(ABSSSI; OASIS-1 and OASIS-2) were completed. OMC and comparator efficacies were examined for molecularly characterized baseline pathogens.

Methods. Gram-positive (24) and -negative (17) isolates from the OPTIC (26), OASIS-1 (10) or OASIS-2 (5) trials were selected for characterization. Susceptibility testing and interpretation was performed by CLSI methods. Gram-positive isolates were selected based on tetracycline and/or macrolide, lincosamide, streptogramin$_B$ (MLS_B) phenotypes, and tetracycline-nonsusceptible (NS) Gram-negative isolates were selected. Isolates were subjected to next-generation sequencing followed by screening for known tetracycline and/or MLS_B genes. The efficacy endpoint was investigator's assessment of clinical response at post therapy evaluation (PTE).

Results. All *S. aureus* (eight isolates) exhibited a doxycycline-NS phenotype (MIC, 8–16 µg/mL) and OMC MIC values of 0.25–0.5 µg/mL. All *S. aureus* carried *tet*(M), except one isolate with *tet*(K), and one isolate with *tet*(M) and *tet*(L). All but one *S. pneumoniae* (16 isolates; OMC MIC, 0.03–0.06 µg/mL) carried MLS_B genes, while tetracycline- and doxycycline-NS isolates (12) had *tet*(M). *E. coli* (eight isolates; OMC MIC, 0.5–2 µg/mL), *E. cloacae* (two isolates; OMC MIC, 2 µg/mL), and *K. pneumoniae* (six isolates; OMC MIC, 2–16 µg/mL) carried tetracycline efflux-pump genes, *tet*(A) and/or *tet*(B), *tet*(D), and *tet*(A), respectively. *tet* genes were not detected in one *K. pneumoniae* (OMC MIC, 8 µg/mL). Clinical success was noted in 37/41 (90.2%) patients. Two linezolid-treated patients with *S. aureus* (OMC MIC, 0.25 µg/mL) from OASIS-1 and one OMC-treated patient from OPTIC with *E. coli* (OMC MIC, 2 µg/mL) had indeterminate PTE responses. One OMC-treated patient from OPTIC with *K. pneumoniae* (OMC MIC, 8 µg/mL) was a clinical failure at PTE.

Conclusion. This study expands on OMC efficacy data analysis among patients infected with tetracycline–NS pathogens. These results indicate that OMC *in vivo* efficacy is not affected by tetracycline and/or MLS_B resistance mechanisms.

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1365. *In Vitro* Activity of Lefamulin (LEF) Against Bacterial Pathogens Causing Community-Acquired Bacterial Pneumonia (CABP): SENTRY Surveillance 2016 Results From Asia-Pacific (APAC) and Latin America (LA)

Susanne Paukner, PhD[1]; Robert K. Flamm, PhD[2]; Steven P. Gelone, PharmD[3] and Helio S. Sader, MD, PhD[4]; [1]Nabriva Therapeutics GmbH, Vienna, Austria, [2]JMI Laboratories, Inc., North Liberty, Iowa, [3]Nabriva Therapeutics US, Inc., King of Prussia, Pennsylvania, [4]JMI Laboratories, North Liberty, Iowa

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Background. LEF, a novel pleuromutilin antibiotic for IV and oral use, recently completed a phase 3 trial for the treatment of CABP in adults where it demonstrated noninferiority to moxifloxacin ± linezolid. LEF selectively inhibits bacterial translation. This study investigated the activity of LEF and comparators against contemporary bacterial respiratory pathogens collected from APAC and LA.

Methods. Unique isolates were collected from patients with pneumonia/respiratory (n = 551), blood stream (n = 169), skin and soft tissue (n = 244), and other (n = 55) infections in seven countries in APAC (n = 587) and four countries in LA (n = 432). LEF and comparators were tested by CLSI broth microdilution methods, and susceptibility was determined using CLSI (2018) breakpoints.

Results. In both APAC and LA, LEF showed potent *in vitro* activity against this collection of respiratory pathogens, with 100% of *Streptococcus pneumoniae* inhibited at ≤0.25 µg/mL. *S. pneumoniae* isolates were largely susceptible to moxifloxacin (98.2% APAC, 100.0% LA), amoxicillin/clavulanic acid (84.3% APAC, 89.4% LA), and ceftriaxone (85.2% APAC, 93.6% LA), but less susceptible to azithromycin (56.6% APAC, 68.1% LA) and penicillin (48.2% APAC, 67.0% LA). LEF was also active against *Staphylococcus aureus* with 99.6% of all isolates from both APAC and LA being inhibited at 0.25 µg/mL. 29.5% of methicillin-resistant *S. aureus* in APAC and 24.7% in LA showed particularly high resistance rates to erythromycin (59.3% APAC, 64.2% LA), moxifloxacin (49.4% APAC, 53.7% LA), and clindamycin (39.5% APAC, 59.7% LA). 98.2% and 97.9% of *Haemophilus influenzae* (in APAC and LA, respectively) were inhibited at LEF ≤2 µg/mL, and 100.0% of *Moraxella catarrhalis* were inhibited at LEF ≤0.12 µg/mL in both APAC and LA. Both organisms were largely susceptible to the comparators, except for ampicillin (49.1% and 74.5% susceptible among *H. influenzae* in APAC and LA, respectively) and trimethoprim/sulfamethoxazole (54.4% and 68.1% susceptible among *H. influenzae*) (figure).

Conclusion. In APAC and LA, LEF was highly active against pathogens collected from CABP patients in 2016, and its activity was not affected by resistance to other antibiotic classes. These data support the ongoing development of LEF for the treatment of CABP.

$MIC_{50/90}$ of Lefamulin and Comparators

	MIC_{50} / MIC_{90} (µg/mL)				
Organisms (No. of Isolates)	Lefamulin	Amoxicillin-Clavulanic Acid	Azithromycin	Moxifloxacin	Levofloxacin
Asia-Pacific					
S. pneumoniae (226)	0.06 / 0.12	0.06 / >4	0.06 / >32	0.12 / 0.25	1 / 1
Penicillin susceptible* (192)	0.06 / 0.12	≤0.03 / 2	0.06 / >32	0.12 / 0.25	1 / 1
Penicillin nonsusceptible† (34)	0.06 / 0.12	>4 / >4	>32 / >32	0.12 / 2	1 / >4
S. aureus (275)	0.06 / 0.06	ND	0.5 / >32	≤0.06 / 2	0.25 / >4
MRSA (81)	0.06 / 0.12	ND	>32 / >32	1 / >4	>4 / >4
H. influenzae (57)	0.5 / 1	1 / 8	1 / 2	0.03 / 0.03	0.015 / 0.03
M. catarrhalis (29)	0.06 / 0.12	0.12 / 0.25	0.015 / 0.03	0.06 / 0.06	0.03 / 0.06
Latin America					
S. pneumoniae (94)	0.06 / 0.12	≤0.03 / 4	0.06 / >32	0.12 / 0.25	1 / 1
Penicillin susceptible* (86)	0.06 / 0.12	≤0.03 / 0.25	0.06 / >32	0.12 / 0.25	1 / 1
Penicillin nonsusceptible† (8)	0.06 / –	>4 / –	>32 / –	0.12 / –	1 / –
S. aureus (271)	0.06 / 0.06	ND	0.5 / >32	≤0.06 / 2	0.25 / >4
MRSA (67)	0.06 / 0.12	ND	>32 / >32	2 / >4	>4 / >4
H. influenzae (47)	0.5 / 1	0.5 / 2	0.5 / 2	0.03 / 0.03	0.015 / 0.03
M. catarrhalis (20)	0.06 / 0.12	0.12 / 0.25	0.03 / 0.03	0.06 / 0.06	0.06 / 0.06

MRSA=methicillin-resistant *Staphylococcus aureus*; ND=not determined.
*Penicillin MIC≤2 µg/mL; †Penicillin MIC≥4 µg/mL for the nonmeningitis breakpoint.

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1366. *In Vitro* and *In Vivo* Activity of Cefiderocol against *Stenotrophomonas maltophilia* Clinical Isolates

Akinobu Ito, PhD[1]; Merime Ota, Bachelor[2]; Rio Nakamura, Bachelor[3]; Masakatsu Tsuji, PhD[3]; Takafumi Sato, PhD[3] and Yoshinori Yamano, PhD[3]; [1]Drug Discovery and Disease Research Laboratory, Shionogi & Co., Ltd., Osaka, Japan, [2]Shionogi & Co., Ltd, Toyonaka, Japan, [3]Shionogi & Co., Ltd., Osaka, Japan

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Background. Cefiderocol (S-649266, CFDC) is a novel siderophore cephalosporin against Gram-negatives, including carbapenem (CR)-resistant strains. Its spectrum includes both the Enterobacteriaceae but also nonfermenters, including *Stenotrophomonas maltophilia*—an opportunistic pathogen with intrinsic resistance to carbapenem antibiotics. In this study, *in vitro* activity and *in vivo* efficacy of CFDC and comparators against *S. maltophilia* were determined.

Methods. MICs of CFDC and comparators (trimethoprim/sulfamethoxazole (TMP/SMX), minocycline (MINO), tigecycline (TGC), ciprofloxacin (CPFX), cefepime (CFPM), meropenem (MEPM), and colistin (CL)) were determined by broth microdilution method as recommended by CLSI. The MIC against CFDC was determined using iron-depleted cation-adjusted Mueller–Hinton broth. *In vivo* efficacy of CFDC, CFPM, ceftazidime–avibactam (CAZ/AVI), MEPM, and CL was evaluated using neutropenic murine systemic infection model caused by strain SR21970. The 50% effective doses (ED_{50}s) were calculated by the logit method using the survival number at each dose 7 days after infection.

Results. MIC_{90} of CFDC and comparators against the 216 clinical isolates from global countries collected in SIDERO-CR 2014/2016 study are shown in the table. CFDC, TMP/SMX, MINO, and TGC showed good activity with MIC_{90} of 0.5, 0.25/4.75, 1, and 2 µg/mL, respectively. CFDC, MINO, and TGC inhibited growth of all tested strains at ≤1, ≤4, and ≤8 µg/mL although two strains showed resistance to TMP/SMX. MICs of CFPM, CAZ/AVI, MEPM, and CL were ≥32 µg/mL. The ED_{50} of CFDC against *S. maltophilia* SR21970 with MIC of 0.125 mg/mL was 1.17 mg/kg/dose. Conversely, MICs of CFPM, CAZ/AVI, MEPM/CS, and CL against SR21970 were 32 µg/mL or higher, and ED_{50}s were >100 mg/kg/dose, showing that CFDC had potent *in vivo* efficacy against *S. maltophilia* strain which was resistant to other antibiotics.

	MIC Range (µg/mL)	MIC_{50} (µg/mL)	MIC_{90} (µg/mL)
CFDC	≤0.031 to 1	0.063	0.5
TMP/SMX	≤0.031/≤0.589 to 16/304	0.125/2.375	0.25/4.75
MINO	0.063 to 4	0.25	1
TGC	0.125 to 8	1	2
CPFX	0.5 to >32	2	16
CFPM	2 to >32	32	>32
MEPM	0.25 to >32	>32	>32
CL	0.125 to >32	4	16

Conclusion. CFDC showed potent *in vitro* activity against *S. maltophilia*, including TMP/SMX-resistant isolates. CFDC also showed potent *in vivo* efficacy reflecting *in vitro* activity against *S. maltophilia* in murine systemic infection model.

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1367. Clinical Cure in Secondary Efficacy Populations in Patients With Complicated Urinary Tract Infection Treated With ZTI-01 (Fosfomycin for Injection): Findings From the ZEUS Trial

Keith Kaye, MD, MPH[1]; Louis B. Rice, MD, FIDSA[2]; Viktor Stus, MD, PhD[3]; Olexsiy Sagan, MD[4]; Elena Fedosiuk, MD[5]; Anita Das, PhD[6]; David Skarinksy, BS[7]; Paul B. Eckburg, MD[7]; Kristina Manvelian, MS[7] and Evelyn J. Ellis-Grosse, PhD[7]; [1]Department of Internal Medicine, Division of Infectious Diseases, University of Michigan Medical School, Ann Arbor, Michigan, [2]Brown University, Providence, Rhode Island, [3]Municipal Institution Dnipropetrovsk Medical Academy of MOH of Ukraine, Dnipropetrovsk, Ukraine, [4]Department of Urology, Communal Institution Zaporizhzhia Regional Clinical Hospital, Zaporizhzhia, Ukraine, [5]Brest Regional Hospital, Brest, Belarus, [6]Das Statistical Consulting, Guerneville, California, [7]Zavante Therapeutics, Inc., San Diego, California

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Background. ZTI-01 (fosfomycin for injection) is an investigational epoxide antibiotic with a differentiated mechanism of action (MOA) inhibiting an early step in bacterial cell wall synthesis. ZTI-01 has a broad spectrum of *in vitro* activity, including multidrug-resistant Gram-negative pathogens, and is being developed for the treatment of patients with complicated urinary tract infection (cUTI) and acute pyelonephritis (AP) in the United States.

Methods. ZEUS was a multicenter, double-blind, Phase 2/3 trial in hospitalized adults with cUTI and AP to evaluate safety and efficacy. Randomized patients received 6 g ZTI-01 q8h or 4.5 g IV piperacillin/tazobactam (PIP-TAZ) q8h for 7 days; patients

with baseline bacteremia could receive up to 14 days; study continued to late follow-up (LFU, 26 ± 2 days). Oral step-down therapy was prohibited. ZTI-01 met the primary endpoint of noninferiority to PIP-TAZ. Secondary objectives included comparing clinical cure rates (assessed by investigator) in the modified intent-to-treat (MITT), microbiologic MITT (m-MITT), clinical evaluable (CE), and microbiologic evaluable (ME) populations at test-of-cure (TOC, Day 19 ± 2 days).

Results. There were 464 patients randomized who received study drug. In all populations, clinical cure rates at TOC were high and similar between treatment groups (>90%) (table).

Conclusion. These results demonstrate consistent efficacy in multiple secondary efficacy populations for patients with cUTI and AP who were treated with either ZTI-01 or PIP-TAZ. If approved by FDA, ZTI-01 may provide a new IV option with a differentiated MOA for patients in the United States with serious Gram-negative infections.

Table: Clinical Response at TOC

	ZTI-01	PIP-TAZ		
Population	*n* (%)	*n* (%)	Difference (%)	95% CI
MITT	233	231		
Cure	211 (90.6)	212 (91.8)	−1.2	(−6.8, 4.4)
Failure	11 (4.7)	16 (6.9)		
Indeterminate	11 (4.7)	3 (1.3)		
m-MITT	184	178		
Cure	167 (90.8)	163 (91.6)	−0.8	(−7.2, 5.6)
Failure	9 (4.9)	12 (6.7)		
Indeterminate	8 (4.3)	3 (1.7)		
CE	199	196		
Cure	188 (94.5)	182 (92.9)	1.6	(−3.7, 6.9)
Failure	11 (5.5)	14 (7.1)		
ME	155	145		
Cure	148 (95.5)	135 (93.1)	2.4	(−3.5, 8.3)
Failure	7 (4.5)	10 (6.9)		

95% confidence intervals (CIs, two-sided) were computed using a continuity-corrected Zstatistic.

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1368. Assessment of the *In Vivo* Efficacy of Human-Simulated Epithelial Lining Fluid (ELF) Exposure of Meropenem/Nacubactam (MEM/NAC) Combination Against β-Lactamase-Producing Enterobacteriaceae in Neutropenic Lung Infection Model

Tomefa E. Asempa, PharmD[1]; Ana Motos, MSc[1]; Kamilia Abdelraouf, PhD[1]; Caterina Bissantz, PhD[2]; Claudia Zampaloni, PhD[3] and David P. Nicolau, PharmD, FCCP, FIDSA[1,4]; [1]Center for Anti-Infective Research and Development, Hartford Hospital, Hartford, Connecticut, [2]Roche Pharma Research and Early Development Pharmaceutical Science, Roche Innovation Center Basel, F. Hoffmann-La Roche Ltd., Basel, Switzerland, [3]Roche Pharma Research and Early Development, Immunology, Inflammation and Infectious Diseases, Roche Innovation Center Basel, F. Hoffmann-La Roche Ltd., Basel, Switzerland, [4]Division of Infectious Diseases, Hartford Hospital, Hartford, Connecticut

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Background. NAC is a novel dual action β-lactamase inhibitor with *in vitro* activity against class A, class C, and some class D β-lactamases and antibacterial activity against *Enterobactaeriaceae*. NAC is being developed as a combination therapy with MEM for the treatment of serious Gram-negative bacterial infections. This study evaluated the efficacy of the human-simulated ELF exposure of MEM/NAC, compared with those of MEM or NAC alone against β-lactamase-producing *Enterobacteriaceae* isolates in the neutropenic murine lung infection model.

Methods. Eight clinical MEM-resistant *Enterobacteriaceae* isolates harboring various β-lactamases (IMI, KPC, OXA, TEM, SHV, and AmpC) were utilized in the study. MEM and MEM:NAC (1:1) combination MICs were determined in triplicate via broth microdilution. ICR mice were rendered transiently neutropenic, and lungs were inoculated with 50 µL bacterial suspensions of 10^7 CFU/mL. Regimens in mice that simulated the human ELF exposures following doses of MEM 2g q8h and NAC 2g q8h (1.5 hours infusions) as monotherapies and in combination were established. Treatment mice received MEM human-simulated regimen (HSR), NAC HSR, or MEM/NAC HSR and control mice were vehicle-dosed. Treatment was started 2 hours after inoculation and continued for 24 hours. Efficacy was assessed as the change in $\log_{10}$CFU/lung at 24 hours compared with 0 hours controls.

Results. MEM and MEM/NAC MICs were 8–512 and 0.5–8 mg/L, respectively. The average $\log_{10}$CFU/lung at 0 hours across all isolates was 6.26 ± 0.26. Relative to 0 hours control, the mean bacterial growth at 24 hours in the untreated control mice, MEM HSR, and NAC HSR treatment groups were 2.93 ± 0.29, 2.72 ± 0.42, and 1.75 ± 0.80 $\log_{10}$CFU/lung, respectively. MEM/NAC HSR resulted in up to 2-log bacterial reduction in isolates with MEM/NAC MIC ≤4 mg/L.

Conclusion. MEM/NAC human-simulated ELF exposure produced enhanced efficacy against MEM-resistant β-lactamase-producing *Enterobacteriaceae* isolates with MEM/NAC MIC ≤4 mg/L. These data support a potential role for MEM/NAC for treatment of lung infections due to β-lactamase-producing *Enterobacteriaceae* and warrant further studies.

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1369. Combined Analysis of the In Vitro Activity of Ridinilazole (RDZ) Against More Than 500 *Clostridium difficile* (CD) Clinical Isolates and Impact of RDZ on Cell Morphology

Esther Duperchy, PhD[1]; Eugénie Bassères, PhD[2]; Kevin Garey, PharmD, MS[3] and Richard Vickers, PhD[1]; [1]R&D, Summit Therapeutics, Abingdon, UK, [2]University of Houston College of Pharmacy, Houston, Texas, [3]University of Houston College of Pharmacy, Houston, Texas

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Background. *Clostridium difficile* infection (CDI) is one of the most urgent bacterial healthcare threats in the United States. RDZ is a targeted spectrum, GI restricted, antibacterial currently in clinical development for the treatment of CDI and reducing the recurrence of CDI. Here we report the combined analysis of previously reported and new independent studies assessing the susceptibility of CD clinical isolates collected in North America and Europe between 2010 and 2015, and the effect of RDZ on cell morphology.

Methods. A total of 570 CD clinical isolates across seven independent studies were tested for susceptibility. The majority of isolates (>70%) were sourced from RDZ Phase 2 clinical trials and North American and European surveillance programs. Minimum inhibitory concentrations (MIC) were determined by agar dilution on Wilkins Chalgren agar plates after 48 hours incubation at 37°C, or, by agar or microbroth dilution using supplemented Brucella medium following the CLSI guidelines M11-A7/A8. Up to 11 comparator antibiotics were tested alongside RDZ. PCR ribotyping was performed on 549 isolates by capillary gel electrophoresis. To investigate the impact of RDZ on cell morphology, CD strain R20291 was incubated with RDZ at 0.125–0.5 × MIC concentrations for 24 hours. DAPI and FM4-64 staining was used to visualize DNA and cell membrane by confocal microscopy.

Results. RDZ was highly active against the isolates collected in North America and Europe with MICs distributed over a narrow range (0.015–0.5 µg/mL) and an overall MIC_{90} of 0.25 µg/mL. There was no variation in activity by geographic region or ribotype, including hypervirulent ribotype 027 isolates ($N = 83$). RDZ also maintained activity against antibiotic-resistant isolates, including isolates with reduced susceptibility to metronidazole and vancomycin. When treated with sub-MIC concentrations of RDZ, CD cells formed filamentous structures with a dose-dependent effect on cell length and decreased septum formation. This preliminary data suggest that RDZ may alter CD cell division.

Conclusion. These data show that RDZ was highly active against recent CD isolates independent of geographic origin, ribotype, and antibiotic resistance profile. Mechanism of action studies are ongoing and further susceptibility profiling will be undertaken during the Phase 3.

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1370. Cefepime/VNRX-5133 Broad-Spectrum Activity Is Maintained Against Emerging KPC- and PDC-Variants in Multidrug-Resistant *K. pneumoniae* and *P. aeruginosa*

Denis Daigle, PhD[1]; Jodie Hamrick, BSc[1]; Cassandra Chatwin, BSc[1]; Natalia Kurepina, PhD[2]; Barry N. Kreiswirth, PhD[2]; Ryan K. Shields, PharmD[3]; Antonio Oliver, PhD[4]; Cornelius J. Clancy, MD[5]; Minh-Hong Nguyen, MD[3]; Daniel Pevear, PhD[1] and Luigi Xerri, PhD[1]; [1]VenatoRx Pharmaceuticals Inc., Malvern, Pennsylvania, [2]Public Health Research Institute, Rutgers New Jersey Medical School, Newark, New Jersey, [3]University of Pittsburgh, School of Medicine, Pittsburgh, Pennsylvania, [4]Hospital Son Espases, Palma de Mallorca, Spain, [5]Infectious Diseases, University of Pittsburgh, Pittsburgh, Pennsylvania

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Background. VNRX-5133 is a cyclic boronate β-lactamase inhibitor (BLI) currently in clinical development with cefepime to treat multidrug-resistant (MDR) infections caused by ESBL- and carbapenemase-producing Enterobacteriaceae (ENT) and