Telavancin in the treatment of nosocomial pneumonia: review of the clinical evidence


Nosocomial pneumonia (NP) is a frequent and severe infection arising mainly in intensive care units. Despite recent advances in supportive care and a wide range of prevention measures, it remains a significant cause of patient morbidity and mortality. A risk factor for increased mortality is the inappropriate antimicrobial therapy, often caused by antimicrobial resistance. The increased frequency of multidrug-resistant bacteria, especially methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant strains, has led to an urgent need for new antibiotics. Telavancin is a novel antibiotic-semisynthetic lipoglycopeptide, which deploys a dual mechanism of action that involves the inhibition of cell-wall synthesis and disruption of bacterial cell-membrane barrier functions and is rapidly bactericidal against methicillin-resistant *S. aureus* and Gram-positive bacteria resistant to vancomycin. It has been approved for the treatment of severe NP. This article reviews telavancin’s pharmacological characteristics resulting from clinical trials, giving a detailed picture of recent available data in NP management.

**Keywords:** hospital pneumonia • linezolid • lipoglycopeptides • methicillin-resistant *Staphylococcus aureus* • telavancin • treatment • vancomycin

Nosocomial pneumonia (NP) is the second most common nosocomial infection – accounting for up to 25% of all intensive care unit infections and for more than 50% of antibacterial agent prescriptions – with mortality rates as high as 76% reported under some circumstances in ventilated patients [1–3], or when lung infection is caused by high-risk pathogens. Factors commonly related to this increased mortality are inappropriateness of empiric antibiotic treatment, the existence of bacteremia and the virulence of the microorganism. The most commonly encountered causative pathogens of NP reported are higher-level antibiotic-resistant Gram-negative bacteria, such as *Pseudomonas aeruginosa*, *Acinetobacter spp.*, or methicillin-resistant *Staphylococcus aureus* (MRSA) [1,4]. In an interesting study by Kollef et al. demonstrated that patients infected with MRSA were more likely to receive inappropriate antimicrobial therapy [5]. Moreover, rates of MRSA are over 50% in the USA, accounting for over 125,000 hospitalizations annually, and are still increasing [6].

For decades, vancomycin has been the mainstay of therapy in the treatment of this pathogen. During the 1990s, strains of *S. aureus* with reduced susceptibility to glycopeptides (vancomycin intermediate *S. aureus* [VISA] or glycopeptide-intermediate *S. aureus*, heteroresistant VISA strains [hVISA] and extremely uncommon strains of *S. aureus* fully resistant to vancomycin [VRSA]) have emerged in the clinical arena [7]. At the same time, it has long been recognized that vancomycin exerts a slow bactericidal effect with numerous reports of frank treatment failures on *S. aureus* infections [8,9]. Overall there has been an increase in the minimum inhibitory concentrations (MICs) of vancomycin against MRSA,
Telavancin is a lipoglycopeptide antibiotic with bacterial activity against clinically important Gram-positive bacteria, such as staphylococci (including MRSA, VISA and VRE strains) and streptococci (including penicillin-resistant Streptococcus pneumoniae) as well as Gram-positive aerobic and fastidious aerobic bacterial infections (11,12,102). In vitro, it has a rapid (within 10 min), concentration-dependent bactericidal effect, which is thought to be due to its dual mechanism of action, disrupting bacterial cell wall synthesis and membrane integrity.

Telavancin is a semisynthetic derivative of vancomycin bearing both lipophilic and hydrophilic groups. The chemical structure of telavancin is depicted in Figure 1. It results from the alkylation of the vancomycin for lipid II, a component of the bacterial cell wall, increasing the binding for the terminal diaminopimelic acid residue of the peptidoglycan and preventing cell wall synthesis. It has been demonstrated that telavancin exerts a concentration-dependent increase in bacterial membrane permeability, leading to depolarization of the cell membrane. This lipophilic moiety enhances the affinity of telavancin for lipid II, a component of the bacterial cell membrane (13). In S. aureus, it has been reported using flow cytometry assay, that binding to lipid II is necessary for telavancin to induce membrane depolarization, increasing in permeability and leakage of cellular ATP and K+, although this may not be the decisive step in membrane disruption. This second mechanism of action may promote or be the lone reason for its rapid bactericidal activity. These multiple mechanisms of action may be responsible for the low frequency of spontaneous resistance to telavancin.

Telavancin, like other members of the glycopeptide class, is a large compound with poor oral bioavailability, and thus is administered in a dose of 10 mg/kg over 60 min by an intravenous infusion (14). In a sequential ascending dose study, 54 healthy adult male subjects were given single doses of 5, 10, 15, or 20 mg/kg (mean [SD] weight: 80.5 [10.0] kg) were administered telavancin 0.25–15 mg/kg intravenously (iv) once-daily for up to 7 days (14). Telavancin was reported to have linear pharmacokinetics, supportive of a once-daily regimen. In a single dose of telavancin at the US FDA-approved adult dose (10 mg/kg), the mean Cmax was 87.5 µg/ml, AUC0–24 was 762 (±181) µg h/ml, half-life was 7.5 (±2.8) h, clearance was 11.8 (±1.4) ml/h/kg and the volume of distribution was 115 (±36) ml/kg (14,15).

Wong et al. observed no sex-related differences in the pharmacokinetics of telavancin (16). Telavancin demonstrates a higher degree of plasma protein binding than vancomycin (93 vs 50%) (14). In an in vitro model, the presence of human albumin and human serum had a modest effect on the antibacterial activity of telavancin against the Gram-positive susceptible S. aureus (MSSA) and MRSA as demonstrated by time-kill curves (16,17).

Penetration of telavancin into possible sites of infection has been examined in healthy subjects. Gottfried et al. reported that telavancin penetrated well into epithelial lining fluid (ELF; mean [SD] concentrations of 1.73 [±0.28] µg/ml at 8 h and 0.89 [±0.03] µg/ml at 24 h) and extensively into alveolar macrophages (mean [SD] concentrations of 19.0 [±16.8] µg/ml at 8 h, 45.0 [±22.4] µg/ml at 12 h, and 42.0 [±31.4] µg/ml at 24 h) in 20 healthy volunteers (15). Based on plasma and ELF pharmacokinetic data obtained from 20 healthy subjects, Lodise et al. used population pharmacokinetic modeling and Monte Carlo simulation techniques, and found the mean AUC ELF:free AUC plasma penetration ratio to be 1.01 (±0.96), suggesting that telavancin penetrates well into ELF (18). Unlike daptomycin, the antibacterial activity of telavancin activity is not affected by pulmonary surfactant (19). Telavancin achieves good penetration into skin blister fluid, with the AUC in blister fluid approximately 40% of that in plasma following three daily doses of telavancin 7.5 mg/kg (14).

Published data in humans is lacking to characterize the penetration of telavancin into the CNS. However, in an experimental animal meningitis model, the penetration of telavancin into inflamed meninges was approximately 2%, while penetration into noninflamed meninges was less than 1% (20). Preliminary results from an in vitro MRSA model suggest that free drug AUC/MIC ratios of 50–100 are associated with a 1- to 2-log decrease in bacterial counts and minimal resistance emergence (14). An AUC/MIC ratio of 50, corresponding to a human dosing of 10 mg/kg once-daily and a minimum concentration in plasma of 5 µg/ml, was the lowest concentration that resulted in no bacterial growth at 24 h, while maximal activity was observed at total AUC/MIC of 404 (14).

The post-antibiotic effect (PAE) of telavancin against most Gram-positive organisms has been reported to range from 4–6 h, which is at least four times longer than the PAE observed with vancomycin. In one study, the PAE of telavancin against MSSA, MRSA and VISA strains were ≥84 (21,22). The pharmacokinetic properties of telavancin in pediatrics (<18 years of age) and pregnant females have not been studied.

The primary mode of elimination of telavancin from the body is via the renal route, with up to 70% of the dose excreted in the urine as unchanged drug. When it was administered at doses between 7.5 and 15 mg/kg iv. over 1 h, the mean elimination half-life ranged from 6.0 (±0.6) to 7.5 (±1.3) h (14,15). The dose of telavancin for patients with Creatinine clearance ranging from 30–50 ml/min should be 75% of the dose administered to healthy adults, whereas.

<table>
<thead>
<tr>
<th>Table 1. Pharmacokinetic properties of telavancin in healthy subjects.</th>
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<tbody>
<tr>
<td>10 mg/kg/d single dose over 2 h (n = 5)</td>
</tr>
<tr>
<td>Cmax (µg/ml)</td>
</tr>
<tr>
<td>AUCt (µg h/ml)</td>
</tr>
<tr>
<td>AUC0–24 (µg h/ml)</td>
</tr>
<tr>
<td>Vd (ml/kg)</td>
</tr>
<tr>
<td>Cl (ml/kg)</td>
</tr>
<tr>
<td>t1/2 (h)</td>
</tr>
</tbody>
</table>

Values listed are mean ± SD.

Cl: Clearance; Cmax: Maximum concentration; NR: Not reported; t1/2: Elimination half-life; Vd: Volume of distribution.

Data taken from (14,15,16).
patients with Creatinine clearance ranging from 10–30 mL/min should be administered the full dose (10 mg/kg) every 48 h [25]. Hepatic impairment does not appear to influence telavancin CL <[26].

In order to elucidate any potential synergy, antagonism or indifference resulting from combinations of telavancin with other antimicrobial agents, a checkerboard methodology was employed to yield a fraction inhibitory concentration index. The results of the study demonstrated the absence of antagonism with a halving of combined inoculations and synergy with telavancin combined with cefepime or piperacillin/tazobactam against VISA and VRSAs isolates [26].

Another study was conducted to examine the synergistic activity of telavancin in combination with the antibiotics rifampicin, gentamicin, ceftriaxone, oxacillin, meropenem and ciprofloxacin against 40 strains of S. aureus community-acquired MRSA (15), hospital-acquired MRSA (12), VISA (8), VRSAs (3) and IVISA (2). Telavancin was reported to have synergy when combined with all the examined antibiotics, but the highest synergy rates were observed at 24 h with subinhibitory concentrations of telavancin were combined with clinically relevant, subinhibitory concentrations of gentamicin, ceftriaxone, meropenem and rifampicin [27].

**Antimicrobial activity**

Telavancin is active in vitro against a broad spectrum of Gram-positive organisms (Table 2). The spectrum of its antimicrobial activity closely mirrors that of glycopeptides, except that telavancin maintains its activity against organisms with decreased susceptibility to glycopeptides. MIC<sub>90</sub> are generally two- to sevenfold lower than vancomycin against <i>S. pneumoniae</i>, <i>S. aureus</i>, <i>Staphylococcus epidermidis</i>, Enteroctococcus faecalis and Enteroctococcus faecium [18,22,31–34]. The MIC<sub>90</sub> of telavancin of the <i>S. aureus</i> and coagulase-negative staphylococci strains is ≤1 µg/mL, regardless of the methicillin resistance. Mendes et al. studied the in vitro activity of telavancin against 1071 clinical isolates of MRSA and 950 clinical isolates of coagulase-negative staphylococci strains collected from 28 hospitals in 13 European countries in 2007–2008 [25]. All isolates were inhibited at a concentration of 0.05–0.25 µg/mL. Results from a European surveillance study showed that telavancin MICs range between 0.06 and 0.5 µg/mL for both MSSA and MRSA, which was two- to four-fold lower than that for vancomycin, four- to 80-fold lower than that for linezolid and twofold lower than that for daptomycin [26]. Telavancin was found to be highly active against heterogeneous VISA, maintaining excellent bactericidal activity both at low and high inocula, and at peak and trough concentrations [26]. This activity was superior to vancomycin and linezolid at both inocula.

The development of resistance to telavancin in vitro has been assessed using two different methods including strains of <i>S. aureus</i>, <i>epidermidis</i>, <i>E. faecium</i>, <i>S. pyogenes</i>, Streptococcus pneumonia and <i>S. pneumoniae</i>. No spontaneous resistant mutants were detected when breakthrough growth was evaluated following the exposure of high bacteria inocula to subinhibitory concentrations of telavancin [26]. In another study of Kosowska-Shick et al. comparing the activity of telavancin with other antistaphylococcal agents against MRSA strains, they noticed that single-step mutation frequencies for telavancin (4.0 × 10<sup>−10</sup> to −2.9 × 10<sup>−8</sup>) were lower than the spontaneous mutation frequencies obtained with the comparators [26]. In agreement with these observations, no resistant mutants have been detected for any strain isolated in telavancin clinical trials to date [26]. Evaluation of telavancin against biofilm-producing <i>S. aureus</i>, <i>epidermidis</i> and <i>E. faecalis</i> revealed that MICs for telavancin were eight- to 16-times lower than vancomycin, and telavancin concentrations lower than respective MICs of the isolates inhibited the development of biofilm [26]. These results might be explained by the secondary mechanism of action unique to this drug.

Telavancin has been reported to demonstrate potent in vitro activity against vancomycin susceptible enterococci, for susceptible <i>E. faecalis</i>, the MIC<sub>90</sub> is 0.5–1 µg/mL (compared with 2 µg/mL for vancomycin). However, it was much less active against vanA-positive VRE (MIC<sub>90</sub> 8–16 µg/mL) and had modest activity against vanB-positive VRE (MIC<sub>90</sub> 2 µg/mL) [26]. In vitro data suggests more potent activity (fourfold to 32-times) against VanA VRE strains compared with VanA strains [22]. For <i>E. faecalis</i>, the MIC<sub>90</sub> was 0.25–5 µg/mL. A slightly lower MIC<sub>1/2</sub> (one dilution step) was observed for telavancin against <i>E. faecalis</i> compared with <i>E. faecalis</i>. In a study of Fiegel et al., the antimicrobial activities of telavancin and six comparators were evaluated against 440 isolates of anaerobic bacteria. Telavancin and all comparators demonstrated excellent activity against Gram-positive anaerobes (<i>M. oryzae</i> 2 µg/mL) and was the most potent agent tested against Clostridium difficile (<i>M. oryzae</i> 0.25 µg/mL) [27]. Telavancin’s activity against Gram-negative anaerobes and <i>Corynebacterium sp</i> was assessed in an in vitro model, where it inhibited 90% of the anaerobic isolates and 100% of the <i>Corynebacterium</i> isolates at concentrations of 1 µg/mL or less [27]. The MIC<sub>90</sub> of telavancin for all strains tested was ≤2 µg/mL [27].

**Clinical efficacy**

Unlike other glycopeptides, the FDA approved telavancin for use in complicated skin and soft tissue infections but not for the treatment of HAP, with further studies recommended aimed at a mortality end point [26].

**Hospital-acquired pneumonia**

Two methodologically identical Phase III, multinational, randomized, double-blind, active-controlled clinical trials (ATTAIN 1 and ATTAIN 2) compared the efficacy and tolerability of telavancin and vancomycin in the treatment of hospital-acquired pneumonia, including VAP [26].

The first data from the HAP trials (ATTAIN) were published in 2011 by Rubinstein et al. [26] Table 2 and the rest were available and have been presented as abstracts at scientific meetings. In the ATTAIN study, a total of 1503 patients with HAP, from 38 countries, were enrolled to receive either vancomycin 1 g every 12 h or telavancin 10 mg/kg every 24 h in combination with aztreonam or piperacillin–tazobactam if a polymicrobial infection was identified. <i>S. aureus</i> was the most common pathogen isolated from the respiratory tract with approximately 60% (464 pts) MRSA. In total, 658 (44%) patients were clinically evaluable. Clinical cure (the primary end point) was similar in both groups with significantly better cure rates obtained in telavancin treated high MIC isolates (≥2µg/mL; treatment difference 12.5%, 95% CI 0.5–23%, p = 0.08). Lower cure rates in patients with mixed infections were observed in the telavancin group. In patients with mixed infections who received adequate Gram-negative coverage, cure rates were similar to those seen when the two groups were compared. Moreover, telavancin demonstrated potent activity against recent Gram-positive HAP isolates; MICs for all isolates ranged from 0.008 to 1 µg/mL [27].

The secondary objective was to perform a pooled analysis of the superiority of telavancin over vancomycin in patients with a confirmed MRSA infection.
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Nefrotoxicity has been noted with telavancin, with increased serum creatinine levels of up to 1.5 times the pre-treatment level reported in 16% of patients. Phase III trials included patients who received telavancin and vancomycin. Telavancin was well-tolerated, with a low rate of bacterial resistance. The drug is generally well-tolerated and has been approved for the treatment of hospital-acquired pneumonia due to MSSA and VRE.

**Conclusion & Future Perspective**

The development and introduction of new antibiotics has, unfortunately not kept pace with the development of bacterial resistance and the need for new agents is becoming acute. Telavancin has the potential to be a useful tool in the treatment of Gram-positive pulmonary infections, including drug-resistant organisms, particularly MRSA and it has the advantage of once daily administration. Additionally, unlike other new antibiotics against Gram-positive pathogens, telavancin is bactericidal and is not inactivated by pulmonary surfactant. The drug is generally well-tolerated and has the most common AEs associated with telavancin being rash and nausea. The low rate of bacterial resistance to telavancin can be related to its multiple mechanisms of action. As data from ATTAIN clinical trials accumulates, telavancin may also be a treatment option in patients with pneumonia due to S. aureus with vancomycin MICs ≥1 µg/ml. More data are needed to visualize the drugs.

**Table 3. Safety parameters for the pooled studies safety population.**

<table>
<thead>
<tr>
<th>Safety parameter</th>
<th>Telavancin group (n %; total n = 751)</th>
<th>Vancomycin group (n %; total n = 752)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Death</strong></td>
<td>150 (20)</td>
<td>140 (19)</td>
</tr>
<tr>
<td><strong>Any TEAE</strong></td>
<td>616 (82)</td>
<td>613 (82)</td>
</tr>
<tr>
<td><strong>Any serious AE</strong></td>
<td>234 (31)</td>
<td>197 (26)</td>
</tr>
<tr>
<td><strong>Discontinued medication due to TEAE</strong></td>
<td>60 (8)</td>
<td>40 (5)</td>
</tr>
<tr>
<td><strong>Renal impairment</strong></td>
<td>74 (10)</td>
<td>57 (8)</td>
</tr>
<tr>
<td><strong>Anemia</strong></td>
<td>64 (9)</td>
<td>85 (11)</td>
</tr>
<tr>
<td><strong>Constipation</strong></td>
<td>70 (9)</td>
<td>71 (9)</td>
</tr>
<tr>
<td><strong>Hypokalemia</strong></td>
<td>61 (8)</td>
<td>80 (11)</td>
</tr>
<tr>
<td><strong>Hypotension</strong></td>
<td>48 (6)</td>
<td>52 (7)</td>
</tr>
<tr>
<td><strong>Nausea</strong></td>
<td>40 (5)</td>
<td>31 (4)</td>
</tr>
<tr>
<td><strong>Decubitus ulcer</strong></td>
<td>39 (5)</td>
<td>44 (6)</td>
</tr>
<tr>
<td><strong>Insomnia</strong></td>
<td>34 (5)</td>
<td>47 (6)</td>
</tr>
<tr>
<td><strong>Peripheral edema</strong></td>
<td>34 (5)</td>
<td>38 (5)</td>
</tr>
</tbody>
</table>

**AE: Adverse event; TEAE: Treatment-emergent adverse event. Data taken from [42].**

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**Efficacy in animal models**

The efficacy of telavancin was assessed in the treatment of pneumonia based on findings from various animal models [30–37]. In a neutropenic murine model of pneumonia, telavancin was more efficacious after 24 h against all seven isolates tested. Furthermore, telavancin efficacy increased from 24–48 h against all seven isolates (range: 0.6 to -2.9 log10 CFU/ml), while an additional 24 h of telavancin treatment resulted in a decrease in efficacy (i.e., increased bacterial density) for two of the seven isolates (range: 0.3 to -2.6 log10 CFU/ml).

**Safety & tolerability**

In clinical trials, telavancin was well-tolerated, with a low incidence of drug discontinuation due to adverse effects. Phase I trials of telavancin in 54 healthy adults found that the most common AEs associated with its treatment were taste disturbance (75 vs 14% placebo) and headache (40 vs 29% placebo) [11,12]. Taste disturbances were mild and reversible without treatment. Headaches were reported throughout the range of doses studied (0.25–10 mg/kg). Other reported AEs included dizziness, nausea and rash in two subjects.

Another noteworthy trial evaluated the effects of telavancin on cardiac repolarization using an ECG [50]. In the study, 160 subjects received placebo, telavancin 7.5 mg/kg, or moxifloxacin (Avelox, Bayer) 15 mg/kg or 400 mg for 3 days. The mean effect on cardiac repolarization with telavancin was less than 5 ms with no dose correlation. Although these findings suggest otherwise, the drug should not be used in patients with congenital long QTc syndrome, prolongation of the QTc interval, uncompensated heart failure, or severe left ventricular hypertrophy.

Telavancin should be administered over 60 min to minimize the risk of infusion-related reactions, such as flushing of the upper body, urticaria, pruritus or rash. As with most antimicrobials, the development of Clostridium difficile-associated diarrhea remains a growing concern [11,44]. Foamy urine is thought to be due to the excretion of cycloextrin, a solubilizing agent incorporated into the intravenous formulation of telavancin.

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Corey et al. reported the clinical outcomes of patients infected with MSSA with vancomycin MICs ≥1 µg/ml [46]. The clinical cure rates in patients who received telavancin and vancomycin were 87.1 and 74.3%, respectively (p < 0.05). Based on this information, telavancin might be a treatment option in patients infected with vancomycin due to MSSA. Telavancin was more efficacious after 24 h against all seven isolates tested. Survival cure rates also favored telavancin over vancomycin in pneumonia.

In an interesting recent report, Grandon et al. compared the efficacy of telavancin and vancomycin in the treatment of pneumonia caused by a collection of variable resistant MRSA strains in a neutropenic murine lung infection model [51]. They found that human-simulated dosing regimens of telavancin and vancomycin resulted in similar efficacies against MRSA strains with vancomycin MICs of <2 µg/ml. Against HIVE strains, similar efficacies were noted for telavancin and vancomycin after 24 h, while telavancin was more efficacious after 48 h against one of the two strains tested. Furthermore, telavancin efficacy increased from 24–48 h against all seven isolates (range: -0.6 to -2.9 log10 CFU/ml), while an additional 24 h of telavancin treatment resulted in a decrease in efficacy (i.e., increased bacterial density) for two of the seven isolates (range: 0.3 to -2.6 log10 CFU/ml).

Upon examination of results obtained from older and severely ill patients [52], telavancin was deemed to be equally effective as vancomycin in treating NP, with cure rates in patients with APACHE II scores of >25 being 48% in the telavancin group and 57% in the vancomycin group (95% CI: 13.8–41.3%). The incidence of adverse events (AEs) was similar between treatment groups [53].

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The development and introduction of new antibiotics has, unfortunately not kept pace with the development of bacterial resistance and the need for new agents is becoming acute. Telavancin has the potential to become a useful tool in the treatment of Gram-positive pulmonary infections, including drug-resistant organisms, particularly MRSA and it has the advantage of once daily administration. Additionally, unlike other new antibiotics against Gram-positive pathogens, telavancin is bactericidal and is not inactivated by pulmonary surfactant. The drug is generally well-tolerated and has the most common AEs associated with telavancin being rash and nausea. The low rate of bacterial resistance to telavancin can be related to its multiple mechanisms of action.

As data from ATTAIN clinical trials accumulates, telavancin may also be a treatment option in patients with pneumonia due to S. aureus with vancomycin MICs ≥1 µg/ml. More data are needed to visualize the drugs.

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**Executive summary**

- Nosocomial pneumonia is a common and fatal infection, with mortality reaching 60% in ventilator-associated pneumonia cases.
- Talavancin is a rapidly bactericidal drug with dual mechanism of action against Gram-positive cocci, including organisms with reduced susceptibility to vancomycin (e.g., vancomycin intermediate Staphylococcus aureus). The low rate of bacterial resistance to talavancin can be related to its mechanisms of action.
- The safety profile of talavancin appears acceptable for the treatment of patients with severe infections, especially with methicillin-resistant S. aureus, with the most common adverse effects being taste disturbance, nausea and foamy urine. Due to its nethrotoxicity, the use in patients with acute renal failure or creatinine clearance <30 ml/min, including patients with acute renal failure or creatinine clearance <30 ml/min, is not recommended.

**References**

Papers of special note have been highlighted as:

- of interest
- important
- of particular interest

23. Comprehensive and chemical-oriented review on different glycopeptides under development.


Crucial safety study with detailed evaluation of the effect of telavancin on the QT interval in healthy individuals.


**Websites**


