

Oxazolidinones

Fully synthetic 50S protein synthesis inhibitors reserved for resistant gram-positive infection - MRSA and VRE - defined clinically by serotonin syndrome risk and duration-dependent mitochondrial toxicity.

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

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Quick revision

One Liner: Oxazolidinones bind 23S rRNA on the 50S subunit and stop the 70S initiation complex from ever assembling - a unique early block that keeps cross-resistance rare, buys MRSA and VRE coverage with near-complete oral bioavailability, and costs MAO inhibition plus duration-dependent mitochondrial toxicity.

1. Mechanism: bind the 23S rRNA of the bacterial 50S ribosomal subunit and prevent formation of the functional 70S initiation complex, so protein synthesis never starts. [1]
2. Generally bacteriostatic against staphylococci and enterococci; activity against streptococci is described as bactericidal. [1]
3. Spectrum is resistant gram-positive organisms: MRSA and vancomycin-resistant enterococci (VRE) are the defining targets. [1][2]
4. Linezolid is a reversible, non-selective monoamine oxidase inhibitor - combining it with SSRIs and other serotonergic drugs risks serotonin syndrome. [1]
5. Myelosuppression - especially thrombocytopenia - is duration-dependent and characteristically appears with courses longer than about two weeks. [1]
6. Prolonged therapy also risks peripheral and optic neuropathy and lactic acidosis, attributed to inhibition of mitochondrial protein synthesis. [1][2]
7. Oral bioavailability is essentially complete (~100%), so oral and intravenous routes are interchangeable - a major practical advantage over vancomycin for step-down therapy. [1]
8. Tedizolid is the second-generation agent: once-daily administration, oral or intravenous, approved in 2014 and non-inferior to linezolid in acute bacterial skin and skin-structure infection. [3]

Overview

1. Oxazolidinones are a fully synthetic antibiotic class whose clinical identity is resistant gram-positive infection. Linezolid, the first agent, is used for serious infections caused by resistant enterococci and staphylococci, including vancomycin-resistant enterococcal (VRE) infection and pneumonia caused by methicillin-resistant *Staphylococcus aureus* (MRSA). Tedizolid, approved in 2014, is the second-generation member. [2][3]
2. The mechanism is distinctive: the drug binds the 23S rRNA of the 50S ribosomal subunit and prevents formation of the functional 70S initiation complex, so bacterial protein synthesis is blocked before it begins rather than interrupted mid-stream. Because no other class acts at this step, cross-resistance with other protein synthesis inhibitors is not expected, which is precisely why the class is reserved for organisms that have defeated other options. [1]
3. What limits the class is what it does to the patient rather than what it misses microbiologically. Linezolid is a reversible non-selective monoamine oxidase inhibitor, making serotonin syndrome a real risk with serotonergic co-medication, and its effect on mitochondrial ribosomes drives the duration-dependent toxicities - myelosuppression beyond about two weeks, peripheral and optic neuropathy, and lactic acidosis on prolonged courses. The compensation is essentially complete oral bioavailability, which lets a resistant gram-positive infection be treated by mouth. [1][2]

Classification and drug examples

Intro: A small class, best learned as two generations: linezolid, the workhorse with the full toxicity story, and tedizolid, the once-daily successor.

1. Name: First-generation oxazolidinone

1. Note: The original and most-used agent; carries the class-defining MAO inhibition and duration-dependent mitochondrial toxicities.

Name: Linezolid

Zyvox

Route: Oral/IV

Note: Used for resistant gram-positive infection including VRE infection and MRSA pneumonia; oral bioavailability is essentially complete. [1][2] [1]

2. Name: Second-generation oxazolidinone

2. Note: Once-daily administration and a short treatment course; shown non-inferior to linezolid in acute bacterial skin and skin-structure infection.

Name: Tedizolid

Sivextro

Route: Oral/IV

Note: Approved in 2014 for acute bacterial skin and skin-structure infection, with potent activity against MRSA. [3] [3]

Mechanism of action

Summary: Oxazolidinones bind the 23S rRNA of the bacterial 50S ribosomal subunit and prevent assembly of the functional 70S initiation complex, blocking protein synthesis at its very first step. The effect is generally bacteriostatic against staphylococci and enterococci, with bactericidal activity described against streptococci.

Target: 23S rRNA of the bacterial 50S ribosomal subunit

Cidal Static: bacteriostatic (bactericidal against streptococci)

Kill Kinetics: time-dependent

1. Step: 1

1. Title: The drug reaches the ribosome

1. Detail: Oxazolidinones act on the bacterial ribosome itself, the machinery every protein the organism makes must pass through. [1]

2. Step: 2

2. Title: It binds 23S rRNA on the 50S subunit

2. Detail: The binding site is the 23S rRNA component of the large (50S) ribosomal subunit, at the region where initiation would otherwise occur. [1]

3. Step: 3

3. Title: The 70S initiation complex cannot form

3. Detail: With the drug in place, the 50S and 30S subunits cannot assemble into a functional 70S initiation complex, so translation is blocked before the first peptide bond - earlier than any other protein synthesis inhibitor class acts. [1]

4. Step: 4

4. Title: Growth stops rather than the cell lysing

4. Detail: Because existing proteins are untouched and no lethal error is introduced, the effect is generally bacteriostatic against staphylococci and enterococci; streptococci are an exception, against which activity is described as bactericidal. Whether the static label truly holds against enterococci in vivo is debated, but bacteriostatic is the accepted classification. [1]

5. Step: 5

5. Title: The same trick works on human mitochondria

5. Detail: Mitochondrial ribosomes resemble bacterial ribosomes closely enough that linezolid inhibits mitochondrial protein synthesis as well - the mechanistic root of the lactic acidosis, neuropathy and marrow toxicity seen on prolonged therapy. [2][1]

Spectrum of activity

Intro: A deliberate gram-positive specialist: the class exists for organisms that have defeated beta-lactams and vancomycin.

Subclass Id: class-wide

1. Staphylococcus aureus, including MRSA
2. Enterococci, including vancomycin-resistant enterococci (VRE)
3. Streptococci
1. Gram-negative bacteria
2. No useful role outside gram-positive infection [1][2][3]

Major clinical uses

1. Drug: Linezolid
1. Organism: Vancomycin-resistant enterococci (VRE)
1. Indication: Serious or problematic enterococcal infection resistant to vancomycin
1. Line: reserve
1. Note: One of the defining indications for the class - an organism with few alternatives. [2]
2. Drug: Linezolid
2. Organism: Methicillin-resistant Staphylococcus aureus (MRSA)
2. Indication: Pneumonia and other serious infection caused by resistant staphylococci
2. Line: targeted
2. Note: Complete oral bioavailability makes oral step-down from intravenous therapy practical. [2][1]
3. Drug: Linezolid
3. Organism: Resistant gram-positive organisms
3. Indication: Skin and soft-tissue infection where resistant gram-positive pathogens are proven or suspected
3. Line: targeted
3. Note: Stewardship matters: reserving the class protects it, since it is often the last oral option for these organisms. [1]
4. Drug: Tedizolid
4. Organism: Gram-positive organisms including MRSA
4. Indication: Acute bacterial skin and skin-structure infection
4. Line: targeted
4. Note: Shown non-inferior to linezolid in randomised trials; given once daily, orally or intravenously. [3]

Pharmacokinetics

1. Drug: Linezolid
1. Route: Oral/IV
1. Absorption: Oral bioavailability essentially complete (~100%); oral and intravenous exposures are equivalent
1. Csf Penetration: See prescribing reference
1. Metabolism: Non-enzymatic oxidation to inactive metabolites; not a significant CYP450 substrate
1. Elimination: Renal and non-renal; see prescribing reference
1. Half Life: See prescribing reference
1. Dose Adjust: Weekly complete blood counts on prolonged therapy; review serotonergic co-medication before starting [1]

2. Drug: Tedizolid

2. Route: Oral/IV

2. Absorption: Given orally or intravenously with once-daily administration

2. Csf Penetration: See prescribing reference

2. Metabolism: Prodrug (tedizolid phosphate) converted to active tedizolid

2. Elimination: See prescribing reference

2. Half Life: Long enough to support once-daily administration

2. Dose Adjust: See prescribing reference [3]

1. The class's practical superpower is the oral route: linezolid's near-complete bioavailability means a resistant gram-positive infection that would once have demanded an intravenous line can be treated by mouth. [1]

2. Toxicity tracks duration more than dose route: lactic acidosis is described after one to eight weeks of linezolid, and higher doses, longer courses and chronic liver or renal disease all raise complication risk. [2]

3. This page gives no dose regimens by design. Doses depend on indication, organism, age, weight and organ function, and belong in a prescribing reference used by the treating clinician.

Adverse effects

Effect: Gastrointestinal upset and headache

Detail: Nausea, diarrhoea, abdominal discomfort and headache are the common, usually self-limiting effects reported with both linezolid and tedizolid. [1][3]

1. Effect: Serotonin syndrome

1. Detail: Linezolid's reversible non-selective MAO inhibition means co-administration with SSRIs, SNRIs, MAOIs or other serotonergic drugs can precipitate serotonin syndrome; tedizolid carries the same warning as a rare event.

1. Action: Review the medication list for serotonergic agents before starting, and treat agitation, clonus, hyperthermia and autonomic instability on therapy as serotonin syndrome until excluded. [1][3]

2. Effect: Myelosuppression

2. Detail: Thrombocytopenia is the characteristic cytopenia, with anaemia and leukopenia also described; risk rises with courses beyond about two weeks and it is generally reversible on stopping.

2. Action: Monitor complete blood counts weekly on prolonged therapy and reassess the drug if counts fall. [1]

3. Effect: Peripheral and optic neuropathy

3. Detail: Associated with prolonged courses; unlike the marrow suppression, neuropathy resolves slowly and may be permanent.

3. Action: Ask about numbness, paraesthesiae and visual change on long courses, and reassess therapy promptly if they appear. [1][2]

4. Effect: Lactic acidosis

4. Detail: Attributed to inhibition of mitochondrial protein synthesis; typically arises after one to eight weeks of therapy, can be fatal, and often resolves rapidly once linezolid is stopped. A mitochondrial DNA polymorphism (A2706G) has been reported in predisposed patients.

4. Action: Check lactate in any patient on linezolid with unexplained acidosis, tachypnoea or malaise, and stop the drug if confirmed. [2]

1. Drug: Linezolid

1. Effect: Carries the full duration-dependent toxicity profile - myelosuppression, neuropathy and lactic acidosis - plus documented hepatic mitochondrial injury with microvesicular steatosis in lactic acidosis cases. [2][1]

2. Drug: Tedizolid

2. Effect: Low rate of transient aminotransferase elevations and no link to clinically apparent liver injury to date; serotonin syndrome, thrombocytopenia and neuropathy are listed as rare severe events, with less accumulated long-course evidence than linezolid. [3]

Contraindications, precautions and interactions

Concurrent use with monoamine oxidase inhibitors, because linezolid itself inhibits monoamine oxidase and the combination compounds the risk of serotonin syndrome and hypertensive reactions. [1]

1. Serotonergic co-medication - SSRIs, SNRIs, tricyclics and related agents - where the combination is avoided unless the indication is compelling and the patient is watched for serotonin syndrome. [1]
2. Planned prolonged therapy, pre-existing cytopenias, and chronic liver or renal disease, all of which raise the risk of the duration-dependent toxicities. [1][2]
3. Large intake of tyramine-rich foods during therapy, because MAO inhibition can turn dietary tyramine into a pressor response. [1]
1. With: SSRIs, SNRIs, MAOIs and other serotonergic drugs
 1. Effect: Risk of serotonin syndrome through additive serotonergic activity on top of linezolid's MAO inhibition. [1]
 2. With: Adrenergic agents such as sympathomimetics
 2. Effect: Potential for enhanced pressor response because monoamine breakdown is inhibited. [1]
 3. With: Tyramine-containing foods (aged cheese, cured meats, fermented products)
 3. Effect: Risk of a tyramine pressor reaction while MAO is inhibited. [1]

Resistance mechanisms

1. Mechanism: Mutation of the 23S rRNA binding site
 1. Detail: Point mutations in the 23S rRNA genes alter the drug's ribosomal target and are the classic route to oxazolidinone resistance, described in enterococci and staphylococci under prolonged exposure.
 1. Linezolid-resistant enterococci
 2. Linezolid-resistant staphylococci
 1. Countermeasure: Reserve the class for proven need and keep courses as short as the infection allows. [1]
 2. Mechanism: Unique binding step limits cross-resistance
 2. Detail: Because oxazolidinones block formation of the 70S initiation complex - a step no other class targets - organisms resistant to macrolides, lincosamides or aminoglycosides are not expected to carry pre-existing cross-resistance to this class.
1. MRSA
2. VRE
 2. Countermeasure: This is the property stewardship is protecting: resistance here must arise on its own rather than being imported from another class. [1]

Stewardship Note: Oxazolidinones are often the last oral option for MRSA and VRE. Every casual course erodes that position, so the class is held in reserve for documented resistant gram-positive infection, and duration is kept short both for stewardship and because the drug's own toxicities are duration-dependent.

Comparison tables

Title: Linezolid versus tedizolid

Caption: The two agents at a glance. Actual therapy choices belong to the treating clinician and a current prescribing reference.

1. Key: feature
 1. Label: Feature
 1. Is Row Header: true
 2. Key: linezolid
 2. Label: Linezolid
 3. Key: tedizolid

3. Label: Tedizolid

1. Feature: Generation

1. Linezolid: First (approved 2000)

1. Tedizolid: Second (approved 2014) [2][3]

2. Feature: Routes

2. Linezolid: Oral or IV; oral bioavailability essentially complete

2. Tedizolid: Oral or IV [1][3]

3. Feature: Administration frequency

3. Linezolid: Multiple daily doses (see prescribing reference)

3. Tedizolid: Once daily [1][3]

4. Feature: Headline indications

4. Linezolid: VRE infection, MRSA pneumonia, resistant gram-positive skin infection

4. Tedizolid: Acute bacterial skin and skin-structure infection [2][3]

5. Feature: Documented long-course toxicity

5. Linezolid: Myelosuppression, peripheral and optic neuropathy, lactic acidosis

5. Tedizolid: Same events listed as rare; less long-course evidence accumulated [1][2][3]

6. Feature: Comparative efficacy

6. Linezolid: Reference standard

6. Tedizolid: Non-inferior to linezolid in skin and skin-structure infection trials [3]

High-yield exam pearls

1. Pearl: Oxazolidinones block initiation, not elongation.

1. Why: By binding 23S rRNA on the 50S subunit they prevent the 70S initiation complex from forming at all - a step earlier than macrolides, lincosamides or aminoglycosides act, which is why cross-resistance with those classes is not expected. [1]

2. Pearl: Linezolid is a hidden MAO inhibitor.

2. Why: It reversibly and non-selectively inhibits monoamine oxidase, so co-administration with SSRIs, SNRIs, MAOIs or other serotonergic drugs can precipitate serotonin syndrome, and large amounts of tyramine-rich food can provoke a pressor response. [1]

3. Pearl: The toxicity clock starts at two weeks.

3. Why: Myelosuppression, particularly thrombocytopenia, is associated with treatment beyond about two weeks, which is why courses are kept short and blood counts are monitored weekly on longer therapy. [1]

4. Pearl: The long-course toxicities are mitochondrial.

4. Why: Human mitochondrial ribosomes are similar enough to bacterial ribosomes that linezolid inhibits mitochondrial protein synthesis too - the accepted explanation for lactic acidosis, and the framework for understanding peripheral and optic neuropathy on prolonged use. [2][1]

5. Pearl: Oral linezolid is not a weaker linezolid.

5. Why: Bioavailability approaches 100%, so the oral route achieves exposures equivalent to intravenous therapy - the pharmacokinetic fact behind oral treatment of MRSA and VRE infection. [1]

6. Pearl: Tedizolid's selling points are once-daily administration and a short course.

6. Why: It is given once daily, orally or intravenously, and was shown non-inferior to linezolid in acute bacterial skin and skin-structure infections. [3]

Common exam traps

1. Trap: "Oxazolidinones are bactericidal like other anti-MRSA agents."

1. Correction: They are generally bacteriostatic against staphylococci and enterococci. The honest nuance: activity against streptococci is described as bactericidal, so the static/cidal label depends on the organism. [1]

2. Trap: "Linezolid is safe to continue alongside the patient's SSRI."
2. Correction: Linezolid inhibits monoamine oxidase, and combining it with SSRIs or other serotonergic drugs risks serotonin syndrome. The interaction question is the single most-tested fact about this class. [1]
3. Trap: "Oxazolidinones cover resistant gram-negatives too."
3. Correction: The class is a gram-positive specialist. Its clinical role is MRSA, VRE and other resistant gram-positive infection; it has no useful role against gram-negative pathogens. [1][2]
4. Trap: "Neuropathy from linezolid recovers like the thrombocytopenia does."
4. Correction: Myelosuppression is generally reversible on stopping the drug, but peripheral and optic neuropathy resolve slowly and may be permanent. [2]
5. Trap: "Unexplained acidosis on long-term linezolid is probably sepsis."
5. Correction: Lactic acidosis is a recognised linezolid toxicity from mitochondrial injury, typically arising after one to eight weeks of therapy; it can be fatal but often resolves quickly once the drug is stopped. [2]

Self-test questions

1. Question: Oxazolidinones interrupt bacterial translation at a point no other antibiotic class attacks. Where is it?
 1. At the 16S rRNA A-site of the 30S subunit, causing the genetic code to be misread
 2. At the peptidyl transferase centre, halting elongation of the growing chain
 3. At the 23S rRNA of the 50S subunit, preventing a functional 70S initiation complex from assembling
 4. At the translocation step, freezing movement of peptidyl-tRNA between ribosomal sites
1. Explanation: The binding site sits on the 23S rRNA component of the large subunit, and while the drug is there the two subunits cannot come together into a working 70S initiation complex. Translation is therefore stopped before the first peptide bond rather than interrupted part-way through, which is what makes this class mechanistically unlike every other ribosomal agent. [1]
2. Question: A patient established on an SSRI is about to start linezolid. Which property of the drug makes that combination hazardous?
 1. It saturates hepatic glucuronidation, so the SSRI accumulates
 2. It blocks the presynaptic serotonin reuptake transporter directly
 3. It induces CYP2D6 and generates toxic SSRI metabolites
 4. It is a reversible, non-selective inhibitor of monoamine oxidase
2. Explanation: Monoamine oxidase is the enzyme that degrades serotonin, and linezolid switches it off reversibly and without selectivity. Adding a serotonergic agent on top lets serotonin accumulate, so SSRIs, SNRIs, tricyclics and MAOIs are what to look for on the medication list before treatment starts. The same enzyme block explains why a heavy tyramine load from aged or fermented food can produce a pressor reaction. [1]
3. Question: Which statement about the killing behaviour of this class is correct?
 1. Uniformly bactericidal, in line with the other agents used against MRSA
 2. Generally bacteriostatic against staphylococci and enterococci, with bactericidal activity described against streptococci
 3. Bacteriostatic against every susceptible organism, with no exceptions
 4. Bactericidal against enterococci and bacteriostatic against streptococci
3. Explanation: The label depends on which organism is in front of you. Staphylococci and enterococci are held in growth arrest, whereas streptococci are reported to be killed. Since proteins already made are left untouched and no lethal misreading is introduced, arrest is the default outcome, and bacteriostatic remains the accepted classification even though behaviour against enterococci in patients is argued over. [1]
4. Question: Why is an oxazolidinone a poor choice for a resistant gram-negative bloodstream infection?
 1. The class is a gram-positive specialist with no useful role against gram-negative pathogens
 2. Gram-negative organisms have no 50S ribosomal subunit for the drug to bind
 3. Gram-negative organisms all carry pre-existing 23S rRNA mutations

4. The class exists only as an oral formulation, which such infections cannot rely on

4. Explanation: Its clinical territory is deliberately narrow: resistant gram-positive disease, chiefly MRSA and vancomycin-resistant enterococci, with nothing to offer against gram-negative pathogens. The route distractor fails too, since linezolid can be given intravenously as well as by mouth. [1][2]

5. Question: Which linezolid adverse effect is least likely to resolve completely once the drug is stopped?

1. Thrombocytopenia
2. Lactic acidosis
3. Nausea and diarrhoea
4. Peripheral and optic neuropathy

5. Explanation: Marrow suppression generally recovers after withdrawal, and the acidosis often settles quickly once the drug is stopped as well. Nerve and optic involvement is the outlier, because recovery there is slow and the deficit may prove permanent. [2]

6. Question: A patient several weeks into linezolid therapy develops unexplained lactic acidosis. What is the accepted explanation?

1. Direct renal tubular toxicity producing a renal tubular acidosis
2. Drug-induced haemolysis releasing lactate from red cells
3. Inhibition of mitochondrial protein synthesis, because mitochondrial ribosomes closely resemble bacterial ones
4. Chelation of thiamine, reproducing a beriberi-like state

6. Explanation: Human mitochondria run ribosomes similar enough to bacterial ones that the drug throttles them too. That injury is the accepted origin of the acidosis, which characteristically appears somewhere between one and eight weeks into treatment, can be fatal, and frequently clears rapidly after the antibiotic is withdrawn. A mitochondrial DNA polymorphism, A2706G, has been reported in patients who appear predisposed. [2][1]

7. Question: What makes oral linezolid clinically interchangeable with the intravenous formulation?

1. The tablet is activated by first-pass metabolism into a more potent species
2. Oral bioavailability is essentially complete, so exposures by either route are equivalent
3. Gut bacteria convert the tablet into a longer-acting derivative
4. The oral form is absorbed poorly and is therefore restricted to mild infection

7. Explanation: Absorption approaches 100%, so a tablet delivers what an infusion delivers. That single pharmacokinetic fact is what allows resistant gram-positive infection to be treated by mouth, or stepped down from a drip, instead of tethering the patient to intravenous access. [1]

8. Question: How does an organism become resistant to oxazolidinones, and why is imported cross-resistance uncommon?

1. Point mutation of the 23S rRNA binding site; no other class attacks the initiation step, so resistance has to arise independently
2. Acquisition of a beta-lactamase, so resistance travels together with penicillin resistance
3. Upregulation of efflux pumps shared with the macrolides, giving broad cross-resistance
4. Enzymatic acetylation of the drug, encoded on the same plasmids as aminoglycoside resistance

8. Explanation: Mutations in the 23S rRNA genes reshape the ribosomal target and are the classic escape route, described in enterococci and staphylococci exposed for long periods. Because the drug acts at a step nothing else targets, an organism already resistant to macrolides, lincosamides or aminoglycosides is not expected to arrive resistant to this class as well. That is exactly the property stewardship protects by reserving the drug and keeping courses short. [1]

Frequently asked questions

1. Question: Are oxazolidinones bactericidal or bacteriostatic?

1. Answer: Generally bacteriostatic against staphylococci and enterococci, with bactericidal activity described against streptococci. Whether the static label fully captures behaviour against enterococci in vivo is debated, but bacteriostatic

is the accepted classification - and it clearly has not stopped the class treating serious VRE and MRSA infection. [1]

2. Question: Why does linezolid cause serotonin syndrome?

2. Answer: Because it is a reversible, non-selective inhibitor of monoamine oxidase, the enzyme that degrades serotonin. Add an SSRI or another serotonergic drug and serotonin accumulates - the classic exam-and-real-life interaction for this class. [1]

3. Question: Why do the serious toxicities appear only on longer courses?

3. Answer: The accepted explanation is mitochondrial: human mitochondrial ribosomes resemble bacterial ribosomes, so linezolid slowly throttles mitochondrial protein synthesis. Marrow suppression emerges beyond about two weeks, and lactic acidosis is described after one to eight weeks of therapy. [1][2]

4. Question: Which linezolid toxicities recover and which may not?

4. Answer: Myelosuppression is generally reversible after stopping the drug, and lactic acidosis often resolves quickly on withdrawal. Peripheral and optic neuropathy are the exceptions - they resolve slowly and may be permanent. [1][2]

5. Question: What does tedizolid add over linezolid?

5. Answer: Once-daily administration, oral or intravenous, with trial evidence of non-inferiority to linezolid in acute bacterial skin and skin-structure infection, and to date no link to clinically apparent liver injury. Its rare severe adverse events are listed as the same family as linezolid's. [3]

6. Question: Why is oral linezolid such a big deal for MRSA and VRE?

6. Answer: Because bioavailability is essentially complete, the oral tablet achieves exposures equivalent to the intravenous infusion. Infections by organisms that would otherwise chain a patient to an intravenous line can be treated, or stepped down, by mouth. [1]

References

[1] Linezolid (StatPearls). StatPearls Publishing / NCBI Bookshelf 2024. <https://www.ncbi.nlm.nih.gov/books/NBK539793/>

[2] Linezolid (LiverTox). LiverTox, NIDDK / NCBI Bookshelf 2019. <https://www.ncbi.nlm.nih.gov/books/NBK548245/>

[3] Tedizolid (LiverTox). LiverTox, NIDDK / NCBI Bookshelf 2016. <https://www.ncbi.nlm.nih.gov/books/NBK548697/>

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