Penicillin targets wall building

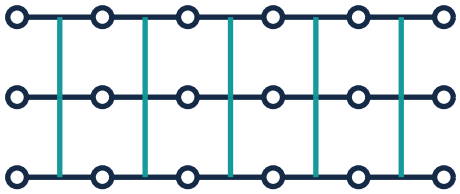
Antibiotics kill bacteria or inhibit their growth. Penicillin is one example: it interferes with the construction of a strong peptidoglycan cell wall.

1. KNOW THE STRUCTURE

Peptidoglycan (murein) consists of sugar chains connected by short peptide cross-links. This mesh surrounds the cell membrane and provides mechanical support. The wall and the membrane are different structures.

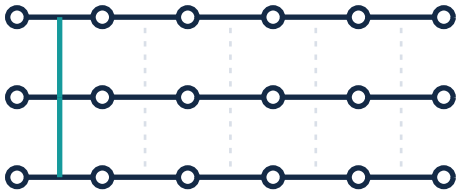
2. CROSS-LINKING MAKES THE MESH STRONG

Normal wall synthesis



Solid teal links join neighbouring sugar chains.

Penicillin present



Fewer new links form. Dashes show missing links, not bonds.

3. BLOCK THE ENZYME, NOT THE WATER

Penicillin binds to penicillin-binding proteins, including the transpeptidase enzyme that forms peptide cross-links. New peptidoglycan is therefore inadequately cross-linked. The wall is weakened as the bacterium grows.

The drawing is a simplified structural model, not the detailed chemical structure. The key relationship is enzyme inhibition followed by reduced cross-linking.

Penicillin targets and selective action: OpenStax, Microbiology 14.3.
Peptidoglycan structure: Essentials of Glycobiology, Eubacteria and Archaea.

Scope: Cambridge Biology 9700 syllabus, section 10.2, outcome 1, p. 30.

From weak wall to cell lysis

A wall helps a bacterium withstand internal pressure. Penicillin is most effective against susceptible bacteria actively growing and making new wall material.

1. FOLLOW THE CAUSAL CHAIN

1. **Cross-link formation is inhibited**

   The growing cell cannot build a sufficiently strong peptidoglycan mesh.

2. **The wall becomes mechanically weak**

   The cell membrane remains partially permeable; a weak wall gives less support.

3. **Water enters by osmosis**

   If the surrounding solution has a higher water potential than the cytoplasm, net water movement is into the cell.

4. **Pressure can cause lysis**

   The weakened wall may fail to restrain swelling, so the membrane ruptures and the cell dies.

2. WHY GROWTH MATTERS

A growing or dividing bacterium must add and remodel cell wall material. Blocking synthesis is especially damaging at this stage. Penicillin does not simply dissolve every pre-existing wall on contact.

3. STATE THE CONDITIONS

Use "susceptible bacteria" and "may lyse": not every bacterium responds to penicillin, and osmotic conditions affect bursting. The central target is wall synthesis, not osmosis itself.

Wall integrity and osmotic lysis: Essentials of Glycobiology.
Cell-wall synthesis inhibitors: OpenStax, Microbiology 14.3.

Scope: Cambridge Biology 9700 syllabus, section 10.2, outcome 1, p. 30.

Why not viruses?

A medicine needs an appropriate molecular target. Viruses do not have the bacterial structures and independent cellular processes that antibiotics attack.

1. COMPARE THE TARGETS

Feature	Bacterium	Virus
Peptidoglycan	Present in most bacterial cell walls.	Absent. A protein capsid is not a cell wall.
Ribosomes	Own 70S ribosomes make proteins.	No ribosomes; host ribosomes make viral proteins.
Cellular metabolism	Own enzyme-controlled cellular pathways.	Depends on a host cell; lacks independent cellular metabolism.

2. EXPLAIN ABSENCE, NOT SIZE

Penicillin cannot block viral peptidoglycan cross-linking because viruses make no peptidoglycan. More generally, viruses lack the bacterial targets of antibiotics. Being small or inside a cell is not the key explanation.

3. SELECTIVE DOES NOT MEAN SIDE-EFFECT-FREE

Human cells also lack peptidoglycan walls, helping explain penicillin's selective action. Nevertheless, antibiotics can cause adverse effects, including allergic reactions, and can affect beneficial bacteria.

Antiviral medicines are a different category. If an antibiotic is used for a bacterial infection alongside a viral illness, it treats the bacteria, not the virus.

Viral structure and host dependence: OpenStax, Biology 2e 21.1.
Antibiotic use, viral illness and adverse effects: CDC, Antibiotic Do's and Don'ts.

Scope: Cambridge Biology 9700 syllabus, section 10.2, outcome 1, p. 30.