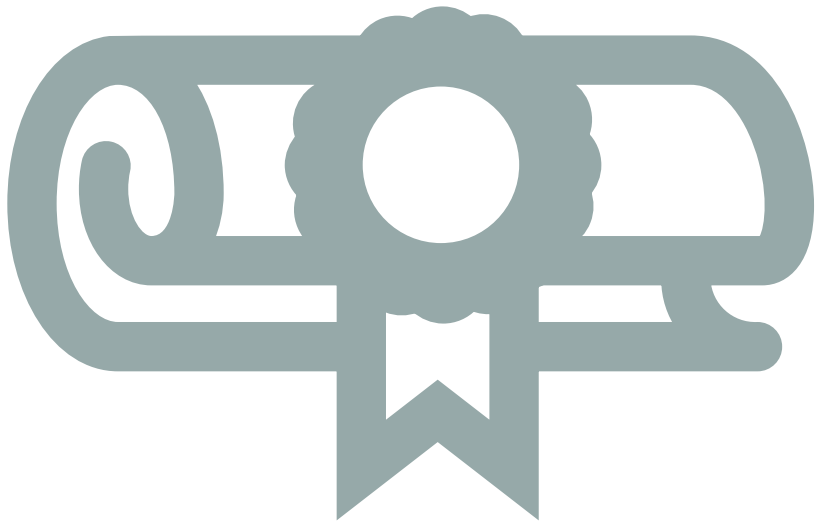
Course Description

This course introduces the fundamental principles of pharmacology, focusing on:

- Drug classification systems
- Mechanisms of drug action
- Pharmacokinetics (ADME)
- Pharmacodynamics
- Drug–drug interactions
- Toxicology and drug safety

Week	Topic
1	Introduction to Pharmacology
2	Pharmacokinetics (ADME)
3	Pharmacodynamics
4	Steroid & Non-Steroid Drugs
5	Nervous System Pharmacology
6	Cardiovascular Pharmacology
7	Antimicrobial Agents
8	Endocrine & Metabolic Drugs
9	Hematology & Chemotherapy
10	General Toxicology
11	Clinical Toxicology & Drug Safety
12	Student Presentations & Review

COURSE SYLLABUS



Outline

- Definition and introduction
- Classification
- Mechanism of action

Learning Objectives

Connect mechanism → drug → disease → resistance

- Define antimicrobial agents and selective toxicity
- Classify antimicrobial drugs based on mechanism and spectrum
- Explain molecular mechanisms of action
- Recognize mechanisms of antimicrobial resistance

Antimicrobial Agents

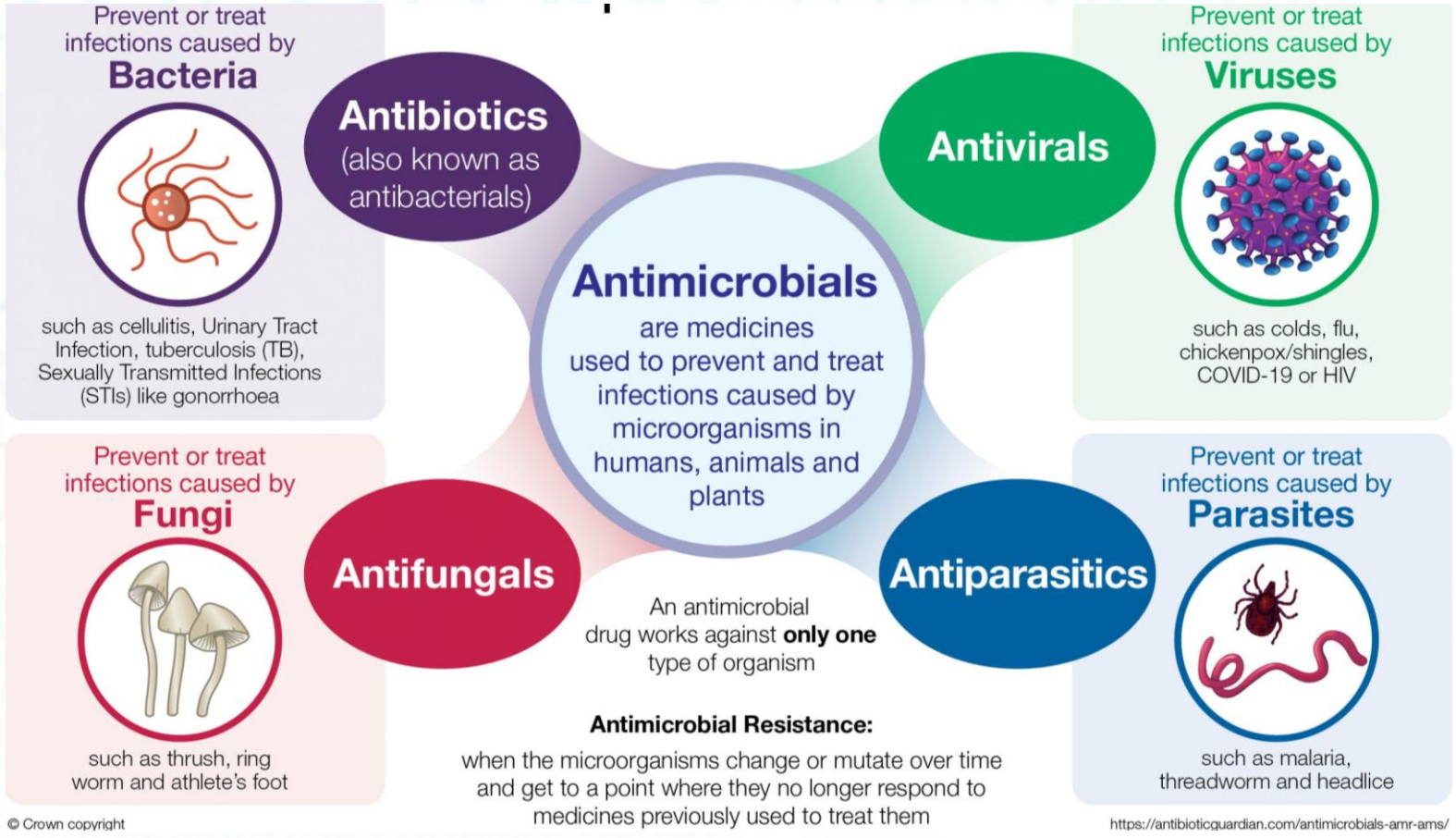
Introduction to Antimicrobial Agents

Chemical substances that:

- Kill microorganisms (microbicidal)
- Inhibit their growth (microbistatic)

Types:

- Antibacterial (antibiotics)
- Antiviral
- Antifungal
- Antiparasitic



Introduction to Antimicrobial Agents

Selective toxicity:

Targets structures unique to microbes:

- Cell wall
- 70S ribosome
- Unique metabolic pathways

Human cells lack:

- Peptidoglycan
- Bacterial ribosomes ⟶ ideal targets

Classification of Antimicrobials

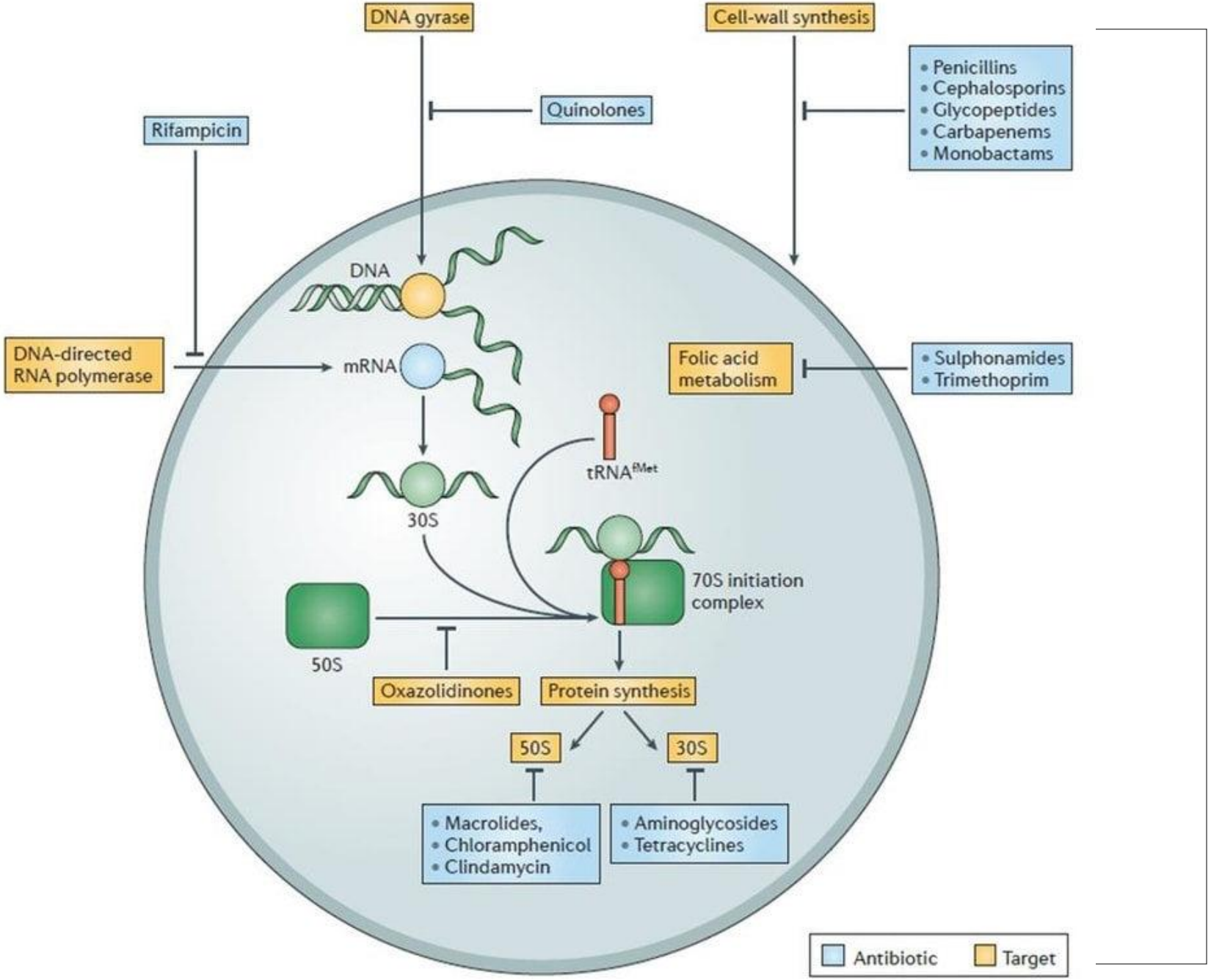
Based on target:

- Bacteria ⟶ Antibiotics
- Viruses ⟶ Antivirals
- Fungi ⟶ Antifungals
- Parasites ⟶ Antiparasitics

Based on activity:

- **Bactericidal**
  - Cause irreversible damage
  - Lead to cell death
- **Bacteriostatic**
  - Reversible inhibition
  - Depend on immune system

✓ Immunocompromised patients ⟶ need **cidal drugs**



Mechanisms of Action

Antibiotics exploit 5 major targets:

1) Cell wall synthesis
2) Protein synthesis (ribosome)
3) DNA replication
4) Metabolic pathways
5) Cell membrane integrity

Cell Wall Synthesis Inhibitors

Target:

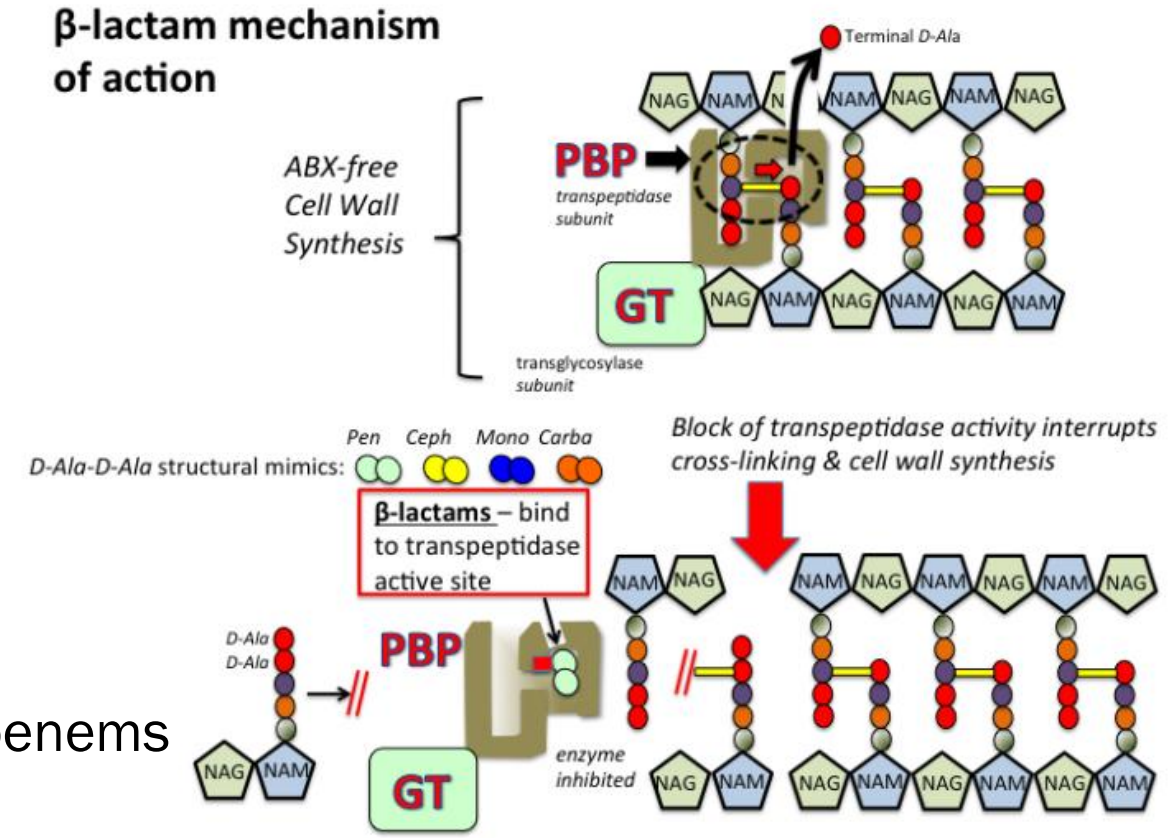
- Peptidoglycan (unique to bacteria)

Mechanism:

- Inhibit transpeptidase enzymes (PBPs)
- Prevent cross-linking ⟶ weak wall ⟶ lysis

Drug classes:

- β-lactams: Penicillin, Cephalosporins, Carbapenems
- Glycopeptides: Vancomycin



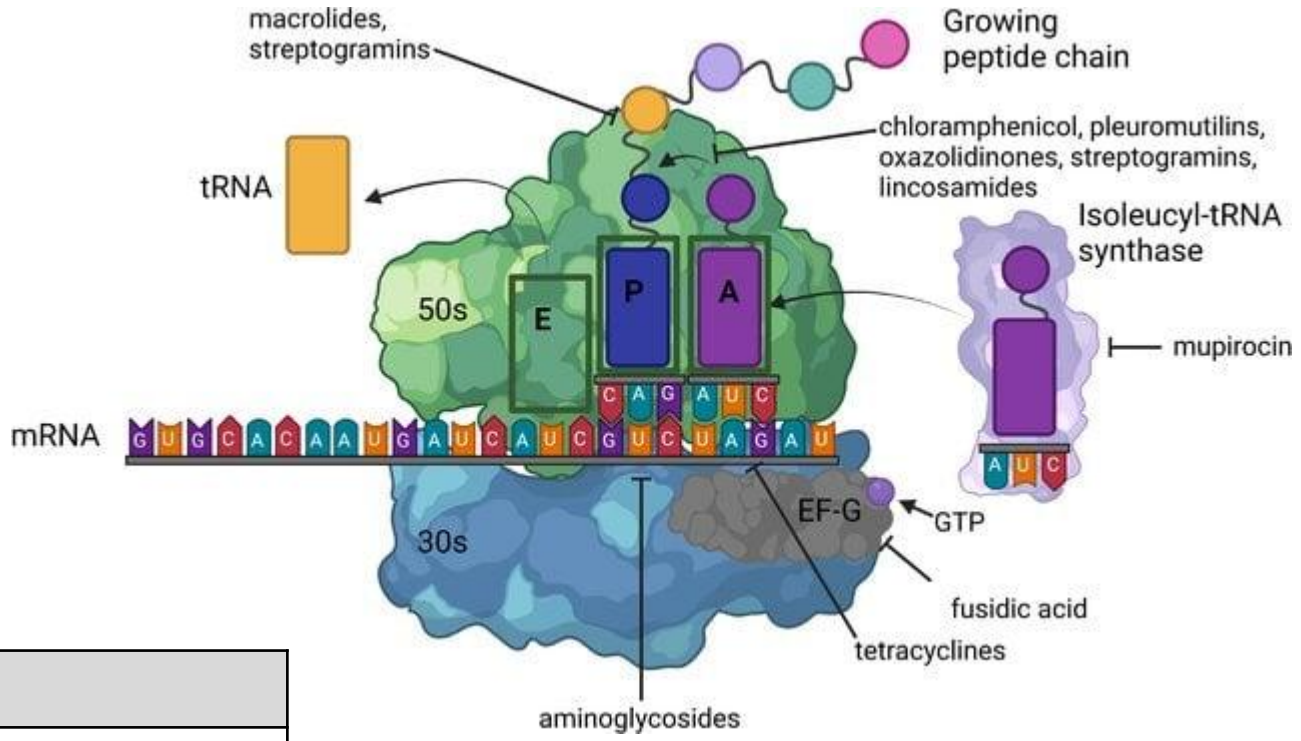
Protein Synthesis Inhibitors

Bacterial ribosome:

- 70S = 30S + 50S

Mechanisms:

- Block tRNA binding
- Prevent peptide bond formation
- Cause misreading of mRNA



Drug	Action
Tetracycline	blocks tRNA entry
Aminoglycosides	misreading mRNA
Macrolides	block elongation

DNA & RNA Synthesis Inhibitors

Fluoroquinolones:

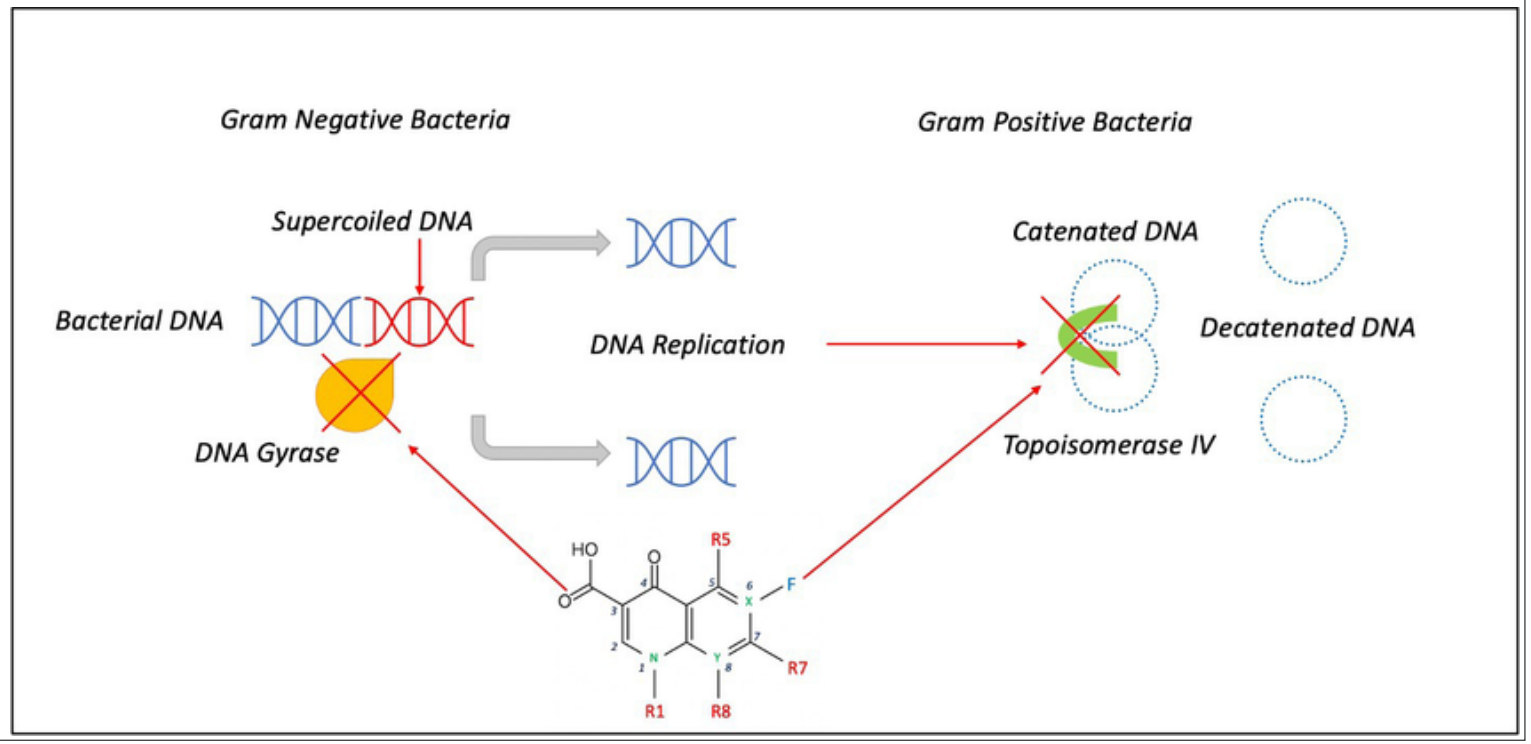
- Inhibit DNA gyrase/topoisomerase II
- Prevent DNA supercoiling

Rifampicin:

- Inhibits RNA polymerase
- Blocks transcription

Clinical importance:

- TB treatment
- Broad-spectrum infections



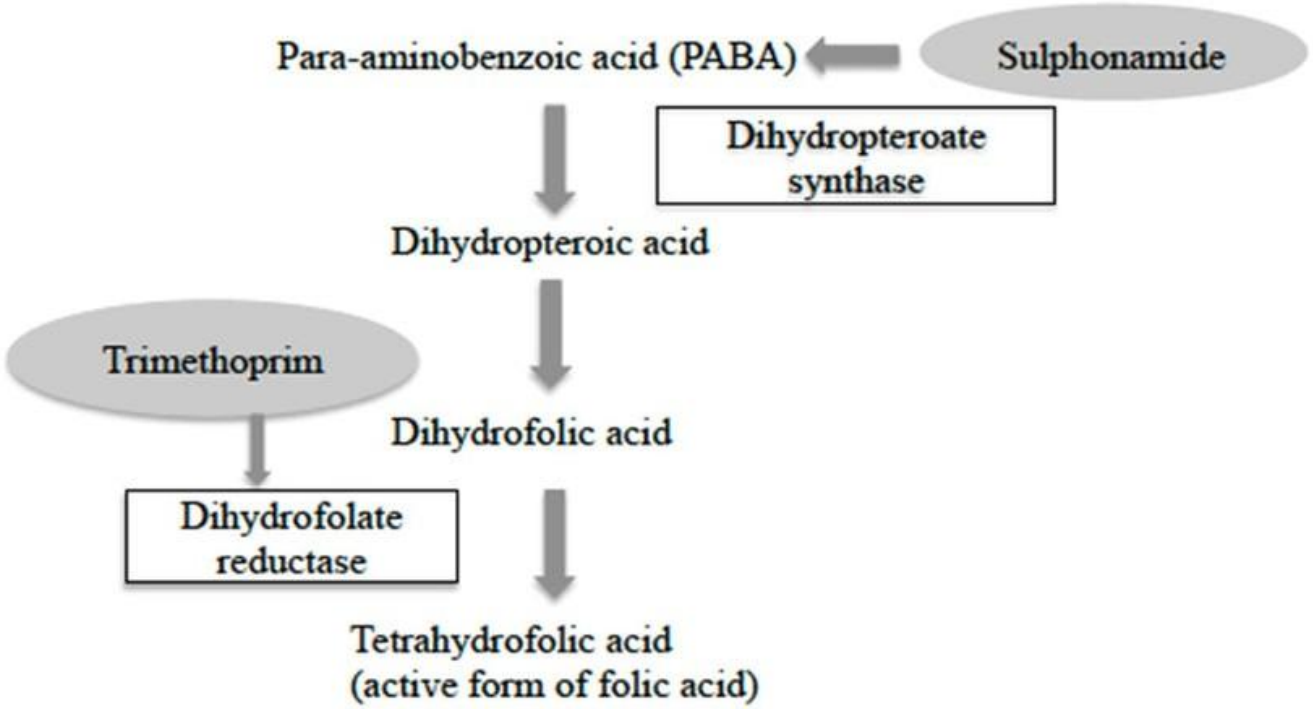
Metabolic Pathway Inhibitors

Target:

- Folic acid synthesis

Drugs:

- Sulfonamides ⟶ inhibit dihydropteroate synthase
- Trimethoprim ⟶ inhibit dihydrofolate reductase



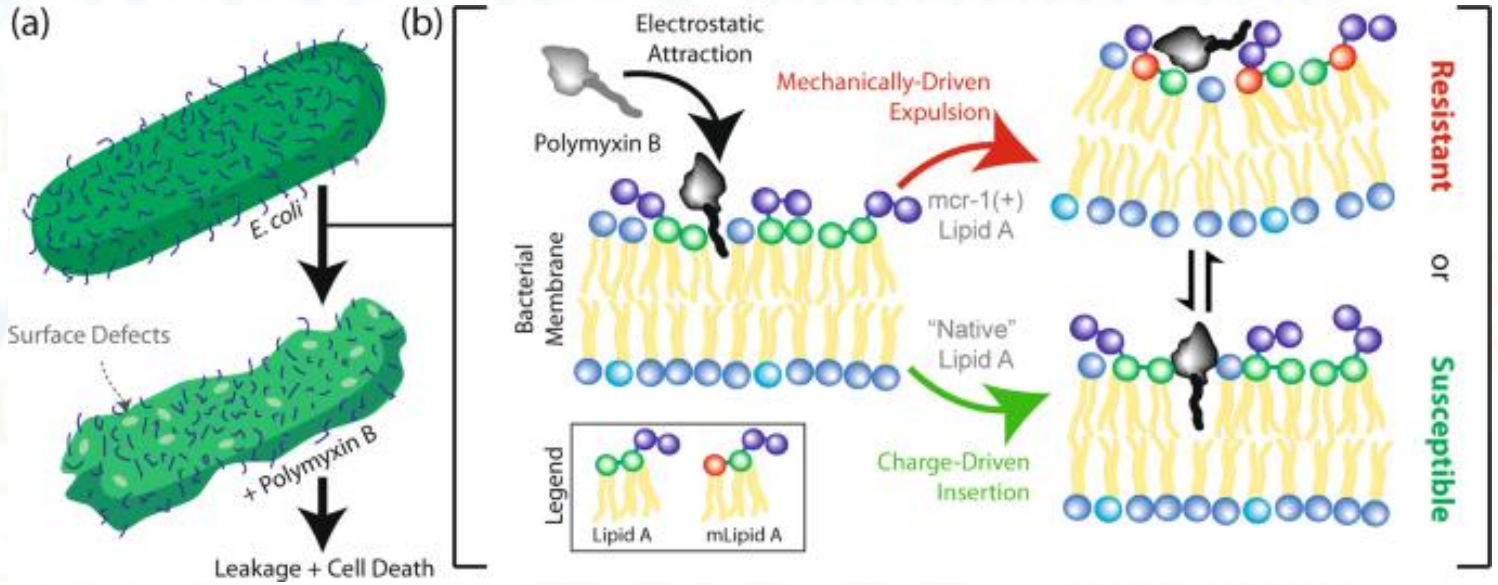
Cell Membrane Disruption

Mechanism:

- Disrupt phospholipid bilayer
- Cause leakage of ions and proteins

Drugs:

- Polymyxins ⟶ Gram-negative
- Amphotericin B ⟶ fungi (binds ergosterol)



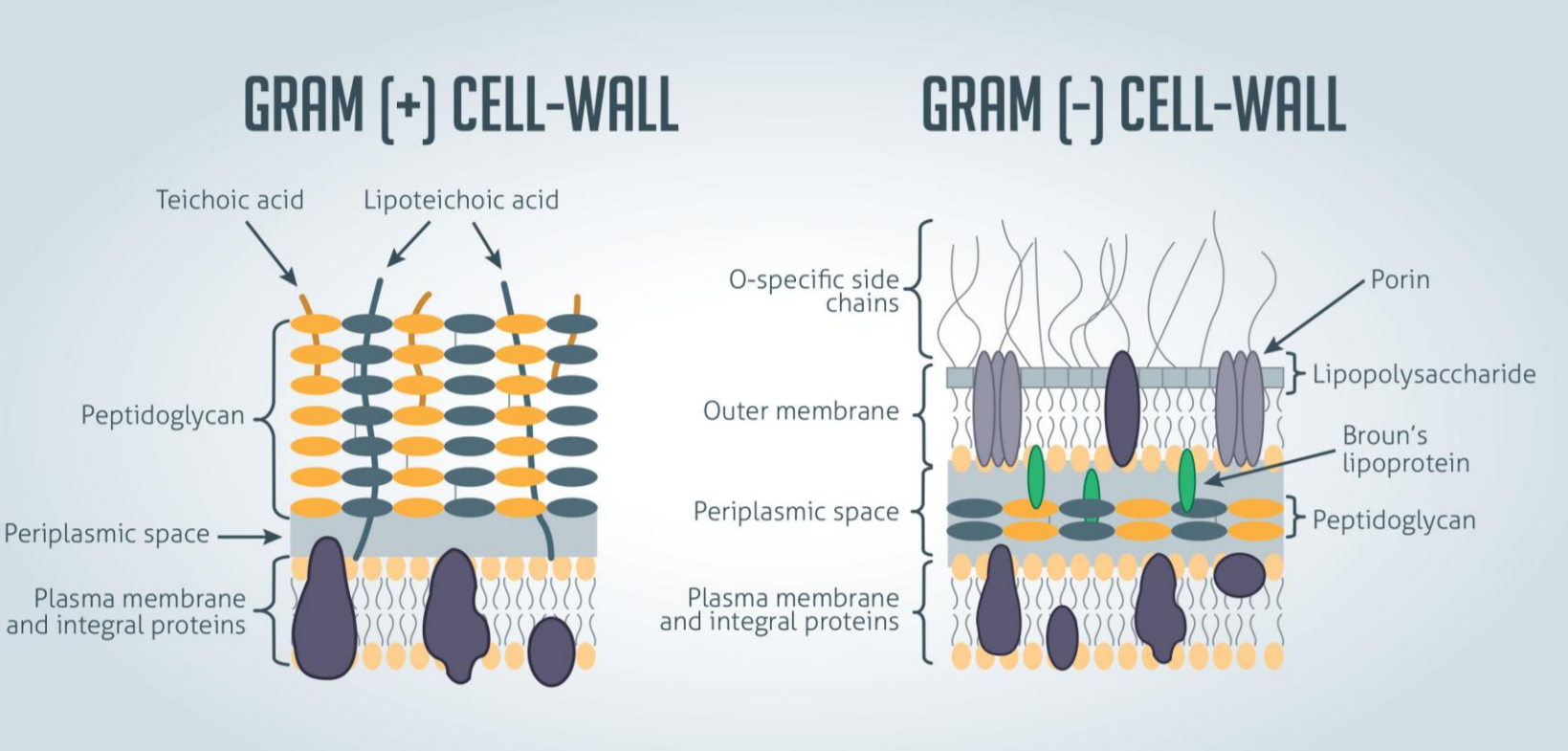
Spectrum of Activity

Narrow spectrum:

- Specific organisms
- Example: Penicillin

Broad spectrum:

- Multiple organisms
- Example: Tetracycline



Broad vs Narrow Spectrum Antibiotics

Feature	Broad-Spectrum Antibiotics	Narrow-Spectrum Antibiotics
Definition	Active against a wide range of bacteria	Active against specific types of bacteria
Target organisms	Gram-positive and Gram-negative	Usually Gram-positive or Gram-negative
Examples	Tetracycline, Amoxicillin-clavulanate, Ciprofloxacin	Penicillin G, Vancomycin
Clinical use	Empirical therapy (unknown pathogen)	Targeted therapy (known pathogen)
Effect on microbiome	High disruption (kills normal flora)	Minimal disruption
Resistance risk	Higher risk of resistance development	Lower risk
Use in practice	Severe infections, mixed infections	Confirmed infections (culture-based)
Side effects	More superinfections (e.g., C. difficile)	Fewer side effects

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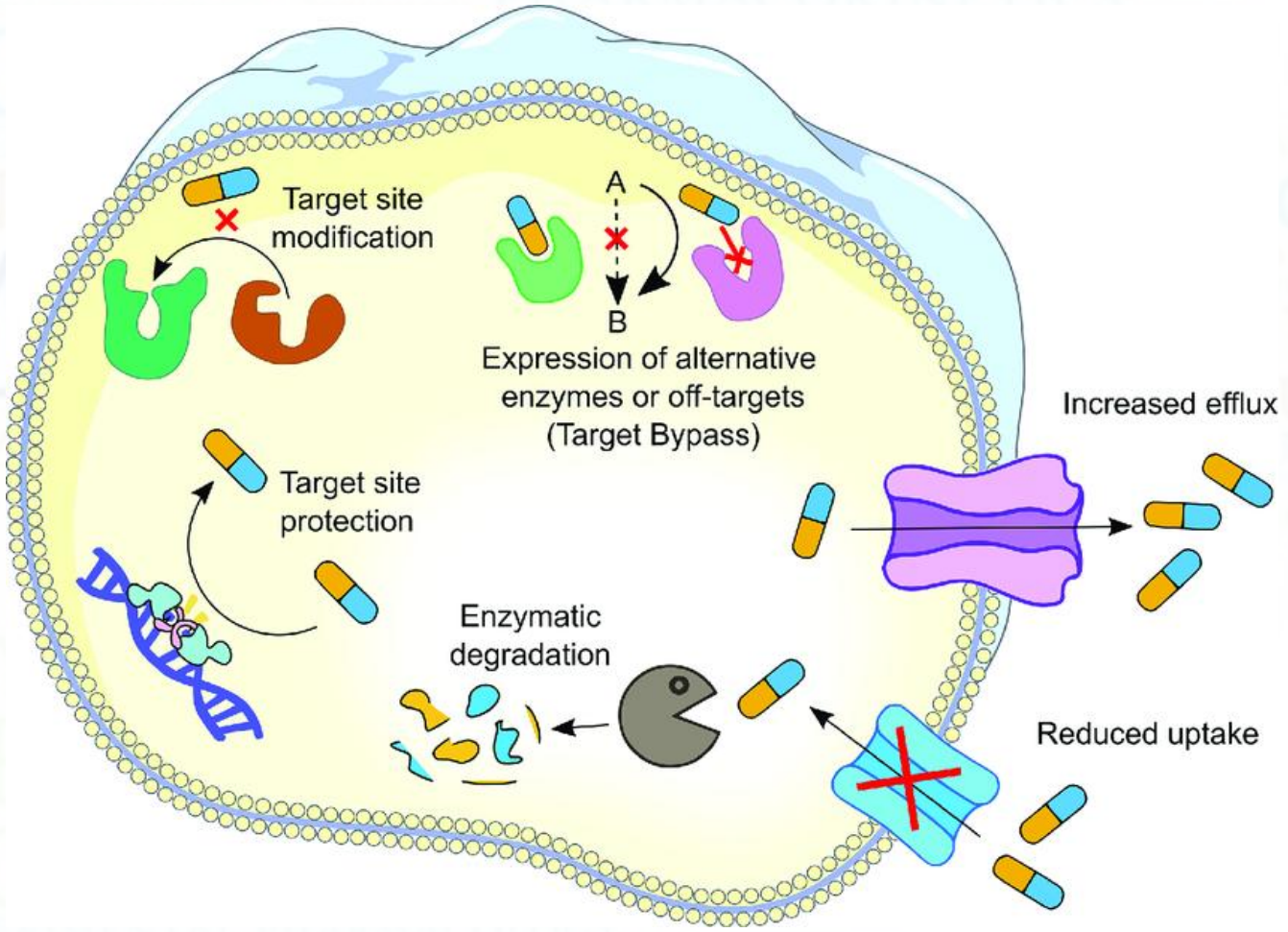
Antimicrobial Resistance

Mechanisms:

1. **Enzymatic degradation**
    1. β-lactamase
2. **Target alteration**
    1. MRSA ⟶ altered PBP
3. **Efflux pumps**
4. **Reduced permeability**



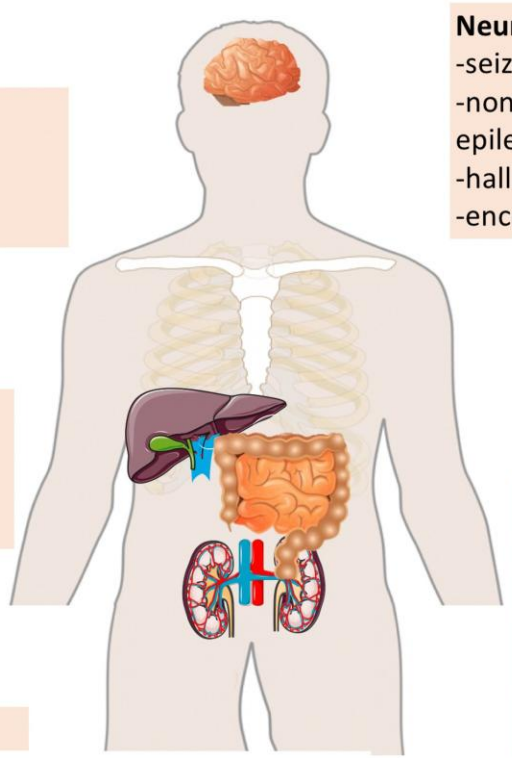
Principles of Antimicrobial Therapy

1. Accurate diagnosis
2. Culture & sensitivity
3. Appropriate drug selection
4. Correct dose & duration

Wrong drug = resistance + treatment failure

Adverse Effects

- Allergic reactions ⟶ immune response
- Nephrotoxicity ⟶ aminoglycosides
- Hepatotoxicity ⟶ metabolism overload
- Superinfection ⟶ microbiome disruption



Hematotoxicity
- Agranulocytosis
- Thrombocytopenia
- Neutropenia

Neurotoxicity:
-seizures
-non convulsivant status epilepticus
-hallucinations
-encephalopathy

Allergy

Mitochondrial toxicity

Hepatotoxicity:
- Drug induced liver injury
- Cholestatic Hepatitis
- Acute hepatitis

Nephrotoxicity:
-acute interstitial nephritis
-renal damage associated with drug induced haemolytic anaemia
-renal obstruction by crystallization

Clostridium difficile Infections

Microbiome changes

Combination Therapy

Advantages:

- Synergy
- Broader coverage
- Resistance prevention

Example:

- TB therapy:
  - Isoniazid + Rifampicin + others

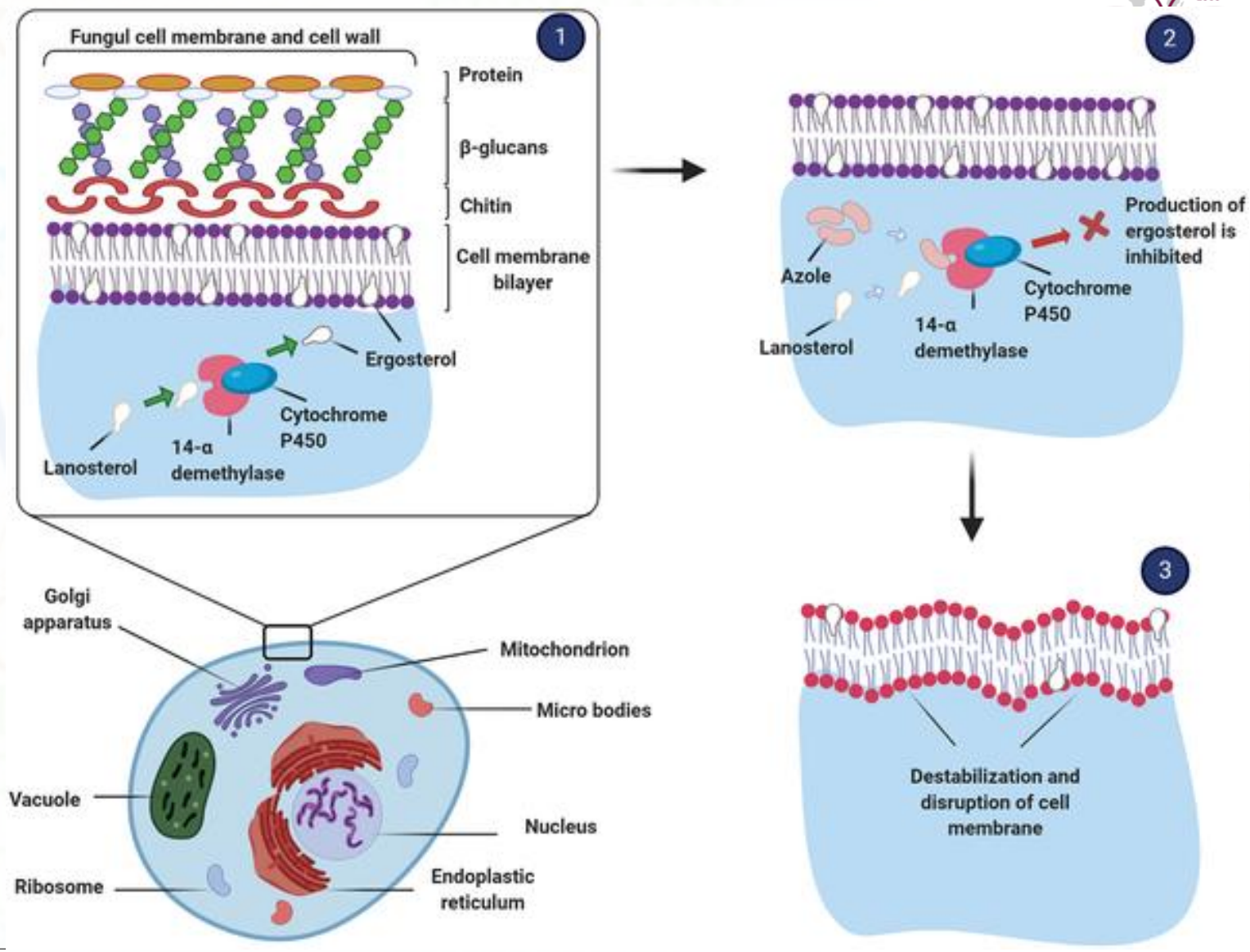
Antifungal Agents

Unique target:

- Ergosterol (fungal membrane)

Drugs:

- Azoles ⟶ inhibit synthesis
- Polyenes ⟶ bind ergosterol



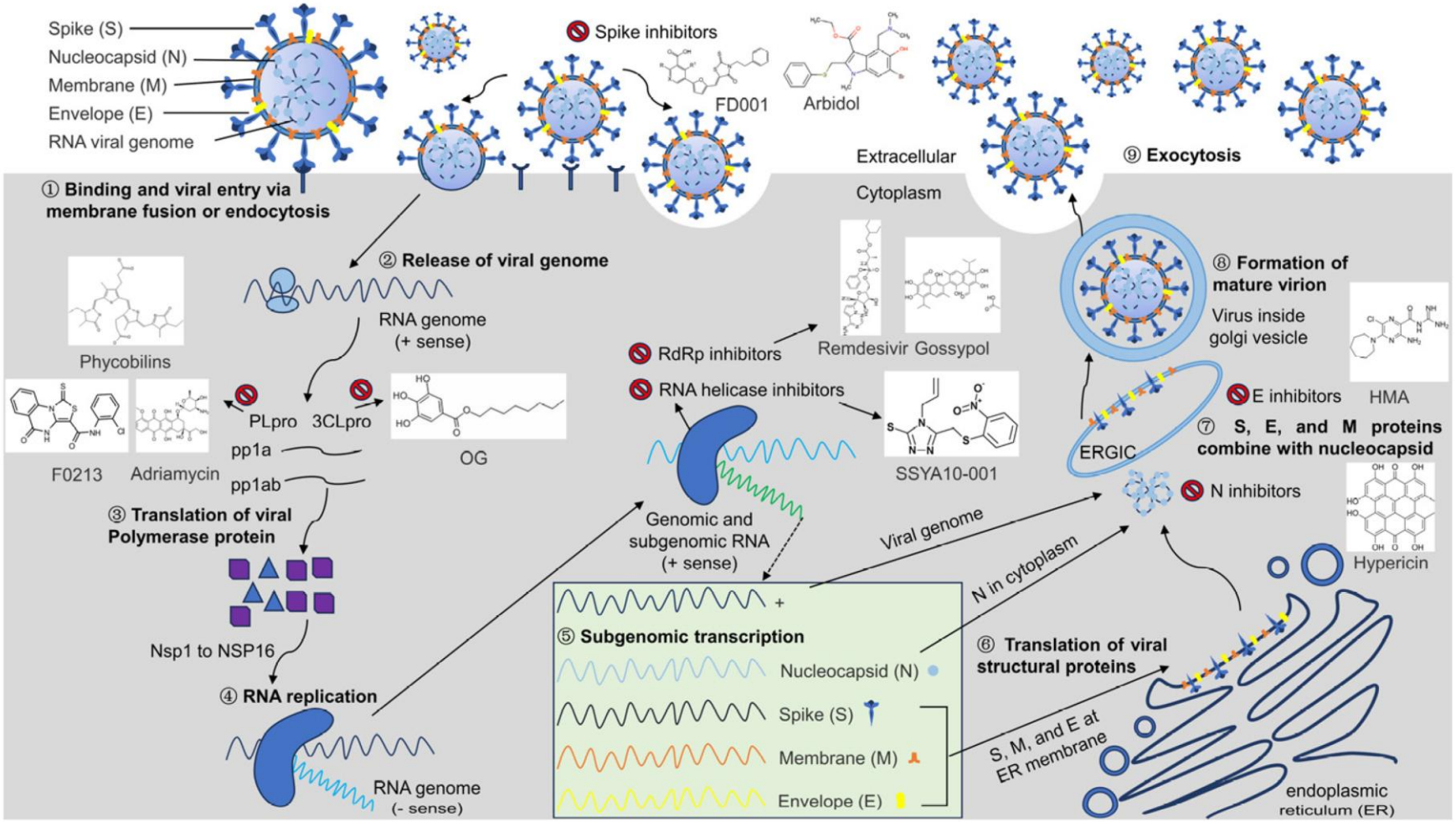
Antiviral Agents

Challenge:

- Viruses use host machinery

Targets:

- Entry
- Replication
- Assembly



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- World Health Organization