Telavancin: A Novel Lipoglycopeptide

Louis D. Saravolatz,1,2 Gary E. Stein,3 and Leonard B. Johnson1,2
1St John Hospital and Medical Center, Detroit, 2Wayne State University School of Medicine and 3Michigan State University School of Medicine, East Lansing, Michigan

Telavancin, a derivative of vancomycin, is a lipoglycopeptide antibiotic that has been shown to be effective for the treatment of complicated skin and skin-structure infections. It has also been effective in the treatment of gram-positive pneumonia. This antibiotic has a dual mechanism of action by inhibiting peptidoglycan synthesis and causing membrane depolarization. Telavancin is consistently active against Staphylococcus aureus, including methicillin-resistant S. aureus, vancomycin-intermediate S. aureus, linezolid-resistant S. aureus, and daptomycin-nonsusceptible strains. The drug is usually administrated intravenously at 10 mg/kg every 24 h. Telavancin is excreted by the kidneys, and thus, dosage adjustments are required in cases of renal failure. Clinical trials have demonstrated non-inferiority, compared with vancomycin, in the treatment of complicated skin and skin-structure infections and pneumonia. Telavancin is associated with higher rates of renal events, altered taste, nausea, and vomiting but lesser rates of pruritus and infusion-related events, compared with vancomycin.

Vancomycin has become the most commonly used antimicrobial agent in many academic health centers in the United States, largely because of the widespread occurrence of methicillin-resistant Staphylococcus aureus (MRSA) [1, 2]. Furthermore, there are now greater numbers of vancomycin-susceptible strains of MRSA that are likely to be associated with higher rates of clinical failure [3]. Recent evidence suggests that MRSA isolates with minimum inhibitory concentrations (MICs) of 1–2 μg/mL do not respond to treatment as well as those with MICs ≤0.5 μg/mL [4]. These findings have prompted active research to discover new glycopeptides with improved antimicrobial properties. Telavancin (TD-6424), a derivative of vancomycin, is a lipoglycopeptide antibiotic that was developed as a novel agent by Theravance to address the challenge of treating resistant gram-positive bacteria infections, specifically MRSA infection [5]. This article will review the chemistry, mode of action, antimicrobial activity, pharmacokinetics and pharmacodynamics, clinical indications, adverse events, and formulary considerations of telavancin.

CHEMISTRY

One approach toward developing newer glycopeptides with improved antimicrobial activity has been the synthesis of lipoglycopeptides, analogs containing hydrophobic groups substituted at the amine position of the disaccharide moiety [6]. Telavancin is structurally similar to glycopeptides but possesses a hydrophobic (decdylaminoethyl) side chain appended to the vancosamine sugar and a hydrophilic (phosphonomethyl aminomethyl) group on the 4 position of amino acid 7 [7]. The decylaminoethyl side chain provides improved binding affinity of the glycopeptide core for D-Ala-D-Ala–containing peptidoglycan intermediates, and the negatively charged phosphonic acid moiety increases the urinary excretion of this compound [7, 8]. The addition of this lipophilic decylaminoethyl substituent to the molecule classifies this antimicrobial as a lipoglycopeptide (Figure 1).

MODE OF ACTION

Telavancin has a dual mechanism of action. It acts by binding the peptidoglycan precursor lipid-linked N-acetyl-glucosamine-N-muramylpentapeptide at the D-Ala-D-Ala terminus. This interaction results in the inhibition of peptidoglycan polymerization (transglycosylation) and subsequent cross-linking (transpeptidation) steps. Telavancin is a potent inhibitor of peptidoglycan synthesis at the transglycosylase step, being associated with a 10-fold greater activity than vancomycin in inhibiting syntheses of peptidoglycan in intact MRSA cells. [8].
The decylaminoethyl side chain promotes interaction with the cell membrane, and this interaction provides improved binding affinity for peptidoglycan intermediates by localizing the molecule to the bacterial cell surface. Telavancin also triggers rapid concentration-dependent dissipation of cell membrane potential [8]. This may occur via binding of lipid intermediate II molecules, which results in membrane pores. Membrane depolarization is further accompanied by leakage of cytoplasmic adenosine triphosphate and potassium ions. This second mode of action is specific for bacterial membranes, not mammalian cells, and appears to contribute to the more rapid bactericidal activity of telavancin, compared with vancomycin.

Telavancin differs from vancomycin in that the majority of the compound is associated with the cell membrane rather than the cell wall. This dual mode of binding allows for contribution of both the carboxylate binding pocket interaction with terminal D-Ala-D-Ala residues and the decylaminoethyl side chain interaction with the cell membrane.

**ANTIMICROBIAL ACTIVITY**

The antimicrobial activity of telavancin is summarized in Table 1 [9–11]. Studies from clinical trials and surveillance studies demonstrate that telavancin has excellent in vitro activity against the species that commonly cause skin and skin-structure infections and gram-positive organisms that cause pneumonia [9]. Telavancin was consistently active against organisms resistant to methicillin, daptomycin, or linezolid. Vancomycin-intermediate \textit{S. aureus} (VISA) and vancomycin-heterogeneous VISA were also susceptible to telavancin. Telavancin showed activity with an MIC of 2–4 \( \mu \text{g/mL} \) for 2 strains of vancomycin-resistant \textit{S. aureus} used in the time kill curve analyses which would suggest that it is active at achievable serum concentrations [12]. One needs to be cautious in concluding that telavancin is active against vancomycin-resistant \textit{S. aureus}, because of the limited number of stains studied at this time. Telavancin does not have activity against the most common types of vancomycin-resistant enterococci (Van A and Van B). However, against Van B and Van C strains, the drug may be active depending on the free concentrations achieved. Notably, telavancin was not affected by the mechanisms that conferred resistance to methicillin, daptomycin, and linezolid. Telavancin is also active against \textit{Clostridium} species, including \textit{Clostridium perfringens} and \textit{Clostridium difficile}, and \textit{Peptococcus anaerobius} (Table 1) [11]. Telavancin was also active against 15 isolates of
Table 1. Comparative in vitro Minimum Inhibitory Concentrations (MICs) of Telavancin, Vancomycin, Linezolid, and Daptomycin for Gram-Positive Organisms

<table>
<thead>
<tr>
<th>Organism (no of isolates tested)</th>
<th>Telavancin</th>
<th>Vancomycin</th>
<th>Linezolid</th>
<th>Daptomycin</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSSA (1217)</td>
<td>0.5</td>
<td>1</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>MRSA (1082)</td>
<td>0.25</td>
<td>1</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>VISA (23)</td>
<td>1</td>
<td>8</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>DNSM (7)</td>
<td>0.5</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Coagulase-negative staphylococci</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methicillin susceptible (100)</td>
<td>0.5</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Methicillin resistant (272)</td>
<td>0.5</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><em>Enterococcus faecalis: vancomycin susceptible</em> (429)</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><em>Enterococcus faecium: vancomycin susceptible</em> (92)</td>
<td>0.25</td>
<td>1</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Van A (223)</td>
<td>8</td>
<td>512</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Van B (17)</td>
<td>2</td>
<td>512</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td><em>Streptococcus pyogenes</em> (68)</td>
<td>0.06</td>
<td>0.5</td>
<td>1</td>
<td>0.06</td>
</tr>
<tr>
<td><em>Streptococcus agalactiae</em> (45)</td>
<td>0.06</td>
<td>0.5</td>
<td>1</td>
<td>0.25</td>
</tr>
<tr>
<td><em>Streptococcus pneumonia</em> (204)</td>
<td>0.03</td>
<td>0.5</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td><em>Actinomyces israelii</em> (13)</td>
<td>0.25</td>
<td>1</td>
<td>16</td>
<td>4</td>
</tr>
<tr>
<td><em>Clostridium difficile</em> (14)</td>
<td>0.25</td>
<td>1</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td><em>Clostridium perfringens</em> (12)</td>
<td>0.125</td>
<td>0.5</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><em>Clostridium ramosum</em> (16)</td>
<td>1</td>
<td>4</td>
<td>8</td>
<td>32</td>
</tr>
<tr>
<td><em>Peptococcus anaerobius</em> (10)</td>
<td>0.25</td>
<td>0.5</td>
<td>8</td>
<td>0.5</td>
</tr>
</tbody>
</table>

**NOTE.** DNSM, daptomycin-nonsusceptible *S. aureus*; MRSA, methicillin-resistant *S. aureus*; MSSM, methicillin-susceptible *S. aureus*; VISA, vancomycin-intermediate *S. aureus*. *S. aureus* breakpoints were for 1 μg/mL for telavancin (proposed), 2 μg/mL for vancomycin, 4 μg/mL for linezolid, and 1 μg/mL for daptomycin [9–11].

*Bacillus anthracis* [13]. In vitro synergy has been demonstrated against *S. aureus* with telavancin when combined with gentamicin and rifampin [14].

**PHARMACOKINETICS**

Glycopeptides are large compounds that are poorly absorbed orally. For treatment of systemic infections, telavancin should be administered by slow intravenous infusion to diminish potential infusion-associated reactions (such as pruritus, flushing, and pain) [15]. In a study of healthy subjects who received multiple ascending doses, telavancin exhibited linear pharmacokinetics from 1 to 12.5 mg/kg, on the basis of maximum concentration (Cmax) and area under the curve (AUC) values [15]. In singles doses of 10 mg/kg of telavancin infused over 120 min in healthy adults, the mean peak serum concentration was 87.5 μg/mL and the area under the serum concentration-time curve (AUCc0–t) was 858 μg·h/mL. The mean half-life (t1/2) and plasma clearance values were 7.5 h and 11.8 mL/h/kg, respectively. Additional studies found that pharmacokinetic values do not change significantly following multiple doses of telavancin [15, 16]. Wong et al also observed no sex-related differences in the pharmacokinetic disposition of telavancin [16]. The post-antibiotic effect of the drug is 1–4 h against *S. aureus* and supports once daily dosing [17].

Telavancin has a high level of protein binding (~93%) and a relatively small volume of distribution (115 mL/kg). Following 3 doses of telavancin (7.5 mg/kg every 24 h) in 8 healthy subjects, the mean Cmax and AUC in plasma and chemically induced blister fluid was 85 and 16 μg/mL and 604 and 241 μg/h·mL, respectively. The mean AUC ratio obtained in blister fluid, compared with that in plasma, was 40% [18]. Good telavancin (10 mg/kg) penetration into pulmonary epithelial lining fluid and alveolar macrophages was observed in healthy subjects throughout the dosing interval [18]. Mean concentrations in epithelial lining fluid peaked at 8 h (3.7 μg/mL) and decreased to 0.9 μg/mL at 24 h. These concentrations were similar to calculated free (unbound) plasma levels [19, 20]. Telavancin exhibits extensive penetration into alveolar macrophages with maximum concentrations (45 μg/mL) occurring at 12 h after the dose [19]. In contrast to daptomycin, pulmonary surfactant did not affect the activity of telavancin in the lung.

Telavancin is eliminated from the body primarily by renal excretion, with 65% (7.5 mg/kg) to 72% (15 mg/kg) eliminated unchanged following multiple doses [15]. Its primary (7-hydroxy) metabolite is also excreted in the urine in a range of 3%–6% of the dose. The clearance of telavancin is not altered in healthy elderly subjects but is significantly diminished in adults with renal impairment (creatinine clearance <30 mL/
resistant pneumococcal strain (telavancin MIC, 0.06 g/mL) [33]. Telavancin was highly bactericidal against a penicillin-resistant VISA strain (telavancin MIC, 4 g/mL) and significantly more effective than vancomycin against the addition of human serum did not influence the bacterial killing effect of telavancin. Moreover, telavancin was able to kill strains of S. aureus that were in a non-growing phase.

The efficacy of telavancin has been studied in several infection models. In immunocompromised mice with bacteremia, telavancin produced greater reductions in blood and spleen bacterial titers of MRSA, including VISA strains, compared with vancomycin [29, 30]. This occurred even though the free AUC/MIC ratio was similar for both drugs. Similar findings were also observed in a murine model of pneumonia [31]. In a rabbit model of aortic valve endocarditis, telavancin was as effective as vancomycin in sterilizing vegetations against a strain of MRSA (telavancin MIC, 1.0 µg/mL; vancomycin MIC, 2 µg/mL) and significantly more effective than vancomycin against a VISA strain (telavancin MIC, 4 µg/mL; vancomycin MIC, 8 µg/mL) [32]. Telavancin sterilized vegetations in 4 of 6 rabbits infected with the VISA strain. In a rabbit meningitis model, the penetration of telavancin into inflamed meninges was 2% [33]. Telavancin was highly bactericidal against a penicillin-resistant pneumococcal strain (telavancin MIC, 0.06 µg/mL) in this model and produced a significant decrease in the viable cell count (−6.12 log). Telavancin was able to sterilize the cerebrospinal fluid in 6 of 10 rabbits, whereas only 3 of 10 rabbits in the vancomycin group had sterile cerebrospinal fluid by 8 h. In a localized osteomyelitis induced in rabbits, telavancin (30 mg/kg every 12 h for 4 weeks) sterilized 12 of 15 (80%) tibial MRSA infections. This success rate was similar to vancomycin and linezolid in this model [34].

In a Sorbact biofilm model, telavancin exhibited bactericidal effects against isolates of S. aureus, including VISA strains [35]. Moreover, telavancin produced greater reductions in bacteria, compared with other glycopeptides. Telavancin has also been shown to provide bactericidal activity in peritoneal dialysis fluid [36]. In this in vitro model, telavancin at 10 µg/mL and 50 µg/mL exhibited significantly better killing than cefazolin and vancomycin against strains of S. aureus.

**PHARMACODYNAMICS**

In contrast to vancomycin, telavancin produces rapid and concentration-dependent killing against both extracellular and intracellular S. aureus [25, 26]. Pharmacodynamic studies in mice have indicated that the surrogate marker that best correlates with the antimicrobial effect of telavancin is the AUC/MIC ratio [27]. In an in vitro kinetic model, the minimum AUC/MIC ratio resulting in >3-log killing without regrowth was 50 (corresponding to a dose of 10 mg/kg) and maximal killing was obtained with an AUC/MIC ratio of 404 [28]. In this model, the addition of human serum did not influence the bacterial killing effect of telavancin. Moreover, telavancin was able to kill strains of S. aureus that were in a non-growing phase.

The eficacy of telavancin has been studied in several infection models. In immunocompromised mice with bacteremia, telavancin produced greater reductions in blood and spleen bacterial titers of MRSA, including VISA strains, compared with vancomycin [29, 30]. This occurred even though the free AUC/MIC ratio was similar for both drugs. Similar findings were also observed in a murine model of pneumonia [31]. In a rabbit model of aortic valve endocarditis, telavancin was as effective as vancomycin in sterilizing vegetations against a strain of MRSA (telavancin MIC, 1.0 µg/mL; vancomycin MIC, 2 µg/mL) and significantly more effective than vancomycin against a VISA strain (telavancin MIC, 4 µg/mL; vancomycin MIC, 8 µg/mL) [32]. Telavancin sterilized vegetations in 4 of 6 rabbits infected with the VISA strain. In a rabbit meningitis model, the penetration of telavancin into inflamed meninges was 2% [33]. Telavancin was highly bactericidal against a penicillin-resistant pneumococcal strain (telavancin MIC, 0.06 µg/mL) in this model and produced a significant decrease in the viable cell count (−6.12 log). Telavancin was able to sterilize the cerebrospinal fluid in 6 of 10 rabbits, whereas only 3 of 10 rabbits in the vancomycin group had sterile cerebrospinal fluid by 8 h. In a localized osteomyelitis induced in rabbits, telavancin (30 mg/kg every 12 h for 4 weeks) sterilized 12 of 15 (80%) tibial MRSA infections. This success rate was similar to vancomycin and linezolid in this model [34].

In a Sorbact biofilm model, telavancin exhibited bactericidal effects against isolates of S. aureus, including VISA strains [35]. Moreover, telavancin produced greater reductions in bacteria, compared with other glycopeptides. Telavancin has also been shown to provide bactericidal activity in peritoneal dialysis fluid [36]. In this in vitro model, telavancin at 10 µg/mL and 50 µg/mL exhibited significantly better killing than cefazolin and vancomycin against strains of S. aureus.

**CLINICAL EXPERIENCE**

Telavancin has been studied in the treatment of complicated skin and skin-structure infections (ATLAS 1 and 2 trials) and hospital-acquired pneumonia (ATTAIN 1 and 2 trials) [37, 38] in 4 double-blinded phase 3 studies. In each study, telavancin was administered in a dosage of 10 mg/kg per 24 h period and compared with vancomycin at 1 g every 12 h. Sites were allowed to adjust vancomycin dosing for renal insufficiency according to their institutional guidelines. Telavancin adjustments on the basis of renal insufficiency were also allowed according to predefined criteria. Results for the 2 protocols were combined for the complicated skin and skin-structure infection studies and included 1897 randomized patients. The all treated population included 1867 of the 1897 patients who received at least 1 dose of study medication. The infections treated included major abscesses requiring surgical incision and drainage, extensive cellulitis, post-surgical/post-traumatic wound infections, infected ulcers (excluding chronic diabetic foot ulcers), and infected burns (<20% of the body surface area). These infections were deemed serious enough clinically to justify parenteral therapy. It was noteworthy that 25% of patients had experienced prior antibiotic therapy failure.

Telavancin met the primary end point, clinical cure on the basis of investigator assessment, by demonstrating non-inferiority compared with vancomycin. The cure rates for the all treated population for the telavancin arm were 77.0% and 75.3% for the vancomycin arm for the combined studies, and the results for each of the 2 studies demonstrated similar findings. S. aureus was the most common pathogen, cultured from 82% (553 of 673) of the telavancin-treated patients and 85% (590 of 698) of the vancomycin-treated patients. MRSA isolates accounted for 63% (717 of 1143) of all S. aureus strains, thus representing the largest clinical trial of MRSA infections performed to date. The clinical response rate was 90.7% for telavancin versus 87.1% for vancomycin, and the microbiologic
response rate was 90% for telavancin versus 86.4% for vancomycin. These differences were not statistically significant. Response rates for other pathogens were similar for vancomycin and telavancin. All isolates had telavancin MICs ≤1 μg/mL and vancomycin MICs ≤2 μg/mL.

In evaluating subgroups for consistency of results, multiple analyses were performed. The only demonstration of a difference was in the population of patients with reduced renal function (P = .086). Although the overall difference in cure rate among patients with renal impairment was not statistically significant, patients with severe reduction in creatinine clearance (<30 mL/min) showed a trend toward lower response rates (69.2% for telavancin vs 89.5% for vancomycin; difference, −20.2%; 95% confidence interval, −40.7% to 5.0%).

To examine telavancin’s efficacy in the treatment of pneumonia, the results from the 2 protocols (ATTAIN 1 and 2) were combined [38]. When combining both studies, a total of 1503 patients were randomized and 658 (312 telavancin and 346 vancomycin) were clinically evaluable at the test-of-cure follow-up, defined as 14 days after receipt of the last dose. The primary end point was clinical cure on the basis of physician-judged resolution of clinical signs and symptoms of hospital-acquired pneumonia. For the ATTAIN trials, telavancin was non-inferior to vancomycin for the primary end point. The clinical cure rate for telavancin was 82.7%, compared with 80.9% for vancomycin. In the analysis of the subgroup of monomicrobial infections due to S. aureus, telavancin was superior to vancomycin (84% vs 74%; difference, 10%; 95% confidence interval, 0.7%–19.2%). In addition, telavancin cure rates were significantly higher (P = .03) than that for vancomycin in patients with pneumonia caused by S. aureus with a vancomycin MIC ≥1 μg/mL. Among the microbiologically evaluable patients who were infected with MRSA alone, treatment with telavancin resulted in numerically higher cure rates of 82%, compared with 74% for vancomycin, but this difference did not reach statistical significance [38].

At the time of this publication, the United States Food and Drug Administration (FDA) has only reviewed the data on complicated skin and skin-structure infection and will soon be reviewing the pneumonia trials. There are no published clinical data on the use of telavancin in catheter-associated bacteremias, infective endocarditis, bone and joint infections, or meningitis.

ADVERSE EVENTS

The overall safety in the complicated skin and skin-structure infection protocols appeared comparable for telavancin and vancomycin during the monitoring of the clinical trials [37]. However, telavancin was associated with a higher number of events than vancomycin for altered taste (22% vs 6%), nausea (26% vs 14%), vomiting (13% vs 7%), foamy urine (12% vs 3%), and constipation (10% vs 7%). Vancomycin had higher rates than telavancin for pruritus (12% vs 6%) and infusion-related events (6% vs 2%). Corrected QT interval (QTc) changes were studied in the complicated skin and skin-structure infection trials and in a separate trial comparing telavancin with moxifloxacin. In a separate trial, the QTc increase was 4.6 ms in telavancin-treated patients and 9.5 ms for the moxifloxacin-treated patients [39]. In the complicated skin and skin-structure infection trials, QTc outliers were similar in the telavancin and vancomycin arms, with 1 patient in the telavancin arm and 2 in the vancomycin arm having a QTc of >500 ms. There was no difference in the cardiac mortality between the 2 treatment arms.

There were more renal events reported among telavancin-treated patients than among vancomycin-treated patients in the complicated skin and skin-structure infection trial (3.4% vs 1.2%). These occurred more often in patients with an elevated baseline serum creatinine level or in those receiving concomitant nephrotoxic drugs. When increases in serum creatinine level were detected, these returned to normal in most patients within 14 days of stopping the drug, and telavancin was stopped because of adverse renal events in <1% of patients [37]. Thus, patients treated with telavancin need to have their serum creatinine level monitored, and dosage adjustments should be made on the basis of estimated creatinine clearances.

Another area of concern that has been raised is the developmental toxicology studies that have demonstrated a decrease in fetal weight and a low incidence of limb defects in rats treated with telavancin. There is no clinical experience in pregnant humans to date, and the decision to use this drug must be left to the clinician deciding appropriate therapy for the patient with an infection. The pregnancy category for this agent is category C. In view of these concerns, the FDA Antimicrobial Advisory Board has recommended a risk reduction strategy that will be defined by the manufacturer with FDA concordance.

FORMULARY ISSUES

Telavancin (Vibativ) will be initially marketed for the treatment of complicated skin and skin-structure infection caused by gram-positive bacteria and represents an important additional agent for the treatment of MRSA infection. Telavancin’s improved potency and bactericidal activity, compared with vancomycin, may prove to be an advantage in the treatment of serious infections, especially those due to MRSA. In addition, it has a lower incidence of infusion reactions. Moreover, its once-daily administration (10 mg/kg) and the lack of serum concentration monitoring may provide additional economic advantages, compared with vancomycin. The other approved agents for MRSA infection include linezolid, tigecycline, and daptomycin. Telavancin’s bactericidal activity represents a potential advantage over linezolid and tigecycline. Telavancin has been shown to be effective for the treatment of pneumonia,
whereas daptomycin is not active in lung tissue, limiting its clinical use.

In a cost-effectiveness analysis of telavancin versus vancomycin for the treatment of skin and skin-structure infection, total hospital costs were similar for both agents [40]. Telavancin was suggested to be cost-effective in the subpopulation of patients infected with MRSA, because of its greater clinical cure rate. The extent of this cost effectiveness will depend on the acquisition cost of telavancin.

As the vancomycin MIC of MRSA continues to rise, the need for additional agents with activity against MRSA will grow. Clinicians at individual hospitals will need to review laboratory information on vancomycin susceptibility against MRSA to assist in critically evaluating the risk benefit of telavancin versus vancomycin or linezolid for the treatment of serious infections due to MRSA. Telavancin is an important addition to our current treatment options for MRSA infection, and the position of this new agent relative to other antimicrobials will evolve with additional clinical trials and experience.

Acknowledgments

Potential conflicts of interest. L.D.S. serves as a consultant for Theravance and has received research funding from Theravance. G.E.S. and L.B.J.: no conflicts.

References

27. Hegde SS, Reyes N, Wiens, T et al. Pharmacodynamics of telavancin...


