

and AmpC), and aztreonam (ATM) is a monobactam stable to hydrolysis by metallo- β -lactamases (MBL).

Methods. A total of 10,451 *Enterobacteriaceae* (ENT) consecutively collected from 84 United States (US) medical centers and 250 carbapenem-resistant ENT (CRE) collected from 38 centers in 25 other countries (ex-US) were tested for susceptibility (S) by reference broth microdilution methods in a central monitoring laboratory (JMI Laboratories). CRE strains were screened for the presence of carbapenemase (CBP)-encoding genes using whole genome sequencing analysis.

Results. All ENT isolates from US (MIC_{50/90} \leq 0.03/0.12 μ g/mL), except for 1 *Escherichia coli* strain with an ATM-AVI MIC of 8 μ g/mL, and all ex-US CRE isolates (MIC_{50/90} 0.25/0.5 μ g/mL) were inhibited at ATM-AVI MIC of \leq 4 μ g/mL (CLSI S breakpoint for ATM). Among US isolates, ATM-AVI was also very active against CRE (n = 120; MIC_{50/90} 0.12/0.5 μ g/mL; highest MIC, 4 μ g/mL), multidrug-resistant (MDR; n = 876; MIC_{50/90} 0.06/0.25 μ g/mL), extensively drug-resistant (XDR; n = 111; MIC_{50/90} 0.12/0.5 μ g/mL), pan-drug resistant (n = 2; MICs \leq 0.03 and 0.12 μ g/mL), and ceftazidime-non-S *Enterobacter cloacae* (MIC_{50/90} 0.25/1 μ g/mL) isolates. Meropenem was very active against US ENT overall (MIC_{50/90} 0.03/0.06 μ g/mL; 98.8% per CLSI), but showed limited activity against MDR (86.2%) and XDR (30.6%) isolates. Amikacin and colistin were active against 74.2% and 81.7% of US CRE, 93.4% and 58.3% US MDR, 65.8% and 57.7% of US XDR, and 58.0% and 79.2% of ex-US CRE isolates, respectively. A total of 106 CBPs were detected in 106 US CRE isolates, including 102 KPC-like, 2 SME-4, 1 NDM-1, and 1 IMP-27. Also, 248 CBPs were identified on 241 ex-US CRE isolates, including 124 KPC-like, 64 OXA-like, 50 NDM-like, 7 VIM-1, 2 IMP-4, and 1 SME-4. All CRE isolates, including all CBP-producing ENT (US and ex-US), were inhibited at ATM-AVI MIC of \leq 4 μ g/mL.

Conclusion. ATM-AVI demonstrated potent *in vitro* activity against a large collection of contemporary (2016) ENT isolated from patients in US hospitals and CRE isolates collected worldwide, including NDM, KPC, OXA, VIM, and SME producers.

Disclosures. H. S. Sader, Allergan: Research Contractor, Research grant; R. E. Mendes, Allergan: Research Contractor, Research grant; D. Shortridge, Allergan: Research Contractor, Research grant; R. K. Flamm, Allergan: Research Contractor, Research grant; M. Castanheira, Allergan: Research Contractor, Research grant

1234. Activity of Meropenem-Vaborbactam Against *Enterobacteriaceae* Isolates Carrying *bla*_{KPC} Collected Worldwide

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Background. Meropenem-vaborbactam (MER-VAB) is a carbapenem- β -lactamase inhibitor combination with enhanced activity against KPC-producing *Enterobacteriaceae* recently evaluated in a phase 3 clinical trials for cUTIs and infections due to CRE. We analyzed the activity of MER-VAB against 517 isolates carrying *bla*_{KPC} collected worldwide during 2014–16.

Methods. *Enterobacteriaceae* isolates (n = 34,069) from 34 countries were susceptibility (S) tested by reference broth microdilution method for MER-VAB (at fixed 8 μ g/mL) and comparators. Carbapenem-resistant *Enterobacteriaceae* (CRE; CLSI criteria) were submitted to PCR/Sanger sequencing or next-generation sequencing for *bla*_{KPC} screening.

Results. A total of 517 (1.5%) carried *bla*_{KPC} and 6 variants were observed: 293 *bla*_{KPC-3}, 218 *bla*_{KPC-2}, 2 *bla*_{KPC-4}, 2 *bla*_{KPC-17} and 1 each of *bla*_{KPC-2}-like and *bla*_{KPC-12}-like. Isolates were mainly *K. pneumoniae* (437), but also 32 *E. cloacae*, 13 *K. oxytoca*, 12 *E. coli*, 12 *S. marcescens*, and 4 other species. Isolates carrying *bla*_{KPC} were detected in 17 countries. The occurrence ranged from 0.1% to 11.3%, being higher in Brazil, Italy (9.3%), Poland (5.6%), and Argentina (5.2%). MER-VAB inhibited 514/517 (99.4%) isolates carrying *bla*_{KPC} at \leq 8 μ g/mL and this compound was the most active agent tested against these isolates (MIC_{50/90} 0.12/1 μ g/mL). Three isolates displaying elevated MER-VAB MIC values ($>$ 8 μ g/mL) co-harbored *bla*_{NDM-1} or *bla*_{OXA-48}-like in addition to *bla*_{KPC} or had a missense mutation on *OmpK35*. MER alone (MIC_{50/90} 32/ $>$ 32 μ g/mL), imipenem (MIC_{50/90} $>$ 8/ $>$ 8 μ g/mL), and doripenem (MIC_{50/90} $>$ 4/ $>$ 4 μ g/mL) were not active against isolates harboring *bla*_{KPC}. Amikacin (MIC_{50/90} 16/ $>$ 32 μ g/mL) and gentamicin (MIC_{50/90} 2/ $>$ 8) μ g/mL inhibited only 54.9% and 57.3% of the isolates (CLSI breakpoint). Colistin (MIC_{50/90} \leq 0.5/ $>$ 8 μ g/mL; 70.4% S/EUCAST breakpoint) and tigecycline (MIC_{50/90} 0.5/1 μ g/mL; 99.4% S/US FDA criteria) were the most active comparators.

Conclusion. The occurrence of *bla*_{KPC} is still low overall, but can be as high as 5–10% in a few countries and occur in species other than *Klebsiella*. KPC-producers are highly resistant to available antimicrobial agents and MER-VAB will be a useful alternative to treat infections caused by these organisms.

Disclosures. M. Castanheira, Rempex, a wholly owned subsidiary of The Medicines Company: Research Contractor, Research grant; R. E. Mendes, Rempex, a wholly owned subsidiary of The Medicines Company: Research Contractor, Research grant; L. R. Duncan, Rempex, a wholly owned subsidiary of The Medicines Company: Research Contractor, Research grant; L. N. Woosley, Rempex, a wholly owned subsidiary of The Medicines Company: Research Contractor, Research grant; R. K. Flamm, Rempex, a wholly owned subsidiary of The Medicines Company: Research Contractor, Research grant

1235. Activity of Plazomicin against *Enterobacteriaceae* Isolates Collected in the United States Including Isolates Carrying Aminoglycoside-Modifying Enzymes Detected by Whole Genome Sequencing

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Session: 147. Expanded Spectrum – New Antimicrobial Susceptibility Testing
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Background. Plazomicin (PLZ) is a next-generation aminoglycoside (AMG) stable against aminoglycoside-modifying enzymes (AME) that completed Phase 3 studies for complicated urinary tract infections and serious infections due to carbapenem-resistant *Enterobacteriaceae* (ENT). We evaluated the activity of PLZ and AMGs against ENT collected in US hospitals during 2016.

Methods. A total of 2,097 ENT were susceptibility (S) tested by CLSI reference broth microdilution methods. *E. coli*, *Klebsiella* spp., *Enterobacter* spp., and *P. mirabilis* isolates displaying non-S MICs (CLSI criteria) for gentamicin (GEN), amikacin (AMK), and/or tobramycin (TOB) were submitted to WGS, *de novo* assembly and screening for AME genes.

Results. Against ENT, PLZ was more active than all 3 clinically available AMGs (Table). PLZ and AMK activities were stable regardless of the infection type; however, differences were observed for GEN and TOB. Bloodstream isolates displayed higher GEN MICs when compared with the other infection sites. TOB activity varied 4-fold, being higher for bloodstream and pneumonia infections and lower for skin/soft tissue and other/unknown specimens. Against 198 isolates carrying 1 or more AME-encoding genes detected among 208 AMG-non-S isolates, the activity of PLZ was 8- to 16-fold greater when compared with the activity of AMK and at least 16-fold higher than the activity of GEN or TOB.

Conclusion. PLZ was active against ENT isolates from US hospitals regardless of infection type. PLZ displayed activity against isolates carrying AME genes that represent 12.0% of selected species. AME-carrying isolates were considerably more resistant to AMK, GEN, and TOB, highlighting the potential value of PLZ to treat infections caused by these organisms.

This project has been funded under BARDA Contract No. HHSO100210100046C.

Organism group/infection type (no. tested)	MIC _{50/90} (μ g/mL)			
	Plazomicin	Amikacin	Gentamicin	Tobramycin
<i>Enterobacteriaceae</i> (2,097)	0.5/1	2/4	0.5/4	0.5/4
Urinary tract infection (587)	0.5/1	2/4	0.5/2	0.5/4
Bloodstream infection (572)	0.5/1	2/4	0.5/ $>$ 8	0.5/8
Pneumonia in hospitalized patients (451)	0.25/1	2/4	0.5/4	0.5/8
Skin/soft tissue infection (298)	0.5/2	2/4	0.5/1	0.5/2
Intra-abdominal infection (152)	0.5/1	2/4	0.5/4	0.5/4
Other sites (37)	0.25/1	1/2	0.5/1	0.5/2
Isolates carrying AME genes (198)	0.5/1	4/16	$>$ 8/ $>$ 8	$>$ 8/ $>$ 8

Disclosures. M. Castanheira, Achaogen: Research Contractor, Research grant; L. M. Deshpande, Achaogen: Research Contractor, Research grant; C. M. Hubler, Achaogen: Research Contractor, Research grant; R. E. Mendes, Achaogen: Research Contractor, Research grant; A. W. Serio, Achaogen: Employee, Salary; K. M. Krause, Achaogen: Employee, Salary; R. K. Flamm, Achaogen: Research Contractor, Research grant

1236. In Vivo Efficacy of Tigecycline-based Therapy Against *Vibrio vulnificus* Sepsis: Comparison with pre-Existing Regimens

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Session: 147. Expanded Spectrum – New Antimicrobial Susceptibility Testing
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Background. The mortality of *Vibrio vulnificus* sepsis is still high, despite the application of various antibiotic regimens. *In-vivo* efficacy of tigecycline against *V. vulnificus* has not been examined.

Methods. Time-kill assay was performed to evaluate the presence of *in-vitro* antibiotic synergism. The cytotoxicity of *V. vulnificus* was measured by using the lactate dehydrogenase assay, and rtxA1 toxin gene transcription was measured by β -galactosidase assay. Subcutaneous injection of *V. vulnificus* was performed with 1×10^8 CFU on iron-overloaded female BALB/c mouse, then intraperitoneal antibiotic therapy was initiated 2 hours after bacterial inoculation.

Results. *In vitro* time-kill assay reveals synergism between tigecycline and ciprofloxacin. Inhibitory effects of tigecycline on rtx A1 transcription (66%) and cytotoxicity (59%) were comparable to those of ciprofloxacin (64% and 53%), but superior to those of minocycline (76% and 69%) or cefotaxime (86% and 83%; $P < 0.05$, each). Survival of tigecycline-treated mice were significantly higher than those of mice treated by current regimens ($P < 0.05$, each; Table). At *Vibrio vulnificus* sepsis mice inoculating 1×10^9 CFU, survival rate for tigecycline-plus-ciprofloxacin was significantly higher than that of tigecycline (0%; 0/19) or tigecycline-plus-cefotaxime (0%; 0/19) ($P < 0.05$, each; Table).

	10 ⁸ CFU, 96hr survival (%)	10 ⁹ CFU, 96hr survival (%)
control	0/24 (0%)	0/13 (0%)
ciprofloxacin	14/25 (56%)	N.A
cefotaxime-minocycline	16/25 (64%)	N.A
tigecycline	22/25 (88%)	0/19 (0%)
tigecycline-cefotaxime	21/25 (84%)	0/19 (0%)
tigecycline-ciprofloxacin	21/25 (84%)	7/19 (37%)

Conclusion. Tigecycline-plus-ciprofloxacin showed superior *in-vivo* efficacy to pre-existing regimens.

Disclosures. All authors: No reported disclosures.

1237. Antimicrobial Activity of Ceftazidime-Avibactam and Comparator Agents Tested against Enterobacteriaceae and Pseudomonas aeruginosa from United States (US) Medical Centers Stratified by Infection Type (2015–2016)

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Background. We evaluated and compared the *in vitro* activities of ceftazidime-avibactam (CAZ-AVI) and comparators against *Enterobacteriaceae* (ENT) and *P. aeruginosa* (PSA) from various infection types.

Methods. 23,440 isolates composed of 19,249 ENT and 4,191 PSA were consecutively collected from 85 US hospitals and tested for susceptibility (S) by broth microdilution methods in a central monitoring laboratory (JMI Laboratories). The antimicrobial S and frequency of key resistance (R) phenotypes, such as multidrug-R (MDR) and extensively drug-R (XDR) among others, were assessed and stratified by these infection types: bloodstream (BSI; 3,434 isolates; 14.7%), pneumonia (6,439; 27.5%), skin/skin structure (SSSI; 4,134; 17.6%), intra-abdominal (IAI; 951; 4.1%), urinary tract (UTI; 7,873; 33.6%), and others combined (609; 2.6%).

Results. CAZ-AVI was active against 99.9% to 100.0% of ENT and 97.0% (pneumonia) to 99.4% (UTI) of PSA isolates. S rates were consistently lower among ENT from pneumonia compared with other infection types for β -lactams such as CAZ (82.3% vs. 87.1–90.8%), piperacillin-tazobactam (P-T; 87.5% vs. 90.2–95.6%) and meropenem (MEM; 96.8% vs. 98.4–99.4%). S to gentamicin (GEN) was also generally lower among isolates from pneumonia, whereas S to levofloxacin (LEV) and colistin (COL) were lowest among BSI and SSSI isolates, respectively. The occurrence of MDR, XDR, and carbapenem-resistant ENT (CRE) phenotypes were markedly higher among isolates from patients with pneumonia compared with other infection types (Table). Among PSA, S rates for CAZ, P-T, and GEN were lowest among isolates from pneumonia, whereas S to MEM was similar among isolates from BSI, pneumonia, and IAI (77.3–77.9%), and S to LEV was markedly lower among UTI isolates (67.1%). The frequency of PSA isolates with MDR and XDR phenotypes, as well as non-S to CAZ, MER, and P-T, were also highest among isolates from patients with pneumonia (Table).

Conclusion. Antimicrobial S rates were generally lower among ENT and PSA isolates from patients with pneumonia compared with other infections. CAZ-AVI was highly active against a large collection of contemporary ENT and PSA isolates from US hospitals (2015–2016), including MDR and XDR organisms, regardless of the infection type.

Infection type	Frequency of occurrence (%) <i>Enterobacteriaceae</i>			<i>P. aeruginosa</i>			CAZ, MER, P-T-non-S	
	No. of isolates	MDR	XDR	CRE	No. of isolates	MDR		XDR
BSI	3,137	8.6	0.9	1.0	297	17.2	10.4	8.4
Pneumonia	3,875	12.2	2.6	3.0	2,564	24.7	11.2	9.3
SSSI	3,289	7.1	0.8	1.0	845	11.5	3.9	4.6
IAI	829	7.4	1.0	1.6	122	20.5	9.0	7.4
UTI	7,526	6.6	0.6	0.6	347	20.7	9.2	5.2
Others	593	7.8	1.2	1.9	16	0.0	0.0	0.0
Total	19,249	8.2	1.1	1.3	4,191	21.0	9.4	7.9

Disclosures. H. S. Sader, Allergan: Research Contractor, Research grant; M. Castanheira, Allergan: Research Contractor, Research grant; L. R. Duncan, Allergan: Research Contractor, Research grant; R. K. Flamm, Allergan: Research Contractor, Research grant

1238. Antimicrobial Activity of Dalbavancin Tested against Staphylococcus aureus with Decreased Susceptibility to Glycopeptides, Daptomycin, and/or Linezolid from United States (US) Medical Centers

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Background. Dalbavancin (DALBA) was approved by the US Food and Drug Administration (2014) and European Medicines Agency (2015) for treating acute bacterial skin and skin structure infections. Dalbavancin activity was assessed against a large collection of *S. aureus* clinical isolates with decreased susceptibility (S) to key antimicrobial agents used to treat severe *S. aureus* infections.

Methods. The organism collection included isolates with decreased S to vancomycin (VAN; MIC ≥ 2 μ g/mL; $n = 1,141$), daptomycin (DAPTO; MIC ≥ 2 μ g/mL [resistant (R) per CLSI and EUCAST]; $n = 48$), telavancin (TLV; MIC ≥ 0.12 μ g/mL; $n = 73$), teicoplanin (TEICO; MIC ≥ 4 μ g/mL [non-S (NS) per EUCAST]; $n = 143$), and/or linezolid (LNZ; MIC ≥ 8 μ g/mL [R per CLSI and EUCAST]; $n = 25$). Isolates were selected among 59,903 US isolates tested in 2002–2016. S testing was performed by CLSI methods and MIC results were interpreted per CLSI and EUCAST criteria.

Results. Only 8 of 59,903 (0.01%) *S. aureus* isolates tested were categorized as DALBA-NS (MIC, >0.25 μ g/mL). DALBA retained activity against 99.3% of isolates

with VAN MICs of ≥ 2 μ g/mL (Table), whereas DAPTO (MIC_{50/90} 0.5/1 μ g/mL) and LNZ (MIC_{50/90} 1/2 μ g/mL) were active against 96.8% and 99.6% of isolates, respectively. DALBA (Table) and VAN (MIC_{50/90} 2/2 μ g/mL) retained activity against 95.8% of DAPTO-NS *S. aureus*. When tested against TEICO-NS (EUCAST) isolates, S rates for DALBA, DAPTO, VAN, and LNZ were 95.1%, 95.8%, 97.9%, and 100.0%, respectively; and DALBA was 4- to 32-fold more potent than these comparator agents. All LNZ-R isolates (100.0%) were S to DALBA (MIC_{50/90} 0.06/0.06 μ g/mL), DAPTO (MIC_{50/90} 0.5/0.5 μ g/mL), and VAN (MIC_{50/90} 1/2 μ g/mL), but DALBA was 8- and 16- to 32-fold more potent than DAPTO and VAN, respectively. MRSA rates ranged from 71.2–96.0% among these R subsets.

Conclusion. DALBA retained potent *in vitro* activity against *S. aureus* isolates, displaying decreased susceptibility to agents commonly used to treat serious infections and was consistently more potent than comparator agents.

Resistance phenotype (no. tested)	Number of isolates (cumulative %) inhibited at DALBA MIC (μ g/mL) of:					MIC ₅₀	MIC ₉₀
	≤ 0.03	0.06	0.12	0.25	>0.25		
VAN MIC ≥ 2 μ g/mL (1,141)	117 (10.3%)	697 (71.3%)	276 (95.5%)	43 (99.3%)	8 (100.0%)	0.06	0.12
DAPTO-NS (48)	3 (6.3%)	25 (58.3%)	16 (91.7%)	2 (95.8%)	2 (100.0%)	0.06	0.12
TLV MIC ≥ 0.12 μ g/mL (73)	9 (12.3%)	31 (54.8%)	27 (91.8%)	3 (95.9%)	3 (100.0%)	0.06	0.12
TEICO MIC ≥ 4 μ g/mL (143)	14 (9.8%)	73 (60.8%)	33 (83.9%)	16 (95.1%)	7 (100.0%)	0.06	0.25
Linezolid-R (25)	5 (20.0%)	18 (92.0%)	2 (100.0)			0.06	0.06
All isolates (59,903)	22,066 (36.8%)	33,879	3,795 (99.7%)	155 (100.0%)	8 (100.0%)	0.06	0.06

Bold data represent dalbavancin modal MIC results.

Disclosures. H. S. Sader, Allergan: Research Contractor, Research grant; R. E. Mendes, Allergan: Research Contractor, Research grant; L. R. Duncan, Allergan: Research Contractor, Research grant; M. A. Pfaller, Allergan: Research Contractor, Research grant; R. K. Flamm, Allergan: Research Contractor, Research grant

1239. Evaluation of In Vitro Activity of Cefataroline Tested against Streptococcus pneumoniae Isolates from United States Hospitals: Results from 7 Years of the AWARE Surveillance Program (2010–2016)

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Background. Cefataroline (CPT) is a broad-spectrum cephalosporin with activity against *S. pneumoniae* (SPN), including multidrug-resistant (MDR) strains. CPT fosamil is approved for clinical use in the United States (US) to treat community-acquired bacterial pneumonia (CABP). The AWARE program monitors the *in vitro* activity of CPT against clinical bacteria from various infection types. We evaluated the activity of CPT against isolated SPN clinical isolates from US hospitals collected in 2010 through 2016.

Methods. A total of 8,768 isolates were consecutively collected (1 per patient) from 47 medical centers in 2010–2016 and tested for susceptibility (S) to CPT and comparator agents using CLSI broth microdilution methods. Resistant subgroups included isolates that were nonsusceptible (NS) to penicillin (PCN), ceftriaxone (CRO), amoxicillin-clavulanate (AMC), erythromycin (ERY), clindamycin (CM), and levofloxacin (LEV) as well as MDR (NS to ≥ 3 classes of agents) and extensively drug resistant (XDR; NS to ≥ 5 classes).

Results. CPT inhibited 99.99% of SPN isolates at ≤ 0.5 mg/L (only 1 isolate had a CPT MIC of 1 mg/L) and remained active against all SPN-resistant (R) subgroups, including PCN-NS (8.7% at ≥ 4 mg/L), CRO-NS (6.9% at ≥ 2 mg/L), MDR (21.7%), and XDR (8.4%) strains. CPT activity remained stable against all R subgroups each year. MDR and XDR frequency decreased from 25.0% and 14.1% in 2011 to 17.8% and 3.2% in 2015, respectively; and S to PCN, CRO, AMC, CM, trimethoprim-sulfamethoxazole (TMX), and tetracycline (TET) increased in the same period (Table). The CPT-NS isolate had multiple substitutions in the penicillin binding proteins (PBP), mainly PBP2x, when compared with reference sequences, and showed 31 amino acid alterations in MurM. For MDR isolates, CPT (99.9% S), tigecycline (99.9% S), linezolid (100.0% S), and vancomycin (100.0% S) were the most active agents.

Antibiotic	Overall susceptibility and MDR - XDR occurrences (%)							
	All years (8,768)	2010 (919)	2011 (1,755)	2012 (1,202)	2013 (1,333)	2014 (1,150)	2015 (1,237)	2016 (1,172)
CPT	>99.9	100.0	100.0	100.0	100.0	99.9	100.0	100.0
CRO	93.1	90.4	88.4	91.4	92.6	94.3	98.1	97.7
PCN	91.3	86.1	84.8	90.0	92.3	94.1	96.6	96.8
AMC	88.1	84.0	80.6	86.5	87.9	89.8	95.0	95.2
ERY	54.8	58.4	55.0	56.4	53.1	52.3	55.7	53.4
CM	82.1	77.9	78.1	81.3	82.5	83.5	87.0	85.0
LEV	98.8	99.1	98.8	99.2	98.9	97.7	99.4	98.8
TMX	68.1	65.9	64.4	66.1	67.0	69.5	73.5	71.3
TET	77.1	75.2	74.3	75.5	76.7	77.6	80.1	81.4
MDR	21.7	25.6	25.0	23.3	21.8	20.2	17.8	17.7
XDR	8.4	12.4	14.1	9.7	8.0	5.9	3.2	3.6

ERY, erythromycin; LEV, levofloxacin

Conclusion. CPT demonstrated potent and consistent (2010–2016) activity against SPN, including several R phenotypes and the less S serotypes. SPN S to many antibiotics increased from 2011 to 2015, but remained stable in 2015–2016. Increases in S rates could be related to the anti-pneumococcal vaccine PVC-13 introduced in 2010.

Disclosures. M. A. Pfaller, Allergan: Research Contractor, Research grant; R. E. Mendes, Allergan: Research Contractor, Research grant; L. R. Duncan, Allergan: Research Contractor, Research grant; R. K. Flamm, Allergan: Research Contractor, Research grant; H. S. Sader, Allergan: Research Contractor, Research grant