

Carbapenems

Parenteral beta-lactams with a fused ring that resists most beta-lactamases, giving the broadest spectrum of the beta-lactam family.

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

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Quick revision

One Liner: Carbapenems are parenteral beta-lactams whose fused ring resists most beta-lactamases, covering gram-positives, gram-negatives and anaerobes - with antipseudomonal reach in every member except ertapenem, and no useful activity against MRSA.

1. Four agents in US practice: imipenem (1985), meropenem (1996), ertapenem (2001) and doripenem (2008). [1]
2. All four are poorly absorbed orally and must be given parenterally. [1]
3. The fused beta-lactam ring is resistant to most beta-lactamases - this is the structural reason for the class's breadth. [1]
4. Imipenem must be given with cilastatin, which inhibits its renal metabolism and prolongs its half-life. [1]
5. Ertapenem is the once-daily agent: half-life about 4 hours versus roughly 1 hour for the others. [1]
6. Most methicillin-resistant staphylococci remain resistant - carbapenems are not an MRSA answer. [1]
7. Class B metallo-beta-lactamases hydrolyse all beta-lactams except aztreonam. [2]

Overview

1. Carbapenems are beta-lactam antibiotics with a broad spectrum of activity against many gram-positive and gram-negative, aerobic and anaerobic organisms. Like other beta-lactams they bind critical penicillin-binding proteins, disrupting the growth and structural integrity of the bacterial cell wall. [1]
2. What sets the class apart structurally is a fused beta-lactam ring that is resistant to most beta-lactamases. That stability is why carbapenems retain activity where penicillins and many cephalosporins have already been defeated, and why they are generally held back for serious or resistant infection rather than used first. [1]
3. The clinical significance of carbapenem resistance follows directly from that role: once an organism produces a carbapenemase, one of the last broad beta-lactam options is gone. Carbapenem-resistant Enterobacterales are therefore a major stewardship concern. [2]

Classification and drug examples

Intro: A small class. Rather than generations, the useful distinctions are dehydropeptidase-1 stability (which decides whether cilastatin is needed) and half-life (which decides dosing frequency).

1. Name: Requires a dehydropeptidase-1 inhibitor

1. Note: Imipenem is co-administered with cilastatin, which inhibits its renal metabolism and prolongs its half-life.

Name: Imipenem with cilastatin

Primaxin

Route: IV

Note: Introduced 1985. Used in nosocomial infections including pneumonia, intra-abdominal and urinary tract infection.

[1] [1]

2. Name: Dehydropeptidase-1 stable, multiple daily doses

2. Note: Given without a dehydropeptidase-1 inhibitor; half-life of roughly 1 hour means dosing several times a day.

1. Name: Meropenem

Merrem

1. Route: IV

1. Note: Introduced 1996. Used for nosocomial infections, meningitis especially. [1]

2. Name: Doripenem

Doribax

2. Route: IV

2. Note: Introduced 2008. [1] [1]

3. Name: Dehydropeptidase-1 stable, once daily

3. Note: A roughly 4-hour half-life makes once-daily administration possible.

Name: Ertapenem

Invanz

Route: IV/IM

Note: Introduced 2001. Used for community-acquired as well as nosocomial infections. [1] [1]

Mechanism of action

Summary: Carbapenems bind critical penicillin-binding proteins, disrupting the growth and structural integrity of the bacterial cell wall. Their fused beta-lactam ring resists most beta-lactamases.

Target: Penicillin-binding proteins (bacterial transpeptidases)

Cidal Static: bactericidal

Kill Kinetics: time-dependent

1. Step: 1

1. Title: The drug survives the enzymatic defences

1. Detail: The fused beta-lactam ring is resistant to most beta-lactamases, so the drug reaches its target in organisms that would inactivate a penicillin or cephalosporin. [1]

2. Step: 2

2. Title: It binds penicillin-binding proteins

2. Detail: Carbapenems bind the critical penicillin-binding proteins responsible for assembling the cell wall - the same transpeptidases every beta-lactam targets. [1][3]

3. Step: 3

3. Title: Cell-wall integrity fails

3. Detail: Growth and structural integrity of the bacterial cell wall are disrupted, and the organism cannot maintain a competent wall. [1]

Spectrum of activity

Intro: A broad spectrum shared by most of the class, with two exceptions that matter: what no carbapenem covers, and what ertapenem alone does not.

Subclass Id: class-wide

1. Streptococci

2. Methicillin-susceptible staphylococci

3. Listeria

4. Enterococcus faecalis (imipenem; ertapenem is not active, and Enterococcus faecium is resistant across the class)

1. Enterobacterales

2. Pseudomonas aeruginosa (imipenem, meropenem and doripenem - not ertapenem)

3. Acinetobacter species (imipenem, meropenem and doripenem - not ertapenem)

Many Bacteroides species

1. Most methicillin-resistant staphylococci

2. Carbapenemase-producing organisms

3. Ertapenem only: Pseudomonas aeruginosa, Acinetobacter species and enterococci are inherently resistant

[1][2][4][5]

Major clinical uses

1. Drug: Imipenem with cilastatin
1. Organism: Susceptible gram-negative and gram-positive organisms
1. Indication: Nosocomial pneumonia, intra-abdominal infection, urinary tract infection
1. Line: reserve
1. Note: Held back for serious or resistant infection rather than used first. [1]
2. Drug: Meropenem
2. Organism: Susceptible meningeal and nosocomial pathogens
2. Indication: Nosocomial infection, and meningitis especially
2. Line: reserve
2. Note: The carbapenem most associated with meningitis treatment. [1]
3. Drug: Ertapenem
3. Organism: Susceptible organisms other than *Pseudomonas aeruginosa*, *Acinetobacter* species and enterococci
3. Indication: Community-acquired infection as well as nosocomial infection
3. Line: reserve
3. Note: Once-daily administration. Its spectrum gap is not a matter of preference: these organisms are listed as inherently resistant, so ertapenem is unsuitable wherever they are suspected. [1][4]
4. Drug: Doripenem
4. Organism: Susceptible gram-negative and gram-positive organisms
4. Indication: Nosocomial infection
4. Line: reserve
4. Note: Used similarly to imipenem and meropenem. [1]

Pharmacokinetics

1. Drug: Imipenem with cilastatin
1. Route: IV
1. Absorption: Poorly absorbed orally
1. Csf Penetration: See prescribing reference
1. Metabolism: Renal metabolism inhibited by cilastatin
1. Elimination: Renal
1. Half Life: About 1 hour
1. Dose Adjust: Renal impairment [1]
2. Drug: Meropenem
2. Route: IV
2. Absorption: Poorly absorbed orally
2. Csf Penetration: Adequate for meningitis use
2. Metabolism: Stable to dehydropeptidase-1
2. Elimination: Renal
2. Half Life: About 1 hour
2. Dose Adjust: Renal impairment [1]
3. Drug: Ertapenem
3. Route: IV/IM
3. Absorption: Poorly absorbed orally
3. Csf Penetration: See prescribing reference
3. Metabolism: Stable to dehydropeptidase-1

- 3. Elimination: Renal
- 3. Half Life: About 4 hours
- 3. Dose Adjust: Renal impairment [1]
- 4. Drug: Doripenem
- 4. Route: IV
- 4. Absorption: Poorly absorbed orally
- 4. Csf Penetration: See prescribing reference
- 4. Metabolism: Stable to dehydropeptidase-1
- 4. Elimination: Renal
- 4. Half Life: About 1 hour
- 4. Dose Adjust: Renal impairment [1]
- 1. Because all four are parenteral only, there is no oral step-down within the class - a practical constraint that shapes how carbapenem courses are planned. [1]
- 2. This page gives no dose regimens by design. Doses depend on indication, organism, renal function, age and weight, and belong in a prescribing reference used by the treating clinician.

Adverse effects

- 1. Effect: Injection site reactions
 - 1. Detail: A common consequence of parenteral-only administration. [1]
- 2. Effect: Gastrointestinal symptoms
 - 2. Detail: Diarrhoea, nausea and vomiting. [1]
- 3. Effect: Skin rash and pruritus
 - 3. Detail: Reported across the class. [1]
- 1. Effect: Seizures and other central nervous system effects
 - 1. Detail: The characteristic serious toxicity of the class. Labels for imipenem, meropenem and ertapenem all report seizures, confusional states and myoclonus, and all three name the same risk factors: pre-existing CNS disorders such as brain lesions or a history of seizures, compromised renal function, and dosing above the recommended range.
 - 1. Action: Dose is adjusted to renal function, and new focal tremor, myoclonus or seizure prompts neurological assessment and reconsideration of the drug. [5][6][4]
- 2. Effect: Clinically significant hepatotoxicity with jaundice
 - 2. Detail: Rare with the carbapenems, though transient liver enzyme elevations occur in roughly 1 to 12% of patients across studies.
 - 2. Action: Assess urgently if jaundice develops. [1]
- 1. Drug: Imipenem with cilastatin
 - 1. Effect: Its label states the drug is not recommended in paediatric patients with CNS infections because of the seizure risk, and is not indicated in meningitis. Meropenem, by contrast, is the carbapenem carrying a meningitis indication. [5][6]
- 2. Drug: Ertapenem
 - 2. Effect: Patients with renal impairment are described as being at higher risk of ertapenem-induced encephalopathy, with resolution potentially prolonged. [4]

Contraindications, precautions and interactions

- 1. Renal impairment, where these renally eliminated agents require dose adjustment. [1]
- 2. Known beta-lactam hypersensitivity - carbapenems are beta-lactams and allergy history must be assessed before use. [1]

Resistance mechanisms

1. Mechanism: Class A carbapenemases (serine)

1. Detail: Serine-based enzymes that hydrolyse penicillins, cephalosporins, carbapenems and aztreonam.

KPC - most prevalent in the United States and globally

1. Countermeasure: Newer beta-lactamase inhibitor combinations, guided by susceptibility testing. [2]

2. Mechanism: Class B metallo-beta-lactamases

2. Detail: Zinc-dependent enzymes that hydrolyse all beta-lactams except aztreonam.

1. NDM - prevalent in South Asia

2. IMP and VIM - Asia-Pacific

2. Countermeasure: Aztreonam remains a therapeutic option against these enzymes precisely because they cannot hydrolyse it. [2]

3. Mechanism: Class D OXA enzymes

3. Detail: Decreased hydrolysing activity against carbapenems and penicillins, and resistant to older inhibitors, but they respond to avibactam.

OXA-48 - common in the Middle East and Europe

3. Countermeasure: Avibactam-containing combinations. [2]

4. Mechanism: Porin loss and efflux

4. Detail: Loss of porin channels decreases the drug's ability to reach its site of action, and efflux pumps remove antibiotic from the cell. These usually need to combine with a carbapenemase to produce high-level resistance.

4. Countermeasure: Susceptibility-directed therapy. [2]

Stewardship Note: Carbapenem resistance removes one of the last broad beta-lactam options, which is the entire argument for reserving this class rather than using it empirically.

Comparison tables

Title: The four carbapenems compared

Caption: Introduction year, dehydropeptidase-1 handling and half-life. Local susceptibility data governs actual therapy.

1. Key: drug

1. Label: Drug

1. Is Row Header: true

2. Key: year

2. Label: Introduced

3. Key: dhp1

3. Label: DHP-1 inhibitor needed

4. Key: half_life

4. Label: Half-life

5. Key: note

5. Label: Typical setting

1. Drug: Imipenem (Primaxin)

1. Year: 1985

1. Dhp1: Yes - cilastatin

1. Half Life: About 1 hour

1. Note: Nosocomial infection [1]

2. Drug: Meropenem (Merrem)

2. Year: 1996

2. Dhp1: No

2. Half Life: About 1 hour

2. Note: Nosocomial infection, meningitis especially [1]
3. Drug: Ertapenem (Invanz)
3. Year: 2001
3. Dhp1: No
3. Half Life: About 4 hours
3. Note: Community-acquired and nosocomial; once daily [1]
4. Drug: Doripenem (Doribax)
4. Year: 2008
4. Dhp1: No
4. Half Life: About 1 hour
4. Note: Nosocomial infection [1]

High-yield exam pearls

1. Pearl: Imipenem is the one that needs a partner: cilastatin.
 1. Why: Cilastatin inhibits renal metabolism of imipenem and prolongs its half-life. Meropenem, ertapenem and doripenem have greater stability to dehydropeptidase-1 and are given without it. [1]
2. Pearl: Ertapenem is the once-daily carbapenem.
 2. Why: Its half-life is roughly 4 hours against about 1 hour for imipenem, meropenem and doripenem. [1]
3. Pearl: Carbapenems do not solve MRSA.
 3. Why: Most methicillin-resistant staphylococci are resistant, because methicillin resistance is an altered penicillin-binding protein rather than a beta-lactamase. [1]
4. Pearl: Meropenem is the carbapenem particularly associated with meningitis treatment.
 4. Why: Imipenem/cilastatin, meropenem and doripenem are used in nosocomial infections including pneumonia, intra-abdominal infection, urinary tract infection and meningitis - meningitis especially with meropenem. [1]
5. Pearl: Aztreonam survives metallo-beta-lactamases.
 5. Why: Class B carbapenemases hydrolyse all beta-lactams except aztreonam, which is why aztreonam becomes a therapeutic option against those organisms. [2]

Common exam traps

1. Trap: "Carbapenems cover everything."
 1. Correction: Their spectrum is very broad but most methicillin-resistant staphylococci remain resistant, and carbapenemase-producing organisms defeat them outright. [1][2]
2. Trap: "Cilastatin is an antibiotic that broadens imipenem's spectrum."
 2. Correction: Cilastatin has no antibacterial role. It inhibits the renal metabolism of imipenem and prolongs its half-life. [1]
3. Trap: "Carbapenem resistance always means a carbapenemase."
 3. Correction: Loss of porin channels reduces the drug's ability to reach its target and efflux pumps export it; these non-enzymatic routes contribute, though high-level resistance usually needs a carbapenemase as well. [2]
4. Trap: "There is an oral carbapenem for step-down therapy."
 4. Correction: All four agents are poorly absorbed orally and require parenteral administration. [1]

Self-test questions

1. Question: What is the role of cilastatin when it is co-administered with imipenem?
 1. It is a second antibiotic that widens imipenem's antibacterial spectrum
 2. It inhibits bacterial beta-lactamases so imipenem survives enzymatic attack
 3. It blocks renal metabolism of imipenem, lengthening the drug's half-life

4. It reduces the risk of injection site reactions

1. Explanation: The partner compound is not an antibacterial at all. Its job is pharmacokinetic: by preventing imipenem from being broken down in the kidney it extends how long the drug persists. Beta-lactamase inhibition is a different concept entirely, and the other three carbapenems need no such partner because they withstand dehydropeptidase-1 on their own. [1]

2. Question: Which carbapenem can be administered once daily, and what property allows this?

1. Ertapenem, because its half-life is around 4 hours rather than the roughly 1 hour of the others

2. Meropenem, because it is stable to dehydropeptidase-1

3. Imipenem, because cilastatin prolongs its half-life

4. Doripenem, because it was the most recently introduced agent

2. Explanation: Among the four agents only one has a meaningfully longer elimination half-life - roughly four hours against about an hour for imipenem, meropenem and doripenem - and that difference is what permits a single daily administration. Dehydropeptidase-1 stability is shared by three of the four and does not by itself change dosing frequency. [1]

3. Question: Which structural feature explains why carbapenems retain activity against organisms that have already defeated penicillins and many cephalosporins?

1. A siderophore side chain that hijacks bacterial iron transport

2. A quaternary ammonium group that improves porin entry

3. The absence of a beta-lactam ring altogether

4. A fused beta-lactam ring that withstands most beta-lactamases

3. Explanation: These drugs are still beta-lactams and still act on penicillin-binding proteins, but their ring is fused in a way that most bacterial beta-lactamases cannot cleave. That enzymatic durability is the whole basis of the class's exceptional breadth, and it is also why the class is generally kept in reserve for serious or resistant infection. [1]

4. Question: A colleague suggests a carbapenem for a confirmed MRSA infection. Why is that reasoning flawed?

1. Carbapenems are bacteriostatic and cannot clear a staphylococcal infection

2. Most methicillin-resistant staphylococci resist carbapenems, because the resistance lies in an altered penicillin-binding protein rather than an enzyme

3. Carbapenems have no gram-positive activity of any kind

4. MRSA produces a metallo-beta-lactamase that destroys carbapenems

4. Explanation: Breadth against beta-lactamases does not help when the target itself has changed. Methicillin resistance in staphylococci rests on a modified penicillin-binding protein, so a drug that survives enzymatic attack still cannot bind. The class does cover streptococci, enterococci, staphylococci and Listeria, but methicillin-resistant strains sit outside that cover. [1]

5. Question: Which carbapenemase class hydrolyses every beta-lactam except aztreonam?

1. Class A serine carbapenemases such as KPC

2. Class D OXA enzymes such as OXA-48

3. Extended-spectrum beta-lactamases

4. Class B zinc-dependent metallo-beta-lactamases such as NDM

5. Explanation: The zinc-dependent enzymes destroy the whole beta-lactam family with one exception, and that exception is aztreonam - which is precisely why aztreonam stays on the table when an organism carries NDM, IMP or VIM. Class A serine enzymes, KPC among them, do hydrolyse aztreonam as well as the penicillins, cephalosporins and carbapenems. [2]

6. Question: Which pair of non-enzymatic mechanisms contributes to carbapenem resistance?

1. Loss of porin channels and active efflux of the drug

2. Ribosomal methylation and target site mutation

3. Increased peptidoglycan thickness and biofilm formation

4. Reduced folate synthesis and thymidine dependence

6. Explanation: Resistance to this class is not always a matter of an enzyme. Losing the channel proteins the drug uses for entry cuts how much reaches the target, and pump systems export what does get in. These routes typically have to act alongside a carbapenemase before high-level resistance emerges, so the presence of one does not exclude the other. [2]

7. Question: Which statement about carbapenem administration is correct?

1. Ertapenem is available as an oral formulation for outpatient step-down
2. Imipenem is absorbed orally provided cilastatin is co-administered
3. All four agents are poorly absorbed from the gut and must be given parenterally
4. Meropenem is given orally when treating meningitis

7. Explanation: None of the available agents survives the oral route well enough to be useful, so every one of them is delivered by injection or infusion. The practical consequence is that a course cannot be stepped down to tablets within this class, which shapes how such courses are planned from the outset. [1]

8. Question: Which carbapenem is particularly associated with the treatment of meningitis?

1. Ertapenem
2. Meropenem
3. Imipenem with cilastatin
4. Doripenem

8. Explanation: Several members of the class are used for hospital-acquired infections such as pneumonia, intra-abdominal infection and urinary tract infection. Meningeal infection is the setting singled out for meropenem, whose central nervous system penetration is described as adequate for that use. [1]

Frequently asked questions

1. Question: Why is imipenem always given with cilastatin?

1. Answer: Cilastatin inhibits the renal metabolism of imipenem and prolongs its half-life. It has no antibacterial activity of its own. Meropenem, ertapenem and doripenem have greater stability to dehydropeptidase-1 and are given without an inhibitor. [1]

2. Question: Do carbapenems treat MRSA?

2. Answer: No. Most methicillin-resistant staphylococci remain resistant to carbapenems, because methicillin resistance works through an altered penicillin-binding protein rather than through a beta-lactamase. [1]

3. Question: Why can aztreonam sometimes be used when carbapenems fail?

3. Answer: Class B metallo-beta-lactamases such as NDM hydrolyse all beta-lactams except aztreonam. That single structural exception is what keeps aztreonam on the table against those organisms. [2]

4. Question: Is there an oral carbapenem?

4. Answer: No. All four available agents are poorly absorbed orally and require parenteral administration, so there is no oral step-down within the class. [1]

References

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