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CLSI

CLSI Subcommittee on Antimicrobial Susceptibility Testing

CLSI AST News Update

CELEBRATING 10 YEARS!

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This biannual CLSI AST News Update highlights current issues related to antimicrobial susceptibility testing (AST) and reporting.

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2. Save the date for the next meetings!
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The first edition of the CLSI AST News Update (Vol 1, Issue 1, Spring 2016) described details about the organization and operation of the CLSI AST Subcommittee.

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Featured Article

Rapid Antimicrobial Susceptibility Testing: Bridging Laboratory Diagnostics and Antimicrobial Stewardship

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Introduction

Accurate and timely antimicrobial susceptibility testing (AST) is essential to guide clinicians toward the most effective, targeted regimen as early as possible. Conventional AST methods (eg, broth microdilution, disk diffusion, commercial methods) require 16 to 24 hour (h) bacterial growth in the presence of antimicrobials once bacteria are isolated from the clinical sample. Therefore, 48 to 72 h can elapse from the time the specimen arrives at the laboratory to the AST result. Some conventional commercial AST systems have shorter run times but typically require isolated colonies. Consequently, the slow time-to-result (TTR) for conventional AST methods can delay administration of active antimicrobial therapy.

Enter "rapid" AST (RAST) methods. Currently, there is no consensus definition for RAST, but it is generally applied to methods that provide results significantly earlier than conventional workflows by using direct-from-positive-blood-culture or direct-from-specimen methods and may involve shortened incubation or run times (often < 8 h). Typically, the term "RAST" is used for the standardized and commercial phenotypic methods that report antimicrobial susceptibility or resistance after exposing bacteria to individual antimicrobial agents. There are also rapid genotypic methods that detect specific antimicrobial resistance (AMR) genes rather than directly measuring antimicrobial activity that have been commercially available for over a decade. Throughout this article, these assays are termed "rapid genotypic methods" to distinguish them from phenotypic RAST.

The Importance of Phenotypic RAST for Clinical Decision-Making

Antimicrobial therapy is often initiated empirically before the causative organism and its susceptibility profile are known. Empiric therapy is informed by the suspected site of infection, patient-specific risk factors, local epidemiology, and patterns of AMR. Antimicrobial susceptibility testing enables the transition from empiric to directed therapy by supporting escalation when initial therapy is inactive, de-escalation when narrower therapy is appropriate, and selection among active agents based on clinical and patient-specific considerations.[1] The clinical value of phenotypic RAST therefore depends not only on how quickly susceptibility results are generated, but also on whether those results become available at a clinically meaningful decision point and can be acted upon. Rapid AST may be especially valuable when the consequences of inappropriate or delayed antimicrobial therapy are high, such as in patients with sepsis or bacteremia.[2,3] Because of this, many RAST methods are directed toward positive blood cultures.

Across randomized and observational studies of bloodstream infections, RAST has consistently shortened AST turnaround times and has frequently resulted in earlier antimicrobial modification or optimization. However, improvements in downstream clinical outcomes such as mortality and length of stay have been less consistent.[3-9] A randomized clinical trial of 500 patients with gram-negative bloodstream infections demonstrated faster AST results and earlier antimicrobial modification with rapid organism identification plus phenotypic RAST compared with the standard of care, including earlier escalation among patients with bloodstream infections due to AMR organisms, but it did not demonstrate differences in patient outcomes.[7] Notably, both cohorts included antimicrobial stewardship program involvement. Similarly, in a randomized clinical trial of 850 patients with gram-negative bloodstream infections, phenotypic RAST shortened the time to antimicrobial escalation or de-escalation by 14 hours but did not impact clinical outcomes.[6]

Collectively, the available evidence suggests that the most consistently measurable effects of RAST are related to antimicrobial decision-making, including earlier optimization, escalation when empiric therapy is inactive, and de-escalation when broader therapy is unnecessary. Rapid AST may also facilitate earlier intravenous-to-oral transitions when clinically appropriate, potentially reducing unnecessary intravenous therapy and its associated catheter-related complications, patient inconvenience, and health care costs.[10-12] The impact of RAST may therefore depend not only on the speed and accuracy of the test, but also on how results are integrated into clinical workflows, communicated to providers, and acted upon by antimicrobial stewardship and other clinical decision-makers.

Rapid Antimicrobial Susceptibility Testing: Bridging Laboratory Diagnostics and Antimicrobial Stewardship (*Continued*)

Rapid Genotypic vs Phenotypic RAST Methods

In general, rapid methods can be characterized as genotypic or phenotypic (Table 1).

Table 1. Comparison of Rapid Genotypic AMR Detection and Phenotypic RAST Methods

Rapid Testing Approach	Methodology	Strengths	Drawbacks	Examples
Genotypic	Identifying genes or mutations associated with AMR, typically by nucleic acid amplification testing	• Provides results within minutes to a few hours • Identifies the mechanism of resistance • Typically includes organism identification • Can lead to antimicrobial escalation for organisms with resistance factors identified	• May not predict resistance for all antimicrobial agents • Requires definitive AST by phenotypic methods • Does not detect all mechanisms of resistance or resistance due to a combination of mechanisms	• Molecular panels for positive blood culture: –BIOFIRE® Blood Culture Identification 2 (BCID2) Panel –VERIGENE® Blood Culture –cobas® eplex Blood Culture Identification Panels –Cepheid Xpert® MRSA/SA Blood Culture • Molecular panels for other specimens: –BIOFIRE® Joint Infection Panel –BIOFIRE® Pneumonia Panel
Phenotypic	Determining the susceptibility or resistance of an organism through growth in the presence of the antimicrobial agent	• Predicts susceptibility to an antimicrobial agent independent of known resistance mechanisms • Includes S/I/R interpretations • Most generate an MIC • Can be used as definitive AST • Can guide antimicrobial selection when genotypic results do not reliably predict susceptibility	• Must be paired with a method for organism identification • Does not determine the mechanism of resistance • Requires monomicrobial culture • Limited organism coverage (all are currently limited to common gram-negative pathogens)	• Commercial systems for positive blood culture: –Affinity Biosensors LifeScale –bioMérieux VITEK® REVEAL™ –PhAST Blood Culture Gram-negative Panel –Q-linea ASTar® • CLSI disk diffusion directly from positive blood culture broth • Disk diffusion after automated inoculum preparation –Avails eQUANT™ System

Abbreviations: AST, antimicrobial susceptibility testing; I, intermediate; MIC, minimal inhibitory concentration; MRSA, methicillin-resistant *Staphylococcus aureus*; R, resistant; S, susceptible; SA, *Staphylococcus aureus*.

Genotypic methods identify bacterial pathogens and detect specific AMR markers directly from blood or other clinical specimens within minutes to a few hours. Commonly identified AMR markers include genes encoding carbapenemases (eg, KPC, NDM) or extended-spectrum β-lactamases (eg, CTX-M) for gram-negative bacteria, *mecA/C* for *Staphylococcus* species, and *vanA/B* genes for *Enterococcus* species. Genotypic testing can support earlier antimicrobial decision-making, particularly escalation to an effective antimicrobial for select resistant bacteria and is currently widely incorporated into diagnostic workflows. However, genotypic testing has limitations in characterizing antimicrobial susceptibility, particularly in gram-negative organisms, where AMR targets are limited and where resistance may result from a wide variety and combinatorial effects of resistance mechanisms. Therefore, rapid genotypic methods cannot completely predict the susceptibility and resistance pattern of an isolate.

Rapid Antimicrobial Susceptibility Testing: Bridging Laboratory Diagnostics and Antimicrobial Stewardship (*Continued*)

By contrast, phenotypic RAST can more comprehensively demonstrate the effects of a wider range of resistance mechanisms, as well as the combination of multiple resistance factors on antimicrobial susceptibility. Because of these advantages over rapid genotypic methods, there has been increased focus on and development of phenotypic RAST over the last several years.

Emerging technologies for phenotypic RAST include bacterial mass detection, volatile compound assessment, and image analysis (eg, time-lapse, single cell). All current US Food and Drug Administration (FDA)–cleared commercial systems[13]:

1. Use positive blood culture broth as the sample type, with little or no sample processing.
2. Are used for detecting monomicrobial, gram-negative infections.
3. Require input of an organism identification before final AST results can be reported.
4. Have TTRs ≤ 8 h (ranging from 2.5 to 8 h).

Most systems report quantitative AST results, with a minimal inhibitory concentration (MIC) and categorical interpretation. There is also a commercial inoculum preparation system that performs automated cleanup and concentration of a positive blood culture sample, creating a 0.5 McFarland-equivalent suspension. This system currently is FDA-cleared only for use with the standard CLSI disk diffusion method.[14]

As an alternative to adopting a commercial system, which may be resource prohibitive for some laboratories, CLSI has also addressed the need for an accessible phenotypic RAST option with the publication of the direct-from-blood-culture disk diffusion method (CLSI M100 Tables 3F-1 to 3F-4).[15] This modification to the standard disk diffusion method uses positive blood culture broth as the inoculum instead of a 0.5 McFarland suspension. Breakpoints specific to this direct-from-blood-culture disk diffusion method are available for Enterobacterales, *Pseudomonas aeruginosa,* and *Acinetobacter baumannii* at 8 to 10 h and/or 16 to 18 h read times. It is important to note that the breakpoints in CLSI M100 Tables 2 must not be applied if using this alternative method.

Unlike the rapid genotypic methods, neither the commercial systems nor the CLSI method for phenotypic RAST identifies specific mechanisms of resistance, the knowledge of which may further inform treatment options or provide epidemiological insight. Additionally, they are restricted for use with positive blood culture broth and for gram-negative bacilli only. While not yet FDA-cleared, there are phenotypic RAST assays under development for direct testing from respiratory and urine specimens that include on-board organism identification and AST of gram-positive organisms.

Because of the limitations described above for both rapid genotypic methods and phenotypic RAST, they might be viewed as complementary rather than competing approaches (Table 1). Integrating these approaches will provide information that is both more timely and more comprehensive.[3] However, the optimal test(s) will differ from one institution to another. Some of these considerations are discussed below.

Considerations for Your Laboratory and Institution

Successful implementation of RAST relies on collaboration between the laboratory, the antimicrobial stewardship team, and other institutional stakeholders. Here are some factors to consider for those interested in using RAST for positive blood cultures.

Laboratory Resources and Workflow

- Does the laboratory have sufficient trained staff to report RAST results at the time when they become available?
- Does the laboratory have a method of rapid bacterial identification for phenotypic RAST methods that require identification for reporting?
- Can the laboratory justify and bear the additional cost of RAST testing?

The major benefit of rapid methodologies over conventional AST methods is the shortened TTR. Genotypic methods typically provide results in 2 hours or fewer, in contrast to phenotypic RAST. Commercial RAST platforms require 2.5 to 8 hours, and CLSI's method for direct disk diffusion can be read at 8 to 10 hours. Therefore, laboratories with the ability to both set up and report results around the clock may see a larger impact, especially for phenotypic RAST methods where results are likely to become available on the shift following setup.

Rapid genotypic methods and phenotypic RAST platforms can be used together to provide organism identification and resistance-marker detection alongside AST results. Alternatively, matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) can be validated for performing organism identifications directly from positive blood cultures as described in CLSI M58.[16]

Rapid Antimicrobial Susceptibility Testing: Bridging Laboratory Diagnostics and Antimicrobial Stewardship (*Continued*)

Laboratories should consider the total cost of commercial rapid genotypic and phenotypic methods. This varies depending on method and commercial vendor agreements, and should include capital equipment, reagents or consumables, maintenance, training, quality assurance, staff time, and the opportunity cost of diverting personnel to rapid testing. CLSI's direct disk diffusion test method may have lower equipment requirements, but manual setup, incubation, reading, interpretation, and result entry require staff time. By comparison, commercial RAST systems may offer workflow advantages, including automated signal detection, analysis, and interpretation.

Each example below represents a different hypothetical laboratory with a specific implementation consideration.

- Laboratory A uses a rapid genotypic method that provides organism identification for gram-negative and gram-positive bacteria as well as detection of common AMR genes. The laboratory is considering implementing phenotypic RAST. However, they are only staffed with microbiologists from 6 AM to 11 PM; overnight staffing is minimal with positive blood cultures handled by a central laboratory section that does not have extensive experience performing AST. In considering workflow for setup and result reporting, as well as staffing patterns, the laboratory opts for a commercial phenotypic RAST system that includes automated result interpretation. This enables them to provide results 24/7 for both molecular-based identification with AMR results and phenotypic RAST for blood cultures with applicable gram-negative organisms.
- Laboratory B does not currently perform rapid genotypic testing on positive blood cultures but wants to implement phenotypic RAST. They use MALDI-TOF for routine identification of isolates and decide to validate MALDI-TOF for use with positive blood cultures. When a blood culture signals positive, they will perform a Gram stain; if it is monomicrobial, they will identify the bacteria by MALDI-TOF and perform phenotypic RAST for applicable gram-negative bacteria.

Local Epidemiology and Patient Population

- What is the patient population served by the laboratory, and would they benefit from RAST?
- Are the currently available methods for rapid genotypic and phenotypic RAST appropriate for the pathogens and AMR commonly seen in that setting?

Laboratories should consider characteristics of their local institution that may impact the utility of rapid methods. For example, phenotypic RAST may be beneficial for ensuring patients receive effective therapy in areas with high rates of antimicrobial resistance. In settings with high rates of carbapenemase-producing organisms, rapid genotypic testing can often identify the specific carbapenemase gene, facilitating faster escalation to active therapy. In areas with low AMR, phenotypic RAST may be used less often for escalating therapy, but this testing may provide comprehensive susceptibility profiles to allow de-escalation of antimicrobial agents where appropriate. For example, switching a patient with a pan-susceptible *Escherichia coli* to cefazolin from the empiric vancomycin and piperacillin-tazobactam initiated at presentation.

Additionally, the patient population served by the laboratory may benefit from different approaches. Institutions such as cancer centers may benefit from phenotypic RAST to ensure immunocompromised patients receive effective therapy as quickly as possible. Additionally, medically complex patients may have limited antimicrobial options, so the comprehensive AST profiles provided by phenotypic RAST may be useful.

Finally, laboratories should consider the most common causes of bacteremia in their patients. All phenotypic RAST methods have organism limitations. Therefore, laboratories should review their blood culture data and see which methodologies would provide the best coverage for their local epidemiology.

- Laboratory C is in an area with high rates of extended-spectrum β-lactamase and carbapenemase-producing gram-negative organisms. Use of a commercial rapid genotypic test that identifies carbapenemase genes and CTX-M would likely be beneficial in this setting. For example, a patient with suspected urosepsis is seen in the emergency department where urine and blood cultures are collected. The patient is admitted and given empiric vancomycin and meropenem. Fifteen hours later, the blood cultures signal positive, and a rapid genotypic test on this specimen is positive for *E. coli* and the KPC gene, so the clinical team quickly switches to treatment with meropenem-vaborbactam while awaiting confirmation of susceptibility using conventional phenotypic AST.
- Laboratory D is in an area of low AMR. In accordance with hospital policy, clinically stable patients with uncomplicated urinary tract infections identified as the source of bacteremia may be eligible for discharge on oral antimicrobial agents. Therefore, this laboratory opts to use the CLSI direct-from-positive-blood-culture disk diffusion method to support earlier assessment of oral treatment options for agent-organism combinations with established direct-testing breakpoints.

Rapid Antimicrobial Susceptibility Testing: Bridging Laboratory Diagnostics and Antimicrobial Stewardship (*Continued*)

Clinical Stakeholders

- How can rapid genotypic and phenotypic RAST results be effectively communicated to clinical teams to increase impact?
- What rapid results do clinical teams find beneficial for their specific patient populations, and how do they intend to use these results to impact care?
- Are there policies that can be developed in collaboration with the antimicrobial stewardship team to ensure timely and appropriate actions from rapid results?

A rapid result has limited value if it is not recognized, communicated, or acted upon in a timely manner. Rapid methods, whether genotypic or phenotypic, are unlikely to improve patient care without provider awareness, education, and support. Successful implementation requires clinicians to understand the significance and limitations of rapid results and requires workflows for communicating and acting on those results. Collaboration between the antimicrobial stewardship team and other institutional stakeholders is essential and can increase the impact and effectiveness of rapid results on patient management.[2]

- Laboratory E simultaneously implemented a rapid genotypic test for organism identification and AMR gene detection as well as phenotypic RAST. However, the antimicrobial stewardship team noticed that when results were reported to overnight clinical teams, the results were rarely acted upon until the following day, even if a change in therapy was likely necessary. Therefore, the laboratory worked with the antimicrobial stewardship team and other institutional stakeholders to develop a policy to address this. Now, both rapid genotypic testing results for detection of an AMR gene and phenotypic RAST results are communicated by the laboratory to on-call antimicrobial stewardship pharmacists, who immediately review the patient's current antimicrobial orders, provide the clinical care team with recommendations for therapy based on institutional guidance, and may facilitate administration of an effective first dose.

Laboratories should consult with their antimicrobial stewardship team and other institutional stakeholders to discuss the potential utility of rapid genotypic and phenotypic RAST testing, which approaches would fit into clinical workflows, and how rapid results can be appropriately and quickly used to impact patient care.

Conclusions

Despite the number of phenotypic RAST options available, there are still several unmet needs and areas for future development, such as:

- Additional specimen types (eg, cerebrospinal fluid)
- Additional organism types (eg, gram-positive bacteria, mycobacteria, fungi)
- Expanded test menu options per platform (eg, multiple specimen types, option for isolate testing)

Nonetheless, the importance of phenotypic RAST is recognized, and diagnostic companies are continuing to pursue development of tests to address these needs. The potential clinical value of phenotypic RAST can be considered at the intersection of 3 factors: the importance of the antimicrobial decision, the uncertainty surrounding activity of the current regimen, and the ability of the health care team to act on new information. The greatest benefit is likely when susceptibility information is available at a clinically meaningful decision point and can be translated into timely antimicrobial optimization.

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Case Study

Successful Treatment of Carbapenem-Resistant *Acinetobacter* spp. With Sulbactam-Durlobactam

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Case Study

A patient in his 60s with a history of lymphoma in remission, and ulcerative colitis (UC), which was diagnosed 3 months earlier and complicated by 2 subsequent hospitalizations, presented to the emergency department afebrile because of worsening bloody diarrhea, chest pain, fatigue, and exertional dyspnea. The presentation was initially attributed to a UC flare, and on hospital day 8, the patient developed fever and toxic megacolon with colonic perforation that required total colectomy and ileostomy. The patient was intubated for surgery, then remained intubated in the intensive care unit (ICU) and received empiric metronidazole 500 mg IV every 8 hours and extended infusion piperacillin-tazobactam 4.5 g IV every 8 hours for 3 days.

Blood cultures grew an extended-spectrum β-lactamase (ESBL)-producing *Klebsiella pneumoniae*, and piperacillin-tazobactam was switched to meropenem 1 g IV extended infusion every 8 hours for 7 days. Subsequent blood cultures remained negative, and the patient was extubated but still required oxygen support and was provided with a bilevel positive airway pressure (BiPAP) device. The patient was experiencing increased respiratory secretions; thus, a tracheal aspirate culture was obtained and grew carbapenem-resistant *Acinetobacter baumannii* (CRAB) that was not susceptible to any of the laboratory's routinely tested antimicrobials. A commercial genotypic carbapenemase assay that is US Food and Drug Administration (FDA)-cleared for *Acinetobacter baumannii* and KPC, NDM, VIM, IMP, and OXA-48 was performed on the isolate, with no targets detected. The patient was empirically placed on a 7-day regimen of sulbactam-durlobactam 1 g/1 g IV extended infusion every 6 hours plus meropenem at the same dose as before, with antimicrobial susceptibility testing (AST) of sulbactam-durlobactam confirming the isolate as susceptible the following day (Table 1).

Table 1. Antimicrobial Susceptibility Profile of the Carbapenem-Resistant *A. baumannii* Isolates[a]

Antimicrobial Agent	Tracheal Aspirate		Abdominal Drainage	
	MIC, µg/mL	Interpretation	MIC, µg/mL	Interpretation
Amikacin	> 64	R	> 64	R
Ampicillin-sulbactam	32/16	R	> 32/16	R
Cefepime	> 32	R	> 32	R
Ceftazidime	> 32	R	> 32	R
Ceftriaxone	> 64	R	> 64	R
Ciprofloxacin	> 4	R	> 4	R
Gentamicin	> 16	R	> 16	R
Imipenem	> 8	R	> 8	R
Levofloxacin	> 8	R	> 8	R
Meropenem	> 16	R	> 16	R
Minocycline	2	I	1	S
Piperacillin-tazobactam	> 128/4	R	> 128/4	R
Tobramycin	> 16	R	> 16	R
Trimethoprim-sulfamethoxazole	> 4/76	R	> 4/76	R
Sulbactam-durlobactam[b]	**2/4**	**S**	**2/4**	**S**

Abbreviations: I, intermediate; MIC, minimal inhibitory concentration; R, resistant; S, susceptible.
[a] Testing performed using reference broth microdilution.
[b] Breakpoints for *Acinetobacter* spp.: MIC (µg/mL): S ≤ 4/4, I 8/4, R ≥ 16/4; disk diffusion (mm): S ≥ 17, I 14–16, R ≤ 13.[1]

Successful Treatment of Carbapenem-Resistant *Acinetobacter* spp. With Sulbactam-Durlobactam (*Continued*)

One week after completion of sulbactam-durlobactam and meropenem therapy, culture of an abdominal abscess grew *A. baumannii*, and sulbactam-durlobactam and meropenem were reinitiated while awaiting AST results. AST revealed the isolate to be CRAB with a profile similar to the initial respiratory isolate, including testing susceptible to sulbactam-durlobactam (Table 1). No additional CRAB isolates were recovered while on therapy. The patient remained afebrile and was discharged to a skilled nursing facility to complete a full course of therapy with sulbactam-durlobactam and meropenem for intra-abdominal infection.

Discussion

Colonization with *A. baumannii* is common, and although difficult, it is essential to distinguish colonization from infection.[2] In the case described here, CRAB was recovered from 2 distinct, clinically relevant sources. Given the patient's prolonged oxygen requirement after extubation, respiratory symptoms, and new onset of fevers, the infectious diseases team treated the CRAB as a true pathogen causing infection, rather than simply associated with colonization.

Acinetobacter spp. were responsible for an estimated 0.4% of US health care-associated infections from 2018 to 2021, of which 28% to 45% were carbapenem not-susceptible.[3,4] The Centers for Disease Control and Prevention (CDC) classifies CRAB as an urgent resistance threat; mortality for CRAB infections exceeds 50%, approaching 75% for pandrug-resistant isolates.[3,5] Carbapenem resistance in CRAB is most often mediated through acquired OXA-23, OXA-24/40, and OXA-58 carbapenemases; however, carbapenemases such as KPC, NDM, VIM, IMP, and OXA-48 are sometimes encountered, with NDM being most common among these.[3,4,6,7]

A. baumannii is intrinsically resistant to many antimicrobial agents, including penicillins, early-generation cephalosporins, aztreonam, and ertapenem, and frequently acquires resistance through plasmid/transposon-mediated mechanisms. Ampicillin-sulbactam has historically been used for *Acinetobacter* infections, as sulbactam is bactericidal against *Acinetobacter* through high-affinity penicillin-binding protein (PBP)1/PBP3 binding. However, resistance to sulbactam, as seen in the isolates described in this case, is common among CRAB, ranging from 45% to over 70% across studies, driven by TEM-1, ADC (AmpC), OXA-type β-lactamases, and PBP3 alterations.[4,8-10] Durlobactam, a diazabicyclooctane β-lactamase inhibitor, protects sulbactam from OXA-carbapenemase hydrolysis and restores activity against most CRAB isolates, though not class B metallo-β-lactamase (MBL) producers (eg, NDM).[10]

The FDA approved sulbactam-durlobactam in May 2023 for *Acinetobacter* hospital-acquired/ventilator-associated pneumonia. Over 95% of global CRAB isolates demonstrate susceptible sulbactam-durlobactam MICs.[4]

In the *Acinetobacter* Treatment Trial Against Colistin (ATTACK) trial, all patients receiving sulbactam-durlobactam also received imipenem-cilastatin, a pairing generally recommended until clinical improvement is observed.[2] In this 16-country, phase 3, noninferiority trial of CRAB pneumonia and bloodstream infection, 28-day mortality was lower with sulbactam-durlobactam plus imipenem than with colistin plus imipenem (19% vs 32%), and nephrotoxicity was significantly lower (13% vs 38%; $P < 0.001$).[8] Outcomes data for sulbactam-durlobactam monotherapy are currently not available.[2,4] Where sulbactam-durlobactam is unavailable, high-dose ampicillin-sulbactam plus a second agent (minocycline, tigecycline, cefiderocol, or polymyxin B) is reasonable; the Infectious Diseases Society of America (IDSA) guidance now discourages the use of colistin-containing regimens for CRAB, favoring polymyxin B.[2,4] Gaps in detection of carbapenemases in CRAB emphasize the need for close laboratory-stewardship collaboration. Commercial rapid molecular and phenotypic carbapenemase assays target KPC, NDM, VIM, IMP, and OXA-48–like genes, but usually not the acquired OXA-23, OXA-24/40, and OXA-58 carbapenemases responsible for most carbapenem resistance in CRAB. In addition, some of these assays are not FDA-cleared for *Acinetobacter* spp. Nevertheless, as durlobactam is inactive against MBLs, it is essential to detect MBL production in *Acinetobacter* spp. in order to prevent committing patients to costly, ineffective therapy. For the isolates described in this case, MBL production was ruled out, sulbactam-durlobactam susceptibility was confirmed, and treatment with sulbactam-durlobactam plus a carbapenem led to clinical resolution.

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Practical Tips

Practical Tips for Laboratory Testing of Sulbactam-Durlobactam for Carbapenem-Resistant *Acinetobacter* spp.

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This article covers practical FAQs for laboratories encountering carbapenem-resistant *Acinetobacter* isolates and testing/reporting of sulbactam-durlobactam.

1. When should the clinical microbiology laboratory perform antimicrobial susceptibility testing (AST) of *Acinetobacter* isolates for sulbactam-durlobactam?

In May 2023, the US Food and Drug Administration (FDA) approved sulbactam-durlobactam for treatment of adults with hospital-acquired or ventilator-associated pneumonia caused by susceptible *A. baumannii-calcoaceticus* complex strains. Laboratories should develop a plan with members of their antimicrobial stewardship program (ASP) that is guided by the recommendations in CLSI M100 Table 1B-2 for a tiered approach to testing and reporting for *Acinetobacter* spp. Sulbactam-durlobactam is listed in tier 3 in the most recent edition of the CLSI M100 standard.[1]

A reflex to sulbactam-durlobactam testing when carbapenem resistance is detected is a reasonable approach for most laboratories. However, primary testing of sulbactam-durlobactam for *A. baumannii-calcoaceticus* complex isolates may be appropriate at institutions serving patients at high risk for multidrug-resistant organisms (MDROs), such as those with long-term health care exposure. It is important to note that AST of *A. baumannii-calcoaceticus* complex should be limited to isolates believed to represent true infection, as recovery alone may reflect colonization and should not warrant AST.[2]

2. The CLSI sulbactam-durlobactam breakpoints are listed for *Acinetobacter* spp.,[1] but the FDA-approved indication is for *Acinetobacter baumannii-calcoaceticus* complex. Can we report sulbactam-durlobactam on any *Acinetobacter* spp., or only on isolates identified as *A. baumannii-calcoaceticus* complex?

The CLSI breakpoints that are recognized by FDA apply to the genus.[1] An isolate therefore does not need to be identified as *A. baumannii-calcoaceticus* complex or be limited to a respiratory source for the breakpoints to be applied, though the approved indication for treatment is limited to these. However, if a commercial device is used to perform AST, the manufacturer's instructions for use will list which organisms are FDA-cleared for that device. A validation study would be necessary to report results for *Acinetobacter* spp. other than those listed in the instructions for use.

3. What AST options are currently available for sulbactam-durlobactam?

The manufacturer of sulbactam-durlobactam provides a listing of current FDA-cleared devices for AST of sulbactam-durlobactam that is reproduced in Table 1.[3] Please note that some devices are designated as being cleared for use with isolates of *A. baumannii-calcoaceticus* complex, while others are cleared for testing isolates of *Acinetobacter* spp. Availability of these tests may vary by region or distributor, and no automated platform is currently available for testing sulbactam-durlobactam.

Cation-adjusted Mueller-Hinton broth is used for reference broth microdilution (BMD) testing, and Mueller-Hinton agar (MHA) is used for standard disk diffusion testing of sulbactam-durlobactam. A multilaboratory study found no bias across multiple brands and media lots of MHA when used for testing sulbactam-durlobactam by disk diffusion.[4]

Table 1. FDA-Cleared Devices for Sulbactam-Durlobactam[3]

Method	Product (Vendor)
Broth microdilution	Sensititre™ 18–24 h MIC system (Thermo Fisher Scientific™)
Disk diffusion	HardyDisk™, 10/10 µg (Hardy Diagnostics)
	Oxoid™ disc, 10/10 µg (Thermo Fisher Scientific™)
Gradient diffusion	ETEST® Sulbactam-Durlobactam (bioMérieux)
	MTS™ (MIC Test Strip) (Liofilchem™)

Abbreviations: FDA, US Food and Drug Administration; MIC, minimal inhibitory concentration.

Practical Tips for Laboratory Testing of Sulbactam-Durlobactam for Carbapenem-Resistant *Acinetobacter* spp. (*Continued*)

4. Where can clinical laboratories obtain isolates to help with validation and verification of sulbactam-durlobactam AST?

A dedicated panel of *A. baumannii-calcoaceticus* complex isolates for verification of sulbactam-durlobactam is not currently available from the CDC and FDA Antimicrobial Resistance (AR) Isolate Bank. However, the *Acinetobacter baumannii* (ACI) panel available from this resource contains 51 isolates spanning diverse resistance mechanisms, including OXA-23/-24/-66/-72/-82, NDM-, and VIM-producers.[5] Isolates from this panel could be used for verification, but results for sulbactam-durlobactam are not currently provided for any of these isolates. Isolates for verification of sulbactam-durlobactam AST with reference BMD minimal inhibitory concentration (MIC) results are available for purchase from commercial sources, such as Laboratory Specialists, Inc.[6]

Selection of isolates for verification is at the discretion of the laboratory director, and CLSI M52 provides guidance on verifying commercial AST systems.[7] Generally, a minimum of 10 to 20 isolates would be selected, and for sulbactam-durlobactam it would be helpful to include some isolates with penicillin-binding protein (PBP)3 mutations and some harboring metallo-β-lactamases (MBLs).[7,8]

Currently, no isolate panel is available for *Acinetobacter* spp. other than *A. baumannii-calcoaceticus* complex, posing a challenge for laboratories wishing to offer sulbactam-durlobactam AST for these organisms.

5. What QC organism should be used when testing sulbactam-durlobactam, and what are acceptable QC ranges for this strain?

CLSI M100 Tables 4A-2 and 5A-2 recommend *A. baumannii* NCTC 13304 for routine QC of sulbactam-durlobactam AST.[1] This strain expresses β-lactamase (OXA-27) that confers sulbactam resistance, which is then reversed by the addition of durlobactam, properly challenging both components of the combination agent. In order to ensure this QC strain has not lost its ability to produce the OXA-27 β-lactamase, its "integrity" should be confirmed by testing with cefepime or meropenem periodically (eg, when the QC strain is removed from the freezer) as described in CLSI M100. The CLSI acceptable QC ranges are 0.5/4 to 2/4 µg/mL for MIC and 24 to 30 mm for disk diffusion using a 10/10 µg disk.[1]

6. What are the breakpoints for sulbactam-durlobactam?

CLSI breakpoints for sulbactam-durlobactam as published in CLSI M100 36th edition are listed in Table 2.[1]

Table 2. CLSI and FDA Breakpoints for Sulbactam-Durlobactam and *Acinetobacter* spp.

Antimicrobial Agent	Disk Content	Interpretive Categories and Zone Diameter Breakpoints, Nearest Whole mm			Interpretive Categories and MIC Breakpoints, µg/mL		
		S	I	R	S	I	R
Sulbactam-durlobactam	10/10 µg	≥ 17	14-16	≤ 13	≤ 4/4	8/4	≥ 16/4

Abbreviations: I, intermediate; MIC, minimal inhibitory concentration; R, resistant; S, susceptible.

7. How frequently are resistant isolates encountered?

Over 95% of carbapenem-resistant *Acinetobacter baumannii* (CRAB) isolates in the United States are susceptible to sulbactam-durlobactam when applying the current breakpoints.[8,9] Resistance to sulbactam-durlobactam is primarily due to PBP3 mutation or production of a metallo-β-lactamase (MBL). CLSI M100 Appendix A designates sulbactam-durlobactam resistance as category I, meaning that resistance has not been reported or (in the case of sulbactam-durlobactam) has only rarely been reported. If a sulbactam-durlobactam-resistant isolate is encountered, Appendix A recommends confirming the organism identification and susceptibility, reporting the result to infection prevention, contacting your local public health laboratory, and saving the isolate.[1] Notably, because MBL-producing *Acinetobacter* isolates would be expected to show resistance to sulbactam-durlobactam, it would be helpful to rule out MBL production by performing a genotypic test, as this information will impact treatment decisions and the need for sulbactam-durlobactam testing.

Practical Tips for Laboratory Testing of Sulbactam-Durlobactam for Carbapenem-Resistant *Acinetobacter* spp. (*Continued*)

8. What should clinical laboratories know about the fact that sulbactam-durlobactam is usually prescribed with a carbapenem?
In the *Acinetobacter* Treatment Trial Against Colistin (ATTACK) trial, all patients receiving sulbactam-durlobactam also received imipenem-cilastatin.[10] Sulbactam-durlobactam is generally administered together with a carbapenem (imipenem or meropenem), at least until there is clinical improvement.[2,10] Laboratories should work with their antimicrobial stewardship program (ASP) team to determine if a comment should be added to laboratory reports to emphasize the need for an additional agent.[2] An example of such a comment would be: "Sulbactam-durlobactam is generally given with a carbapenem (imipenem-cilastatin or meropenem) for carbapenem-resistant *Acinetobacter baumannii* complex."

9. What is important to know about carbapenemase testing for *A. baumannii-calcoaceticus* complex?
The modified carbapenem inactivation method (mCIM) and Carba NP test method as described in CLSI M100 should not be performed on *Acinetobacter* spp. as they perform poorly for isolates in this genus.[1]

At least one commercial system is FDA-cleared for testing *A. baumannii* isolates for KPC, NDM, VIM, IMP, and OXA-48,[11] but not OXA-23 and OXA-24/40, the 2 most common carbapenemases identified in CRAB in the United States.[12,13] Most of the blood culture molecular platforms detect *Acinetobacter* spp., KPC, NDM, VIM, IMP, and OXA-48, but generally not OXA-23 and OXA-24/40. A lateral flow assay, Hardy NG-TEST® Acineto-5® (similar to the Hardy NG-TEST® CARBA-5®), was granted FDA Breakthrough Device designation in February 2026 and is available for research use only to detect NDM, VIM, OXA-23-like, OXA-24-like/OXA-143-like, and OXA-58-like targets in isolates of *Acinetobacter* spp.[14] These methods or more sophisticated methods, such as whole genome sequencing, can be used to identify isolates with carbapenemases, including rarer genes, and sequencing also allows for detection of PBP3 mutations.

As part of CDC's Antibiotic Resistance Laboratory Network program, state and some jurisdictional public health laboratories offer carbapenemase testing of *A. baumannii-calcoaceticus* complex isolates, primarily for surveillance purposes.[15] Local public health laboratories can advise clinical laboratories on how to proceed if carbapenemase testing of *A. baumannii-calcoaceticus* complex is desired.

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Practical Tips for Laboratory Testing of Sulbactam-Durlobactam for Carbapenem-Resistant *Acinetobacter* spp. (*Continued*)

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Hot Topic

Azithromycin Susceptibility Testing for *Shigella:* Clinical, Laboratory, and Public Health Implications

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Increasing Resistance to Azithromycin

Resistance to azithromycin and other antimicrobial agents used to treat shigellosis has increased in the United States. Before clinical breakpoints were established, epidemiological cutoff values were used to identify wild-type and non-wild-type isolates. During 2011 to 2018, the percentage of isolates with azithromycin minimal inhibitory concentrations (MICs) above the CLSI epidemiological cutoff value increased from 12% to 53% among *Shigella flexneri* isolates and from 0.9% to 25% among *S. sonnei* isolates.[1] An MIC above the epidemiological cutoff value indicates a non-wild-type phenotype rather than clinical resistance. In 2021, CLSI established azithromycin MIC and disk diffusion clinical breakpoints for *Shigella* spp., allowing isolates to be classified as susceptible, intermediate, or resistant.[1] The current CLSI MIC breakpoints are ≤ 8 µg/mL for susceptible, 16 µg/mL for intermediate, and ≥ 32 µg/mL for resistant.[2] Centers for Disease Control and Prevention (CDC) subsequently adopted these breakpoints for *Shigella* surveillance through the National Antimicrobial Resistance Monitoring System.[3]

When antimicrobial treatment is indicated, azithromycin is among the first-choice agents for shigellosis treatment.[4] CDC defines extensively drug-resistant (XDR) *Shigella* as resistant to all 5 of the following agents: ampicillin, azithromycin, ceftriaxone, ciprofloxacin, and trimethoprim-sulfamethoxazole. In a national analysis of 16,788 *Shigella* isolates, the percentage that were XDR increased from 0% during 2011 to 2015 to 8.5% in 2023.[5] Treatment options for XDR shigellosis are limited. Potential options include carbapenems, pivmecillinam, and fosfomycin. Among these agents, meropenem has CLSI clinical breakpoints for *Shigella* spp.; pivmecillinam and fosfomycin do not have *Shigella*-specific clinical breakpoints.[2,5] Because azithromycin is 1 of the 5 agents used to define XDR *Shigella*, an azithromycin susceptibility result is necessary for complete phenotypic XDR classification. Azithromycin susceptibility results also inform treatment decisions and antimicrobial resistance surveillance.

California Data on Azithromycin AST and XDR Surveillance Completeness

The California Department of Public Health (CDPH) used routinely reported laboratory data to examine the availability of azithromycin AST results and their effect on XDR surveillance. In 2026, CDPH reviewed AST results reported through electronic laboratory reporting (ELR) for confirmed shigellosis cases, excluding Los Angeles and San Diego jurisdictions. Of 612 confirmed cases from January through August 15, 2026, 352 (58%) had at least 1 AST result reported. Results for all 5 XDR-defining agents were available for 141 of those 352 cases (40%), equivalent to 141 of 612 (23%) of all confirmed cases.[6] Of the 352 cases with any AST results reported, 87 (25%) were missing a result for azithromycin but had results for the other 4 XDR-defining agents. If azithromycin results had also been reported for those 87 cases, the proportion with complete 5-agent results would have increased from 40% to approximately 65%.[6]

CDPH also compared reporting for each of the 5 XDR-defining agents. The analysis included 405 individual AST records, including more than 1 AST record per case.[6] Azithromycin results were reported for 208 records (51%), compared with ceftriaxone in 269 (66%), ciprofloxacin in 371 (92%), ampicillin in 395 (98%), and trimethoprim-sulfamethoxazole in 400 (99%). A missing azithromycin result could be due to lack of azithromycin testing by the laboratory or lack of reporting of the azithromycin result through ELR. Of 59 isolates from 2021 to 2026 that were resistant to the other 4 XDR-defining agents, lacked an azithromycin result, and had whole-genome sequencing (WGS) results available, 53 (90%) had predicted azithromycin resistance. CDC NARMS data show less than 1% discordance between WGS-predicted and phenotypic azithromycin results in approximately 3,000 isolates.[7] Because WGS results were available for only a subset of isolates, the 53/59 finding should not be interpreted as an estimate of statewide prevalence.[6]

Azithromycin Susceptibility Testing for *Shigella:* Clinical, Laboratory, and Public Health Implications (*Continued*)

A 2025 Los Angeles County survey found that azithromycin testing was not routine for most clinical laboratories. Of 46 responding laboratories, 37 (80.4%) performed AST for *Shigella*. Of the laboratories that answered the question about azithromycin testing, 18.2% tested routinely, 51.5% tested upon physician request, and 30.3% used another approach. Los Angeles County was not included in the CDPH ELR analysis.[8]

Laboratory Testing Considerations

These findings have real implications for laboratory practice. FDA recognizes CLSI azithromycin breakpoints for *Shigella*.[2,9] However, azithromycin is not included on commonly used automated commercial AST panels for *Salmonella* or *Shigella* spp. CLSI has established azithromycin disk diffusion zone diameter clinical breakpoints for *Shigella* spp., but some isolates show hazy zone edges that can make the zone diameter difficult to determine; in these cases, CLSI M100 recommends testing by a MIC method.[2] Gradient diffusion strips are commercially available, but the gradient diffusion products reviewed for this article are not FDA-cleared for *Shigella*.[10,11] Laboratories using a non-FDA-cleared method for *Shigella* must validate the method before reporting patient results. Characterized isolates from the CDC and FDA Antimicrobial Resistance Isolate Bank may support method verification or validation.[12]

For laboratories that do not perform azithromycin AST in-house, referral testing is another option. Laboratories can retain timely diagnostic testing by knowing in advance where azithromycin testing is available, how to request it, and the expected turnaround time. Public health laboratories may perform azithromycin AST when feasible. WGS can predict azithromycin susceptibility for surveillance, but it does not replace a phenotypic AST result. Azithromycin susceptibility results are important for patient treatment, XDR determination, and antimicrobial resistance surveillance.

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Practical Tips

CLSI M100 Recommendations for Antimicrobial Agents to Test and Report Against *Shigella* spp.

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In the Hot Topic article in this edition of the *CLSI AST News Update*, public health officials have shared concerns surrounding multidrug resistance in *Shigella* spp., with an emphasis on azithromycin resistance. It is important for clinical laboratorians to understand the implications of testing and reporting appropriate antimicrobial agents to aid both public health and clinicians in addressing this resistance threat.

CLSI M100 includes specific guidelines for selection of antimicrobial agents to test and report against a variety of organisms and organism groups. Before 2023, guidance for *Shigella* spp. and *Salmonella* spp. was included in the table specific for Enterobacterales. Now, there is a separate table (Table 1A-2) for *Shigella* spp. and *Salmonella* spp. that clearly reflects what is recommended. There is also a corresponding table (Table 2A-2) that contains breakpoints specific for these genera.[1]

The footnotes for CLSI M100 Table 1A-2, reproduced below in Table 1, indicate that antimicrobial susceptibility testing (AST) should be performed on all *Shigella* spp. isolates from fecal sources as well as extraintestinal sources. There are several agents in tier 1, suggesting that they are appropriate for routine testing and reporting.[1] These antimicrobial agents have strong clinical evidence for use and are included in clinical practice guidelines for the treatment of shigellosis and extra-intestinal salmonellosis.[2-4] Azithromycin is in tier 2, which suggests it be tested routinely and reported following cascade reporting on isolates that are resistant to 1 or more antimicrobial agents listed in tier 1. There are no consensus guidelines on when to cascade report azithromycin, but approaches include:

- Reporting azithromycin if all other oral treatment options (ampicillin, ciprofloxacin or levofloxacin and trimethoprim-sulfamethoxazole) are resistant

OR

- Routinely reporting azithromycin on all isolates, particularly in areas of known resistance

OR

- Reporting routinely for pediatric patients, but reporting for adult patients only on request

Of note, laboratories should always report a resistant result, even if not meeting cascade criteria.

As mentioned in the "Hot Topic" article in this *CLSI AST News Update*, there are concerns with testing azithromycin due to the lack of any US Food and Drug Administration (FDA)-cleared methods for this agent for *Shigella* spp. and some challenges with reading results when using disk diffusion[1] and gradient diffusion methods.[5] Nevertheless, it is essential for all clinical laboratories to identify a strategy for testing and reporting azithromycin on isolates resistant to the agents in tier 1.

The agents listed in tier 4, which include carbapenems and tetracycline, should only be tested and reported following physician requests. It would be inappropriate, for example, to report meropenem on an isolate that is susceptible to any agent in tier 1 or azithromycin. It would also be inappropriate to report a carbapenem or tetracycline if azithromycin has not been tested and reported. Even if an isolate is resistant to all agents in tiers 1 and 2, reporting of tier 4 agents should not occur without interacting with a physician as there are concerns regarding these agents that must be considered before a physician would prescribe one of them for treatment of shigellosis. In particular, little to no clinical evidence exists for the treatment of shigellosis with carbapenems, and, as such, these agents should be used only as a last resort.[6]

There is 1 antimicrobial agent, chloramphenicol, for which breakpoints are listed in CLSI M100 Table 2A-2 but not in Table 1A-2.[1] Although Table 1A-2 recommendations may be followed in any country, they were initially developed based on antimicrobial agents used within the United States. Chloramphenicol is rarely used in the United States.

CLSI M100 Recommendations for Antimicrobial Agents to Test and Report Against *Shigella* spp. (*Continued*)

Beyond the considerations with tier 4 agents, some physicians may ask the laboratory, "What else can be tested?" when an isolate of *Shigella* spp. is resistant to all antimicrobial agents for which breakpoints are available. In these cases, it is critical to suggest the physician interact with their antimicrobial stewardship team for further guidance.

Considering the escalating clinical and public health concerns surrounding multidrug resistance in *Shigella* spp., CLSI is working with the Centers for Disease Control and Prevention (CDC) to determine if modifications to testing and reporting recommendations are needed for CLSI M100.[1]

Table 1. Antimicrobial Agents That Should Be Considered for Testing and Reporting by Microbiology Laboratories[2]

Table 1A-2. *Salmonella* and *Shigella* spp.[a,b]

Tier 1: Antimicrobial agents that are appropriate for routine, primary testing and reporting	Tier 2: Antimicrobial agents that are appropriate for routine, primary testing but may be reported following cascade reporting rules established at each institution	Tier 3: Antimicrobial agents that are appropriate for routine, primary testing in institutions that serve patients at high risk for MDROs but should only be reported following cascade reporting rules established at each institution	Tier 4: Antimicrobial agents that may warrant testing and reporting by clinician request if antimicrobial agents in other tiers are not optimal because of various factors
Ampicillin			
Ciprofloxacin Levofloxacin			
Trimethoprim-sulfamethoxazole			
Cefotaxime or ceftriaxone			Ertapenem[c] Imipenem[c] Meropenem[c]
	Azithromycin[d]		
			Tetracycline

Abbreviations: AST, antimicrobial susceptibility testing; MDRO, multidrug-resistant organism.

Footnotes

a. Table 2A-2 should be used for interpreting AST results for *Salmonella* and *Shigella* spp.

b. **WARNING:** For *Salmonella* and *Shigella* spp., aminoglycosides, first- and second-generation cephalosporins, and cephamycins may appear active *in vitro* but are not effective clinically and should not be reported as susceptible. Routine susceptibility testing is not indicated for nontyphoidal *Salmonella* spp. isolated from intestinal sources. However, susceptibility testing is indicated for all *Shigella* isolates. When fecal isolates of *Salmonella* and *Shigella* spp. are tested, only ampicillin, a fluoroquinolone, and trimethoprim-sulfamethoxazole should be reported routinely. In addition, for extraintestinal isolates of *Salmonella* spp., a third-generation cephalosporin should be tested and reported. Azithromycin may be tested and reported per institutional guidelines.

c. Ertapenem, imipenem, and/or meropenem might be considered for testing and/or reporting for isolates resistant to all agents in tiers 1 and 2, although there are limited clinical data suggesting their effectiveness for treating salmonellosis or shigellosis.[1]

d. Report only on *Salmonella enterica* ser. Typhi and *Shigella* spp.

Reference for Table 1A-2

1 Centers for Disease Control and Prevention. Typhoid fever and paratyphoid fever: data summary: emerging strain of *Salmonella* Typhi (REP JPP01). Accessed 10 October 2025. https://www.cdc.gov/typhoid-fever/php/rep-strain/index.html#:~:text=REPJPP01%20is%20an%20emerging%20strain,not%20leave%20the%20United%20States

CLSI M100 Recommendations for Antimicrobial Agents to Test and Report Against *Shigella* spp. (*Continued*)

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Practical Tips

Newer Antimicrobial Agents for Antimicrobial-Resistant Gram-Negative Bacteria and Placement in CLSI M100

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The Infectious Diseases Society of America (IDSA) 2026 Guidance on the Treatment of Antimicrobial Resistant Gram-Negative Infections[1] is an excellent resource for clinicians when making treatment decisions and for laboratorians when selecting agents for *in vitro* testing. CLSI considers guidelines like these along with input from clinical microbiologists, infectious disease physicians, and pharmacists from health care, government, and industry when updating CLSI M100[2] and other antimicrobial susceptibility testing (AST) documents.

In the current IDSA 2026 guidance, which contains specific treatment recommendations for various gram-negative species and susceptibility profiles, there are some agents (Table 1) for which there are no breakpoints currently listed in CLSI M100. However, breakpoints for these agents are listed on the US Food and Drug Administration (FDA) Susceptibility Test Interpretive Criteria (STIC) website.[3] In addition, there are 2 antimicrobial agents that were FDA approved for antimicrobial-resistant gram-negative infections that are not addressed in the IDSA guidance and for which there are no CLSI breakpoints. Cefepime-zidebactam was FDA approved in May 2026 and tebipenem in June 2026. Timing of the FDA approval likely contributed to their absence in the IDSA guidance.

Table 1. Antimicrobial Agents Without Breakpoints in CLSI M100 36th Edition[2a]

Antimicrobial Agent	FDA Approval Date
Cefepime-enmetazobactam	2024
Eravacycline	2018
Fosfomycin (IV)	2025
Gepotidacin[b]	2025
Omadacycline	2018
Sulopenem	2024
Tigecycline	2005

Abbreviations: FDA, US Food and Drug Administration; IDSA, Infectious Diseases Society of America; IV, intravenous.
[a] Agents referenced in IDSA guidance for which there are no breakpoints in CLSI M100.
[b] Breakpoints will be published in CLSI M100 37th edition (2027).

What are the timelines for FDA and CLSI breakpoint approval?

According to FDA regulations, part of the FDA approval process for a new antimicrobial agent requires the pharmaceutical company to submit *in vitro* testing data and breakpoints used to interpret these data. Once the agent and breakpoints are FDA approved, the breakpoints are published on the STIC website.[3] Generally, CLSI breakpoints are only reviewed after FDA breakpoints are published.

The processes and timelines used by CLSI to develop AST recommendations, including processes for establishing QC ranges and breakpoints, are detailed in CLSI M23.[4] It is important to keep in mind that, in contrast to FDA, CLSI is a standards development organization and does not have regulatory authority to require that pharmaceutical companies seek CLSI publication of breakpoints.

When does a new antimicrobial agent get published in CLSI M100; why do QC RANGES come first?

Early in the antimicrobial agent development process, the pharmaceutical company may voluntarily submit data to CLSI to establish minimal inhibitory concentration (MIC) QC ranges. For pharmaceutical manufacturers to proceed with clinical trials involving treatment of patients infected with specific bacteria, accurate AST methods must be used, and reliable QC testing is essential. CLSI is recognized by the FDA, pharmaceutical companies, diagnostic manufacturers, and others as having a robust process for setting both MIC and disk diffusion QC ranges. The CLSI AST Subcommittee (AST SC) reviews QC data submitted by pharmaceutical companies, and QC ranges are often published in CLSI M100 several years before the antimicrobial agent is FDA approved. Concurrently with publishing QC ranges in CLSI M100, the agent is added to the CLSI M100 Glossary and tables (Tables 6), which describe how to prepare antimicrobial stock solutions.[2]

Newer Antimicrobial Agents for Antimicrobial-Resistant Gram-Negative Bacteria and Placement in CLSI M100 (*Continued*)

MIC QC ranges are often published before disk diffusion QC ranges. CLSI offers, in addition to CLSI M23,[4] other guidance documents that are typically used by pharmaceutical companies for establishing disk content when defining a disk diffusion method for a new antimicrobial agent.[5]

When do BREAKPOINTS for a new antimicrobial agent get published in CLSI M100?

After FDA approval of a new antimicrobial agent and corresponding FDA breakpoints, pharmaceutical manufacturers may **voluntarily** submit data to the CLSI AST SC for CLSI breakpoint approval. If CLSI approves the same breakpoints as FDA, they are published in the next annual edition of CLSI M100. Subsequently, FDA removes breakpoints from the STIC website and adds a notation to refer to CLSI M100 to access the breakpoints for the agent. If CLSI does not agree with the FDA breakpoints and publishes different breakpoints in CLSI M100, the FDA STIC website retains the FDA breakpoints. When this happens, there are 2 sets of breakpoints. Discussions between CLSI and FDA often continue in an attempt to achieve harmonization of breakpoints. As part of this process, CLSI prepares a rationale document that explains the reasons for their breakpoint decision and why the CLSI breakpoints differ from FDA breakpoints.[6] Diagnostic manufacturers must use FDA breakpoints in order to receive device clearance. Clinical laboratories may use FDA or CLSI breakpoints.

Although most pharmaceutical companies find it beneficial to get breakpoints published in CLSI M100, on some occasions they do not. And sometimes there is a considerable delay in submitting data and getting breakpoints published in CLSI M100. This may be the case for some of the antimicrobial agents listed in Table 1. It is acceptable for clinical laboratories to use the STIC breakpoints.

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Next Up!

Celebrating 10 Years of the *CLSI AST News Update*: Highlights, Resources, and Search Tips

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Do you enjoy reading the *CLSI AST News Update?* Do you ever find yourself wanting more information on a particular antimicrobial susceptibility testing (AST) topic but don't know where to look?

Over the last 10 years, we have published over 80 articles covering a variety of AST topics, including mechanisms of resistance, susceptibility testing, novel antimicrobial agents, quality control, results interpretation, clinical breakpoints, antifungal susceptibility testing, public health, and more. In honor of the *CLSI AST News Update's* 10th anniversary, we assembled a curated index of the newsletter articles. Highlighted below are 5 of our most impactful articles, listed in order of publication year.

- Cefazolin, Urine, and *Escherichia coli, Klebsiella pneumoniae,* and *Proteus mirabilis:* Entertaining Solutions for Antimicrobial Susceptibility Testing and Reporting, Volume 3, Issue 2, Spring 2018
- The Importance of Being Specific: *Staphylococcus* Species-Specific Breakpoints for Methicillin (Oxacillin) Resistance, Volume 5, Issue 1, January 2020
- Implementation of the CLSI Method for Direct Disk Diffusion Testing From Positive Blood Cultures, Volume 7, Issue 1, June 2022
- Cefepime Reporting Strategies for Carbapenemase-producing Isolates of Enterobacterales, Volume 10, Issue 1, April 2025
- A Clinical Case of *Burkholderia cepacia* Complex and Changes to CLSI Antimicrobial Susceptibility Testing Recommendations, Volume 11, November 2025

For a complete list of articles, which can be sorted by title, volume/publication date, or topic, **click here** and then scroll down to the AST News Update Index.

