

## Note on Penicillin's Mechanism of Action

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### EDITORIAL NOTE

Penicillins are antibiotics that were first discovered in *Penicillium* moulds, primarily *P. chrysogenum* and *P. rubens*. The majority of penicillins used in medicine are chemically synthesised from naturally occurring penicillins. Penicillin G (intramuscular or intravenous usage) and penicillin V (intramuscular or intravenous use) are the only two pure penicillins in clinical use (given by mouth). Penicillin was one of the first antibiotics to improve significantly against a wide spectrum of staphylococcal and streptococcal infections. They are antibiotics that belong to the  $\beta$ -lactam family. They are still commonly used today to treat a variety of bacterial infections, while many bacteria have developed resistance as a result of their widespread use.

Penicillin's chemical structure is triggered by a pH-dependent directed process involving a unique spatial arrangement of molecular components that can be activated by protonation. It can pass through bodily fluids, targeting and inactivating enzymes involved in gram-positive bacteria's cell wall construction while avoiding non-targets. Penicillin can shield itself from spontaneous hydrolysis in the body by remaining an anionic acylating agent that is only activated when it comes into contact with the target transpeptidase enzyme and protonated in the active core. The carboxylic acid moiety is neutralised, leading in a weakening of the N-C(=O) link in the  $\beta$ -lactam ring, resulting in self-activation. Construction of the ideal mousetrap for trapping selected animals is analogous to certain structural requirements.

Penicillin kills bacteria by preventing the formation of peptidoglycans, the structural component of the bacterial cell wall, from being completed. It inhibits the activity of enzymes that are required for the final step in cell wall formation, the cross-linking of peptidoglycans. It accomplishes this by using the  $\beta$ -lactam ring, a structure found in penicillin molecules, to bind to penicillin binding proteins. As a result of fewer cross-links, the

cell wall weakens, allowing water to flow freely into the cell as it struggles to maintain the proper osmotic gradient. This causes cell death and lysis. Even though the enzymes that hydrolyze peptidoglycan cross-links continue to function, those that generate them do not. The bacterium's cell wall is weakened as a result, and osmotic pressure becomes progressively unbalanced, finally leading to cell death (cytolysis). Furthermore, the accumulation of peptidoglycan precursors activates bacterial cell wall hydrolases and autolysins, which further breakdown the peptidoglycans in the cell wall. Penicillins' tiny size boosts their efficacy by allowing them to permeate all the way through the cell wall. The glycopeptide antibiotics vancomycin and teicoplanin, on the other hand, are both substantially larger than penicillins.

When Gram-positive bacteria lose their cell walls, they are referred to as protoplasts. After treatment with penicillin, Gram-negative bacteria retain their cell walls and are referred to as spheroplasts. Penicillin and aminoglycosides work together because peptidoglycan production suppression allows aminoglycosides to enter the bacterial cell wall more easily, disrupting bacterial protein synthesis within the cell. As a result, the MBC of vulnerable organisms is reduced. Some bacteria create enzymes called  $\beta$ -lactamases that break down the  $\beta$ -lactam ring, making them resistant to penicillin. For usage against antibiotic-resistant bacteria or in immunocompromised people, several penicillins are changed or administered in combination with other medications. When  $\beta$ -lactamase inhibitors like clavulanic acid or tazobactam are combined with penicillin, it gives penicillin activity against  $\beta$ -lactamase-producing bacteria.  $\beta$ -lactamase inhibitors bind to  $\beta$ -lactamase in an irreversible manner, preventing it from breaking down the antibiotic's  $\beta$ -lactam rings. Flucloxacillin, on the other hand, is modified penicillin with anti  $\beta$ -lactamase activity due to an acyl side chain that protects the  $\beta$ -lactam ring from  $\beta$ -lactamase.

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