

Penicillins

Beta-lactam antibiotics that inhibit bacterial cell-wall synthesis by binding penicillin-binding proteins.

Online guide: <https://www.medguideglobal.com/pharmacology/antibiotics/penicillins>

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Quick revision

One Liner: Penicillins are beta-lactams that block the final cross-linking step of peptidoglycan synthesis by binding penicillin-binding proteins; spectrum differs sharply between subclasses.

1. Four core subclasses: natural penicillins, aminopenicillins, antistaphylococcal (penicillinase-resistant) penicillins, and ureidopenicillins. [1]
2. Mechanism: acylation of transpeptidases (penicillin-binding proteins) prevents peptidoglycan cross-linking, causing loss of viability and autolysis. [1]
3. Selective toxicity comes from the fact that human cells have no peptidoglycan cell wall. [2]
4. Piperacillin is the subclass member with activity against *Pseudomonas aeruginosa*. [1]
5. Penicillin G given parenterally is the preferred drug for treating patients in all stages of syphilis. [4]
6. Four resistance routes: beta-lactamase production, reduced penetration, altered penicillin-binding proteins, and efflux pumps. [1]
7. Around 10% of US patients report a penicillin allergy, but fewer than 1% are truly allergic. [5]

Overview

1. Penicillins are beta-lactam antibacterial drugs that inhibit bacterial cell-wall synthesis. They act by binding bacterial penicillin-binding proteins, the transpeptidase enzymes responsible for cross-linking peptidoglycan, which leaves the cell wall unable to withstand normal osmotic stress. [1][2]
2. Although every member of the class shares the beta-lactam ring and the same molecular target, the individual drugs differ substantially in antibacterial spectrum. Those differences come from side-chain chemistry and from how vulnerable each drug is to bacterial beta-lactamase enzymes, which is why the class is always studied subclass by subclass rather than as a single agent. [1][3]
3. Penicillins remain first-choice therapy in a number of common and serious infections, and they are prescribed across primary care, hospital medicine and dentistry. They are also the class most often blocked by an inaccurate allergy label, which makes understanding both their spectrum and their true allergy risk clinically important. [6][5]

Classification and drug examples

Intro: Penicillins are conventionally divided by spectrum and by resistance to staphylococcal penicillinase.

Beta-lactamase inhibitor combinations are listed separately because the inhibitor is a partner drug, not a chemically distinct penicillin.

1. Name: Natural penicillins

1. Note: Used for susceptible Gram-positive organisms and selected Gram-negative infections.

1. Name: Benzylpenicillin

Penicillin G

1. Route: IV/IM

1. Note: Parenteral natural penicillin; the preferred agent for syphilis at all stages. [4][1]

2. Name: Phenoxymethylpenicillin

Penicillin V

2. Route: Oral

2. Note: Oral natural penicillin used for a range of susceptible infections. [9] [1]

2. Name: Aminopenicillins

2. Note: Broader coverage than the natural penicillins, extending to additional Gram-negative organisms and anaerobes.

1. Name: Amoxicillin

1. Route: Oral

1. Note: Widely used for bacterial infections including chest infections and dental abscesses. [6][10]

2. Name: Ampicillin

2. Route: IV/Oral

2. Note: Parenteral aminopenicillin with a comparable spectrum to amoxicillin. [1] [1]

3. Name: Antistaphylococcal (penicillinase-resistant) penicillins

3. Note: Structurally protected against staphylococcal penicillinase, so they retain activity against methicillin-susceptible staphylococci.

1. Name: Flucloxacillin

1. Route: Oral/IV

1. Note: Used for infections including chest infection, ear infection, osteomyelitis and pneumonia. [7]

2. Name: Oxacillin

2. Route: IV

2. Note: Parenteral antistaphylococcal penicillin. [1]

3. Name: Nafcillin

3. Route: IV

3. Note: Parenteral antistaphylococcal penicillin. [1]

4. Name: Dicloxacillin

4. Route: Oral

4. Note: Oral antistaphylococcal penicillin. [1] [1]

4. Name: Ureidopenicillins (antipseudomonal penicillins)

4. Note: Active against Gram-negative bacilli that resist aminopenicillins, including *Pseudomonas aeruginosa*.

Name: Piperacillin

Route: IV

Note: Commonly given combined with a beta-lactamase inhibitor. [1][3] [1]

5. Name: Beta-lactamase inhibitor combinations

5. Note: Not a chemical penicillin subclass. A susceptible beta-lactam is paired with an inhibitor of serine beta-lactamases so the partner antibiotic survives enzymatic attack.

1. Name: Amoxicillin + clavulanic acid

Co-amoxiclav

1. Route: Oral/IV

1. Note: Clavulanic acid is a first-generation serine beta-lactamase inhibitor. [3]

2. Name: Ampicillin + sulbactam

2. Route: IV

2. Note: Sulbactam is a first-generation serine beta-lactamase inhibitor. [3]

3. Name: Piperacillin + tazobactam

3. Route: IV

3. Note: Used for indications including community-acquired pneumonia, intra-abdominal infection, skin and skin-structure infection and female pelvic infection. [3] [3]

Mechanism of action

Summary: Penicillins acylate the bacterial transpeptidases known as penicillin-binding proteins, blocking the final

cross-linking step of peptidoglycan synthesis.

Target: Penicillin-binding proteins (bacterial transpeptidases)

Cidal Static: bactericidal

Kill Kinetics: time-dependent

1. Step: 1

1. Title: The drug reaches its target

1. Detail: The penicillin crosses the outer layers of a susceptible organism and reaches the penicillin-binding proteins situated on the cytoplasmic membrane. [1]

2. Step: 2

2. Title: It binds penicillin-binding proteins

2. Detail: The beta-lactam ring acylates the transpeptidase active site, forming a stable complex that inactivates the enzyme. [1]

3. Step: 3

3. Title: Peptidoglycan cross-linking stops

3. Detail: Transpeptidases normally cross-link peptide chains to complete the peptidoglycan mesh. With the enzyme inactivated, this last step of cell-wall synthesis cannot proceed. [1]

4. Step: 4

4. Title: The cell wall fails and the organism lyses

4. Detail: The incompletely cross-linked wall cannot resist normal internal osmotic pressure, and loss of viability and lysis follow, aided by the organism's own autolytic processes. [1]

5. Step: 5

5. Title: Human cells are spared

5. Detail: Selective toxicity arises because human cells have no peptidoglycan cell wall and therefore no equivalent target for the drug to attack. [2]

Spectrum of activity

Intro: There is no single penicillin spectrum. Coverage must be learned subclass by subclass, and local susceptibility data always governs actual therapy.

1. Subclass Id: natural

Susceptible Gram-positive organisms

Selected Gram-negative organisms

Penicillinase-producing staphylococci [1]

2. Subclass Id: aminopenicillins

Gram-positive organisms

Gram-negative cover broader than the natural penicillins

Anaerobic organisms

Pseudomonas aeruginosa [1]

3. Subclass Id: antistaphylococcal

Methicillin-susceptible staphylococci

MRSA, which carries an altered penicillin-binding protein [1][3]

4. Subclass Id: ureidopenicillins

1. Aminopenicillin-resistant Gram-negative bacilli

2. *Pseudomonas aeruginosa* [1]

Major clinical uses

1. Drug: Benzylpenicillin (penicillin G)

1. Organism: *Treponema pallidum*

1. Indication: Syphilis, all stages
1. Line: preferred
1. Note: Administered parenterally; the specific preparation, dose and duration vary by stage and clinical presentation. [4]
2. Drug: Phenoxymethylpenicillin (penicillin V)
2. Organism: Susceptible Gram-positive organisms
2. Indication: Selected infections treatable with an oral natural penicillin
2. Line: first-line
2. Note: Oral option where a natural penicillin is appropriate. [9]
3. Drug: Amoxicillin
3. Organism: Susceptible bacteria
3. Indication: Bacterial infections including chest infections and dental abscesses
3. Line: first-line
3. Note: Also used in regimens for conditions including ear infection and stomach ulcers. [6]
4. Drug: Flucloxacillin
4. Organism: Methicillin-susceptible staphylococci
4. Indication: Infections including chest infection, ear infection, osteomyelitis and pneumonia
4. Line: first-line
4. Note: Chosen when a penicillinase-producing staphylococcus is the likely pathogen. [7][1]
5. Drug: Piperacillin with tazobactam
5. Organism: Mixed flora including *Pseudomonas aeruginosa* and beta-lactamase producers
5. Indication: Community-acquired pneumonia, intra-abdominal infection, skin and skin-structure infection, female pelvic infection
5. Line: broad empirical
5. Note: The tazobactam component protects piperacillin from susceptible serine beta-lactamases. [3][1]

Pharmacokinetics

1. Drug: Benzylpenicillin (penicillin G)
1. Route: IV/IM
1. Absorption: Not suitable for oral use
1. Csf Penetration: Improved when meninges are inflamed
1. Metabolism: Limited
1. Elimination: Predominantly renal
1. Half Life: Short
1. Dose Adjust: Renal impairment [2]
2. Drug: Phenoxymethylpenicillin (penicillin V)
2. Route: Oral
2. Absorption: Acid-stable enough for oral administration
2. Csf Penetration: Poor
2. Metabolism: Limited
2. Elimination: Predominantly renal
2. Half Life: Short
2. Dose Adjust: Renal impairment [9]
3. Drug: Amoxicillin
3. Route: Oral
3. Absorption: Well absorbed orally
3. Csf Penetration: Poor unless meninges inflamed

- 3. Metabolism: Limited
- 3. Elimination: Predominantly renal
- 3. Half Life: Short
- 3. Dose Adjust: Renal impairment [10]
- 4. Drug: Piperacillin
- 4. Route: IV
- 4. Absorption: Not orally bioavailable
- 4. Csf Penetration: Limited
- 4. Metabolism: Limited
- 4. Elimination: Predominantly renal
- 4. Half Life: Short
- 4. Dose Adjust: Renal impairment [1]
- 1. Penicillins are, as a class, short-half-life drugs cleared largely by the kidney, which is why dosing intervals are frequent and why renal function drives dose adjustment. [2]
- 2. This page deliberately gives no dose regimens. Doses depend on the indication, the organism, renal function, age and weight, and belong in a prescribing reference used by the treating clinician.

Adverse effects

- 1. Effect: Gastrointestinal upset
- 1. Detail: Nausea, vomiting and diarrhoea are commonly reported with oral penicillins. [6][10]
- 2. Effect: Rash
- 2. Detail: Skin rash is among the most frequently reported reactions and is not always allergic in nature. [6][5]
- 1. Effect: Jarisch-Herxheimer reaction
- 1. Detail: Fever, headache and myalgia within the first 24 hours of treating syphilis. It follows rapid lysis of spirochaetes and release of endotoxin, and is a reaction to the treatment rather than an allergic reaction to penicillin - a distinction that matters, because mislabelling it as allergy can cost a patient the only agent that works.
- 1. Action: It is self-limiting and is not a reason to withhold or delay indicated therapy. [4][2]
- 2. Effect: Haematological and neurological effects at high parenteral exposure
- 2. Detail: Coombs-positive haemolytic anaemia and neutropenia are described with very high daily intravenous exposure and resolve once treatment stops. Neurological features including hyperreflexia, myoclonus, seizures and coma follow high intravenous exposure and are more likely where renal function is impaired.
- 2. Action: Blood counts and neurological state are monitored on prolonged high-exposure intravenous therapy, and exposure is reviewed against renal function. [2]
- 3. Effect: Serious allergic reaction (anaphylaxis)
- 3. Detail: Sudden swelling of the lips, mouth, throat or tongue, wheezing or difficulty breathing, throat tightness, skin turning blue, grey or pale, sudden confusion, drowsiness or dizziness, or loss of consciousness.
- 3. Action: Treat as a medical emergency requiring immediate hospital treatment. [8]
- 4. Effect: Liver injury
- 4. Detail: Pale stools with dark urine and yellowing of the whites of the eyes or the skin can indicate liver problems.
- 4. Action: The drug is stopped and urgent clinical assessment arranged when these features appear. [8]
- Drug: Flucloxacillin
- Effect: Hepatic reactions presenting as jaundice with pale stools and dark urine are specifically flagged in its safety information. [8]

Contraindications, precautions and interactions

- A previous serious, immediate hypersensitivity reaction to a penicillin. [5]
- 1. Renal impairment, where clearance is reduced and dose adjustment is required. [2]

2. An unverified penicillin allergy label. Because fewer than 1% of the roughly 10% of US patients reporting penicillin allergy are truly allergic, an unexamined label may push a patient onto broader second-line therapy without benefit. Evaluation uses allergy history, validated risk assessment, skin testing and, where appropriate, a supervised direct oral challenge. [5]

Resistance mechanisms

1. Mechanism: Beta-lactamase production

1. Detail: Bacterial enzymes hydrolyse the beta-lactam ring and inactivate susceptible drugs before they can reach their target.

1. Staphylococcal penicillinase

2. Extended-spectrum beta-lactamases

1. Countermeasure: Use a penicillinase-resistant agent, or pair a susceptible beta-lactam with a serine beta-lactamase inhibitor. [1][3]

2. Mechanism: Altered penicillin-binding proteins

2. Detail: The target enzyme itself changes so the drug can no longer bind it effectively.

Methicillin-resistant *Staphylococcus aureus*

2. Countermeasure: No beta-lactamase inhibitor reverses this; a different drug class is required. [1][3]

3. Mechanism: Decreased penetration

3. Detail: Reduced permeability limits how much drug reaches penicillin-binding proteins on the cytoplasmic membrane.

3. Countermeasure: Agents that penetrate better, guided by susceptibility testing. [1]

4. Mechanism: Efflux

4. Detail: Dedicated pump systems export the antibiotic out of the bacterial cell before it can act.

4. Countermeasure: Selection guided by susceptibility testing. [1]

Stewardship Note: Because different mechanisms defeat different beta-lactams, resistance cannot be summarised as "beta-lactamase equals resistance to all penicillins". Therapy is directed by local susceptibility data.

Comparison tables

Title: Penicillin subclasses at a glance

Caption: Distinguishing feature and representative use for each subclass. Spectrum statements are deliberately general; local susceptibility data governs actual therapy.

1. Key: group

1. Label: Group

1. Is Row Header: true

2. Key: examples

2. Label: Examples

3. Key: feature

3. Label: Main distinguishing feature

4. Key: uses

4. Label: Representative uses

1. Group: Natural penicillins

1. Examples: Penicillin G, penicillin V

1. Feature: Original narrow-spectrum agents; vulnerable to penicillinase

1. Uses: Susceptible Gram-positive infections; syphilis at all stages [1][4]

2. Group: Aminopenicillins

2. Examples: Amoxicillin, ampicillin

2. Feature: Extended Gram-negative reach compared with natural penicillins

2. Uses: Chest infections, dental abscess, ear infection [1][6]
3. Group: Antistaphylococcal penicillins
3. Examples: Flucloxacillin, oxacillin, nafcillin, dicloxacillin
3. Feature: Resist staphylococcal penicillinase
3. Uses: Methicillin-susceptible staphylococcal infection, including bone and chest infection [1][7]
4. Group: Ureidopenicillins
4. Examples: Piperacillin
4. Feature: Activity against aminopenicillin-resistant Gram-negative bacilli
4. Uses: Serious infection where *Pseudomonas aeruginosa* is a concern [1]
5. Group: Beta-lactam + inhibitor
5. Examples: Amoxicillin-clavulanate, ampicillin-sulbactam, piperacillin-tazobactam
5. Feature: Partner inhibitor protects the beta-lactam from serine beta-lactamases
5. Uses: Mixed or beta-lactamase-producing infections [3]

High-yield exam pearls

1. Pearl: The molecular target of every penicillin is the penicillin-binding protein (a transpeptidase), not the ribosome or DNA.
1. Why: Beta-lactams acylate the transpeptidase that cross-links peptidoglycan, which is the last step of cell-wall assembly. [1]
2. Pearl: Antistaphylococcal penicillins such as flucloxacillin, oxacillin, nafcillin and dicloxacillin exist specifically to resist staphylococcal penicillinase.
2. Why: They cover methicillin-susceptible staphylococci, which plain penicillin G no longer reliably does. [1]
3. Pearl: Piperacillin is the antipseudomonal penicillin.
3. Why: Ureidopenicillins retain activity against aminopenicillin-resistant Gram-negative bacilli including *Pseudomonas aeruginosa*. [1]
4. Pearl: Penicillin remains the preferred therapy for syphilis at every stage.
4. Why: CDC STI treatment guidance names parenteral penicillin G as the preferred drug across all stages of the disease. [4]
5. Pearl: A beta-lactamase inhibitor is not itself a penicillin subclass - it is a partner drug that protects a susceptible beta-lactam.
5. Why: Clavulanic acid, sulbactam and tazobactam inhibit serine beta-lactamases; they are co-administered, not chemically reclassified. [3]
6. Pearl: MRSA is not solved by adding a beta-lactamase inhibitor.
6. Why: Staphylococcal methicillin resistance works through an altered penicillin-binding protein, a target change no beta-lactamase inhibitor can reverse. [3]

Common exam traps

1. Trap: "All penicillins have the same spectrum."
1. Correction: Spectrum varies substantially by subclass: natural penicillins, aminopenicillins, antistaphylococcal penicillins and ureidopenicillins each cover a different range of organisms. [1]
2. Trap: "A beta-lactamase inhibitor makes a beta-lactam work against any resistant organism."
2. Correction: These inhibitors act on susceptible serine beta-lactamases only. They do not reverse altered penicillin-binding proteins, reduced permeability, or efflux. [3][1]
3. Trap: "A recorded penicillin allergy always rules out every beta-lactam for life."
3. Correction: Roughly 10% of US patients carry a penicillin allergy label but under 1% are truly allergic; validated risk assessment, skin testing and supervised direct oral challenge can correct an inaccurate label. [5]
4. Trap: "Penicillins are bacteriostatic because they only stop wall building."

4. Correction: Blocking cross-linking leads to loss of viability and lysis through the organism's own autolytic processes - the effect is bactericidal, not merely growth-arresting. [1]

Self-test questions

1. Question: Which bacterial structure does a penicillin bind in order to exert its antibacterial effect?

1. The 30S ribosomal subunit
2. DNA gyrase within the bacterial nucleoid
3. Penicillin-binding proteins, the transpeptidase enzymes of the cell wall
4. The bacterial outer-membrane porin channel

1. Explanation: Every drug in this class acts on the same molecular target: the transpeptidase enzymes called penicillin-binding proteins. The beta-lactam ring acylates the enzyme's active site, and the resulting stable complex leaves the enzyme unable to finish cross-linking peptidoglycan. Neither the ribosome nor bacterial DNA is involved. [1]

2. Question: What explains the selective toxicity of penicillins for bacteria over human cells?

1. Human transpeptidase enzymes bind the drug with much lower affinity
2. Penicillins cannot cross the human plasma membrane
3. Hepatic metabolism inactivates the drug before it reaches human tissue
4. Human cells have no peptidoglycan cell wall, so there is no comparable target

2. Explanation: The structure these drugs attack simply does not exist in us. Because our cells build no peptidoglycan wall, there is no equivalent enzyme for the beta-lactam to inactivate, and that structural absence - rather than any difference in absorption or metabolism - is the basis of the class's safety margin. [2]

3. Question: A patient needs a penicillin-class agent with activity against *Pseudomonas aeruginosa*. Which subclass and representative drug fit?

1. Ureidopenicillins - piperacillin
2. Aminopenicillins - amoxicillin
3. Antistaphylococcal penicillins - flucloxacillin
4. Natural penicillins - benzylpenicillin

3. Explanation: Antipseudomonal reach within this class belongs to the ureidopenicillin group, and piperacillin is its representative member. That group holds activity against Gram-negative bacilli which the aminopenicillins cannot cover, *Pseudomonas aeruginosa* included; the aminopenicillins list that organism as a recognised gap. [1]

4. Question: Adding clavulanic acid, sulbactam or tazobactam to a beta-lactam will NOT overcome which form of resistance?

1. Hydrolysis by staphylococcal penicillinase
2. Inactivation by susceptible serine beta-lactamases
3. Methicillin resistance in staphylococci
4. Enzymatic destruction of the beta-lactam ring before it reaches its target

4. Explanation: These partner agents disable serine beta-lactamase enzymes, so they rescue a beta-lactam that would otherwise be hydrolysed. Staphylococcal methicillin resistance is a different problem entirely - the target enzyme itself has changed shape - and no amount of enzyme inhibition restores binding to an altered penicillin-binding protein. [3]

5. Question: Approximately what proportion of US patients who report a penicillin allergy are found to be genuinely allergic on evaluation?

1. About half of those who report it
2. Fewer than 1%, although roughly 10% carry the label
3. Roughly 10%, matching the proportion who report it
4. Around 25% of those who report it

5. Explanation: Reported and confirmed allergy diverge sharply. About one in ten US patients carries the label, yet under one percent prove truly allergic when assessed. This is why an unexamined label is treated as a caution rather than a settled fact - allergy history, validated risk assessment, skin testing and a supervised direct oral challenge can

correct it. [5]

6. Question: Which statement about penicillin therapy in syphilis is correct?

1. Oral phenoxymethylpenicillin is preferred for early-stage disease only
2. Penicillin is preferred only in late-stage disease
3. Amoxicillin combined with clavulanic acid is the agent of choice
4. Parenteral benzylpenicillin is the preferred agent across every stage

6. Explanation: Benzylpenicillin given by injection holds preferred status for this infection at all stages of disease, not merely early or late presentations. The particular preparation and duration differ with the stage and clinical picture, but the choice of drug itself does not change. [4]

7. Question: A student argues that penicillins must be bacteriostatic because they only prevent new wall material being assembled. Why is this reasoning wrong?

1. Penicillins additionally inhibit bacterial protein synthesis, which kills the organism
2. Interrupted cross-linking leaves a wall that cannot resist osmotic pressure, and the organism's own autolytic processes complete the killing
3. The drugs dissolve the existing peptidoglycan mesh directly
4. Growth arrest and killing mean the same thing for organisms with a cell wall

7. Explanation: Halting the final cross-linking step does more than pause growth. The partly assembled wall can no longer contain normal internal osmotic pressure, and the bacterium's own autolytic machinery finishes the job, so viability is lost and the cell lyses. The class is therefore classified as bactericidal. [1]

8. Question: Which adverse effect is specifically flagged in the safety information for flucloxacillin?

1. Irreversible cochlear hearing loss
2. Peripheral neuropathy with prolonged use
3. Hepatic reactions presenting as jaundice with pale stools and dark urine
4. Photosensitivity rash on sun-exposed skin

8. Explanation: Liver injury is the drug-specific warning attached to this antistaphylococcal penicillin. The presentation to recognise combines yellowing of the skin or the whites of the eyes with pale stools and darkened urine, and it is treated as a reason to stop the medicine and seek urgent medical review. [8]

Frequently asked questions

1. Question: Why are there so many different penicillins?

1. Answer: Because a single beta-lactam cannot cover every organism. Side-chain chemistry changes which bacteria a penicillin reaches and how well it survives bacterial beta-lactamase enzymes, so each subclass was developed to fill a specific gap left by the ones before it. [1][3]

2. Question: Are penicillins bactericidal or bacteriostatic?

2. Answer: Bactericidal. Blocking peptidoglycan cross-linking leads to loss of viability and lysis, aided by the bacterium's own autolytic processes. [1]

3. Question: Why do penicillins not damage human cells?

3. Answer: Human cells have no peptidoglycan cell wall, so there is no equivalent structure for the drug to disrupt. That is the basis of the class's selective toxicity. [2]

4. Question: Does a beta-lactamase inhibitor make a penicillin work against MRSA?

4. Answer: No. Methicillin resistance in staphylococci works through an altered penicillin-binding protein rather than through beta-lactamase, and inhibiting the enzyme does nothing about a changed target. [3]

5. Question: How common is genuine penicillin allergy?

5. Answer: Approximately 10% of US patients report a past allergic reaction to a penicillin-class antibiotic, but fewer than 1% turn out to be truly allergic on evaluation. [5]

References

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