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

Organism (n)	$MIC_{50/90}$ (mg/L)					
	Lefamulin	Amoxicillin/ Clavulanic acid	Azithromycin/ Erythromycin*	Ceftaroline/ Ceftriaxone†	Moxifloxacin	Tetracycline/ Doxycycline‡
S. pneumoniae (1,441)	0.12/0.25	≤0.03/2	0.06/>16	0.03/1	0.12/0.25	0.5/>4
Penicillin resistant (156)	0.12/0.25	2/>4	16/>16	1/2	0.12/0.25	1/>4
Macrolide resistant (657)	0.12/0.25	0.25/4	8/>16	0.25/1	0.12/0.25	0.5/>4
Tetracycline resistant (293)	0.12/0.25	0.25/>4	>16/>16	0.25/2	0.12/0.25	>4/>4
S. aureus (297)	0.06/0.12	ND	4/>8	0.25/1	≤0.06/>4	0.12/0.5
MRSA (133)	0.06/0.12	ND	>8/>8	1/2	2/>4	0.12/1
Macrolide resistant (144)	0.06/0.12	ND	>8/>8	0.5/2	2/>4	0.12/1
Fluoroquinolone resistant (97)	0.06/0.12	ND	>8/>8	1/2	>4/>4	0.12/1
H. influenzae (382)	0.5/2	0.5/2	1/2	0.004/0.015	0.03/0.06	0.5/1
M. catarrhalis (165)	0.06/0.12	≤0.25/≤0.25	≤0.03/≤0.03	0.25/1	0.06/0.06	0.25/0.5
Beta-lactamase positive (161)	0.06/0.12	≤0.25/≤0.25	≤0.03/≤0.03	0.25/1	0.06/0.06	0.25/0.5
Beta-hemolytic streptococci (14)	0.03/0.06	ND	0.03/4	0.03/0.06	0.12/0.25	ND

MRSA=methicillin-resistant *S. aureus*; ND=not determined.

*Erythromycin for *S. pneumoniae*, *S. aureus*, and beta-hemolytic streptococci; azithromycin for *H. influenzae* and *M. catarrhalis*.

†Ceftriaxone for *S. pneumoniae*, *H. influenzae*, *M. catarrhalis*, and beta-hemolytic streptococci; ceftaroline for *S. aureus*.

‡Tetracycline for *S. pneumoniae*, *H. influenzae*, and *M. catarrhalis*; doxycycline for *S. aureus*.

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704. Incidence and Patient Outcomes of *S. aureus* Isolates from Acute Bacterial Skin and Skin Structure Infections (ABSSSI) with High Iclaprim MIC values in Phase 3 REVIVE Trials

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Background. The incidence and outcomes of patients with *S. aureus* isolates with an iclaprim MIC >8 µg/mL, a concentration that is not systemically achievable, were determined among patients from two Phase 3 studies for the treatment of ABSSSI, REVIVE-1 and -2.

Methods. REVIVE-1 and REVIVE-2 studies were 600-patient, double-blinded, randomized (1:1), active-controlled trials among patients with ABSSSI that compared the safety and efficacy of iclaprim 80 mg fixed dose with vancomycin 15 mg/kg (adjusted for renal function), both administered intravenously over 2 hours every 12 hours for 5–14 days. Patients had a bacterial skin infection suspected or confirmed to be due to a Gram-positive pathogen with a lesion size ≥75 cm^2. An early clinical response (ECR) was defined as a ≥20% reduction in lesion size compared with baseline at the early time point (ETP) 48–72 hours after the start of administration of the study drug in the intent-to-treat (ITT) population. A clinical cure, defined as complete resolution of all signs and symptoms of ABSSSI was measured at the end of therapy (EOT) and test of cure (TOC) visit, 7–14 days after the last dose of study drug. At baseline, EOT and TOC visits, ABSSSIs were sampled for microbiological culture and broth microdilution susceptibility testing conducted in accordance with CLSI M7.

Results. The incidence of culture confirmed *S. aureus* isolates among patients with ABSSSI with an iclaprim MIC >8 µg/mL was 2.0% (16/790). Six were MSSA and 10 were MRSA. The clinical outcomes of these infections included ECR of 63% (10/16), EOT response of 81.3% (13/16) and the TOC response of 75% (12/16). For microbiological outcomes of these infections, the end of therapy response was 92.9% (13/14) and the test of cure response was 92.3% (12/13). In comparison, there was less variation in vancomycin MICs among the *S. aureus* isolates. For patients who were randomized to vancomycin and had a pathogen identified from their ABSSSI, the pooled ECR was 82.6% (242 of 293) at a vancomycin MIC of 0.5–1 µg/mL and one isolate from a patient with ECR had a MIC of 2 µg/mL.

Conclusion. Patients receiving iclaprim had good clinical and microbiological responses against *S. aureus* isolates with an iclaprim MIC >8 µg/mL, which are uncommon (2.0%).

Disclosures. **All authors:** No reported disclosures.

705. Pharmacokinetics (PK) and Safety of Lefamulin (LEF) After Single Intravenous Dose Administration in Subjects With Impaired Renal Function and in Those Requiring Hemodialysis

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Background. Renal comorbidities are common in patients hospitalized with community-acquired bacterial pneumonia (CABP). LEF, a novel pleuromutilin antibiotic (IV/oral), was generally well tolerated and noninferior to moxifloxacin in two phase 3 studies of adults with CABP. We investigated the PK and safety of LEF and its main metabolite, BC-8041, in subjects with severe renal impairment and those requiring hemodialysis (HD).

Methods. In this open-label study, subjects were allocated to 1 of 3 groups based on renal function level. Severe subjects (estimated glomerular filtration rate <30 mL/minute/1.73 m^2, not on HD, Severe) were matched (gender, age, and weight) to subjects with normal renal function (estimated creatinine clearance ≥90 mL/minute, Normal). Subjects in the Normal and Severe groups received a single 1-hour 150 mg LEF infusion. Subjects in the HD group started HD within 1 hour after LEF infusion ("On-dialysis") and on a nondialysis day ("Off-dialysis"). Blood and urine samples were collected predose and over a 36-hour period postdose for PK analysis; LEF and BC-8041 were assayed in plasma and urine with validated methods. Safety assessments included treatment-emergent adverse events (TEAEs), labs, vital signs, and electrocardiograms.

Results. 23 subjects enrolled in and completed the study (n = 7, Normal; n = 8, Severe; n = 8, HD). LEF and BC-8041 pharmacokinetic parameters (table) were comparable between the Normal and Severe groups and between the On-dialysis and Off-dialysis treatment periods for the HD group. The majority of LEF and BC-8041 were excreted nonrenally in Normal and Severe subjects and were not measurably filtered into dialysate. TEAEs were reported in 2 (28.6%) subjects in the Normal group, 4 (50%) in the Severe group, and 4 (50%) in the HD group. None of the TEAEs were serious or led to study drug discontinuation. Within 4 h post-dose, the maximum mean change from baseline in the QTcF interval was 8.9, 6.6, 15.9, and 17.6 msec in the normal, severe, on-dialysis, and off-dialysis groups, respectively.

Conclusion. No dosage adjustment is required for LEF when treating subjects with severe renal impairment, and LEF can be administered without regard to HD timing. LEF was generally well tolerated in all subjects regardless of renal function status.

Table. Mean (SD) Lefamulin and BC-8041 PK Parameters by Renal Functional Status Group

PK Parameter	Normal (n=7)	Severe (n=8)	Hemodialysis (n=8)	
			On-Dialysis	Off-Dialysis
Lefamulin				
C_{max}, ng/mL	3182 (697)	3138 (990)	3341 (916)	2893 (653)
t_{max}, h	1.0 (0.0)	1.1 (0.1)	1.0 (0.0)	1.0 (0.0)
AUC, h•ng/mL	9004 (2591)	12262 (7798)	8955 (3103)	8606 (2815)
CL, L/h	17.9 (5.4)	15.7 (7.2)	18.6 (6.4)	19.0 (5.6)
$t_{1/2}$, h	10.1 (1.9)	9.4 (0.9)	9.3 (1.4)	9.1 (0.9)
BC-8041				
C_{max}, ng/mL	48.7 (12.8)	56.1 (15.7)	60.0 (40.0)	51.2 (21.9)
t_{max}, h	1.3 (0.0)	1.3 (0.1)	1.4 (0.1)	1.4 (0.3)
AUC, h•ng/mL	413 (134)	695 (448)	734 (716)	643 (408)
$t_{1/2}$, h	13.5 (4.5)	11.4 (2.2)	15.1 (4.4)	12.8 (2.0)

AUC=area under the plasma concentration-time curve extrapolated through infinity; CL=systemic clearance (observed) estimated using AUC; C_{max}=maximum observed concentration; PK=pharmacokinetic; SD=standard deviation; $t_{1/2}$=elimination half-life; t_{max}=time of maximum observed concentration.

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706. *In Vitro* Activity of Ceftazidime–Avibactam and Comparator Agents Against MDR *Enterobacteriaceae* and *Pseudomonas aeruginosa* Collected in Latin America During the ATLAS Global Surveillance Program 2016–2017

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Background. Ceftazidime–avibactam (CAZ-AVI) is a β-lactam/non-β-lactam β-lactamase inhibitor combination that can inhibit class A, C and some class D β-lactamases. Resistance caused by these β-lactamases often results in multidrug-resistance (MDR). This study evaluated the *in vitro* activity of CAZ-AVI and comparators against MDR *Enterobacteriaceae* and *Pseudomonas aeruginosa* isolates collected from patients in Latin America.

Methods. Nonduplicate clinical isolates were collected in 2016–2017 in 6 countries in Latin America. Susceptibility testing was performed using CLSI broth microdilution and interpreted using CLSI 2019 and FDA (tigecycline) breakpoints. MDR was defined as nonsusceptible (NS) (intermediate or resistant) to ≥3 of 7 sentinel drugs: amikacin, aztreonam, cefepime, levofloxacin, colistin, meropenem, and piperacillin–tazobactam.

Results. The activity of CAZ-AVI and comparators against all isolates and MDR subsets is shown in the table. MDR rates ranged from 28.4% among *E. cloacae* to 41.5% among *K. pneumoniae*. CAZ-AVI was active against >97% of *Enterobacteriaceae* isolates and maintained activity against >92% of MDR isolates of the examined species. No other tested drug consistently exceeded this activity. Among *P. aeruginosa*, CAZ-AVI was active against 87% of all isolates and 63% of MDR isolates; only colistin was more active. The two most common MDR phenotypes among *Enterobacteriaceae* were (1) NS to aztreonam, cefepime, and levofloxacin (n = 580, 41% of all MDR *Enterobacteriaceae;* 100% susceptible to CAZ-AVI) and (2) NS to aztreonam, cefepime, levofloxacin, and piperacillin–tazobactam (n = 301, 21% of all MDR isolates; 99.7% susceptible to CAZ-AVI). The two most common MDR phenotypes among *P. aeruginosa* were (1) NS to all sentinel drugs except colistin (n = 154, 33% of all MDR isolates;

30.5% susceptible to CAZ-AVI) and (2) NS to all drugs except colistin and amikacin (*n* = 97, 21% of all MDR isolates; 70.1% susceptible to CAZ-AVI).

Conclusion. These *in vitro* data suggest that CAZ-AVI can be an effective treatment option for infections caused by MDR *Enterobacteriaceae* and *P. aeruginosa* collected in Latin America.

	Drug (% susceptible)					
Species/phenotype (n)	**CAZ-AVI**	**CAZ**	**MEM**	**AMK**	**CST**	**TGC**
All *Enterobacteriaceae* (5381)	99.3	70.5	94.4	96.7	82.7	97.3
MDR (1426)	97.3	11.4	79.1	88.3	88.1	97.3
All *E. coli* (1848)	99.95	71.2	99.2	98.4	99.1	99.9
MDR (547)	99.8	13.5	97.4	95.2	98.5	100
All *K. pneumoniae* (1499)	98.7	54.2	84.7	93.1	94.7	97.5
MDR (622)	97.1	4.8	63.3	84.6	88.9	96.1
All *E. cloacae* (356)	97.8	61.8	93.8	95.5	94.4	97.8
MDR (101)	92.1	2.0	79.2	84.2	95.0	96.0
All *P. aeruginosa* (1347)	87.2	69.9	66.1	82.4	99.6	NA
MDR (462)	62.8	22.3	17.1	51.9	99.6	NA

CAZ-AVI, ceftazidime-avibactam; CAZ, ceftazidime; MEM, meropenem; AMK, amikacin; CST, colistin; TGC, tigecycline; NA, not applicable

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707. QPX9003: Pharmacology of a Novel Polymyxin in Mice and Rats

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Background. Currently available polymyxins are limited by toxicity and poor efficacy at tolerated doses. We have developed a new series of polymyxin derivatives with improved safety profiles and *in vitro* potency against major MDR bacteria. The following describes studies on the *in vivo* antimicrobial activity and toxicity of QPX9003 in mice and rats.

Methods. Mouse studies. The minimum lethal dose (MLD by IV bolus) and nephrotoxicity (6 IP doses administered 2 hours apart) of QPX9003 and polymyxin B (PMB) were determined in Swiss mice. For the neutropenic mouse thigh infection using *A. baumannii*, Swiss mice were infected with ~10^6 CFU/thigh. Doses were administered IP at various intervals starting 2-hour post-infection and continued over 24 hours. *Rat studies*. For the rat lung infection model, Sprague-Dawley rats were infected with ~10^7 CFU/lung. QPX9003 and PMB were administered IV every 4 hours starting 2 hours post-infection and continued over 24 hours. *Bacteria*. For both infection models, animals were infected with *A. baumannii* AB1016 (QPX9003 MIC of 0.5 mg/L and PMB MIC of 1.0 mg/L). Untreated control groups were sacrificed at the start of treatment and both untreated and treated groups were sacrificed 24 hours after the start of treatment, infected tissues harvested, homogenized, and plated to determine colony counts.

Results. QPX9003 had reduced acute toxicity and nephrotoxicity compared with PMB in mice. QPX9003 showed better bacterial killing of *A. baumannii* than PMB at similar plasma exposures in both the mouse thigh model (−0.41 vs. +0.83 log CFU/thigh) and rat lung infection model (−1.10 vs. +1.44 log CFU/lung).

Conclusion. QPX9003 was less acutely toxic, less nephrotoxic, and was more efficacious in mouse and rat infection models compared with PMB. QPX9003 is a promising new polymyxin. (This work was supported in part by federal funds from the National Institutes of Allergy and Infectious Diseases [R01AI098771], and the Department of Health and Human Services; Office of the Assistant Secretary for Preparedness and Response; Biomedical Advanced Research and Development Authority (BARDA), under OTA number HHSO100201600026C).

Compounds	Mouse: Single Dose MLD IV (mg/kg)	Mouse: Kidney Changes (10 mg/kg IP x 6 doses)	Rat Lung Model: 24h AUC for 1-log bacterial killing vs. *A. baumannii*
PMB	7.5	Minimal to Severe Nephrosis	>160
QPX9003	20	No change	46

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708. *In Vitro* Activity of Plazomicin vs. Clinical Isolates of Gram-Negative Bacilli, Including Aminoglycoside Nonsusceptible and Multidrug-Resistant Subsets, Recovered from Patients Across Canada as Part of the CANWARD study, 2011–2018

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Background. Plazomicin (PLZ) is a next-generation aminoglycoside currently approved by the US FDA for the treatment of complicated urinary tract infections, including pyelonephritis. The purpose of this study was to evaluate the *in vitro* activity of PLZ against a large collection of Gram-negative bacilli obtained from patients attending Canadian hospitals.

Methods. Annually from 2011 to 2018, sentinel hospitals across Canada submitted blood, respiratory, urine, and wound isolates from patients attending ERs, medical and surgical wards, hospital clinics, and ICUs (CANWARD). Susceptibility testing was performed using broth microdilution (and breakpoints) as described by CLSI (FDA breakpoints used for PLZ).

Results. See table.

S, susceptible; NS, nonsusceptible; ESBL, extended-spectrum β-lactamase; MDR, multidrug-resistant (NS to antimicrobials from three or more classes); n.d., not defined.

Conclusion. PLZ demonstrated excellent *in vitro* activity vs. *E. coli* and *K. pneumoniae* clinical isolates, including aminoglycoside NS, ESBL-positive, and MDR subsets.

Organism/Phenotype (Number tested)	PLZ		Gentamicin		Meropenem	
	$MIC_{50/90}$	%S	$MIC_{50/90}$	%S	$MIC_{50/90}$	%S
Escherichia coli						
ALL (4793)	0.5/1	99.4	≤0.5/2	90.4	≤0.03/≤0.03	99.9
Gentamicin NS (458)	0.5/1	98.9	32/>32	0.0	≤0.03/≤0.03	99.6
Tobramycin NS (405)	0.5/1	98.8	32/>32	31.6	≤0.03/≤0.03	99.5
ESBL-positive (489)	0.5/1	99.8	1/>32	66.5	≤0.03/0.06	99.8
MDR (570)	0.5/1	99.1	2/>32	50.4	≤0.03/0.06	99.6
Klebsiella pneumoniae						
All (1627)	0.25/0.5	99.8	≤0.5/≤0.5	96.5	≤0.03/0.06	99.3
Gentamicin NS (57)	0.25/1	96.5	32/>32	0.0	≤0.03/1	91.2
Tobramycin NS (78)	0.25/0.5	97.4	32/>32	38.5	0.06/2	89.7
ESBL-positive (104)	0.25/0.5	98.1	≤0.5/>32	53.9	≤0.03/1	91.4
MDR (116)	0.25/0.5	97.4	1/>32	53.4	≤0.03/1	90.5
***Klebsiella aerogenes* (174)**	0.5/0.5	99.4	≤0.5/≤0.5	99.4	0.06/0.12	98.9
***Klebsiella oxytoca* (447)**	0.25/0.5	99.8	≤0.5/≤0.5	98.2	≤0.03/0.06	100.0
***Enterobacter cloacae* (736)**	0.25/0.5	99.6	≤0.5/≤0.5	97.2	≤0.03/0.12	99.2
***Morganella morganii* (90)**	2/4	73.3	≤0.5/2	92.2	0.06/0.25	100.0
***Proteus mirabilis* (357)**	4/8	43.1	≤0.5/2	93.3	0.06/0.12	100.0
***Serratia marcsesens* (419)**	0.5/1	97.9	≤0.5/1	99.3	0.06/0.06	99.5
***Pseudomonas aeruginosa* (2665)**	4/16	n.d.	1/8	88.7	0.5/8	79.4

Disclosures. **All authors:** No reported disclosures.

709. *In Vitro* Antibacterial Activity and *In Vivo* Efficacy of Sulbactam–Durlobactam (ETX2514SUL) Against Pathogenic *Burkholderia* Species

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Background. The genus Burkholderia contains several pathogenic species with distinct etiologies, including *Burkholderia pseudomallei* the biothreat pathogen responsible for melioidosis and *Burkholderia mallei* which causes glanders. β-Lactams, such as ceftazidime and meropenem, are important therapeutic options for these infections. However, clinical resistance to β-lactams, which is primarily mediated by multiple types of β-lactamases in these species, is a growing concern. Durlobactam (ETX2514, DUR) is a novel β-lactamase inhibitor with broad-spectrum activity against Ambler class A, C, and D β-lactamases. Sulbactam (SUL) is an Ambler Class A β-lactamase inhibitor with intrinsic antibacterial activity against a limited number of species, including *Acinetobacter* spp. SUL-DUR is currently in Phase 3 clinical testing for the treatment of carbapenem-resistant infections caused by *Acinetobacter* spp. In this study, SUL-DUR was tested for *in vitro* antibacterial activity against *B. pseudomallei* and *B. mallei* as well as for *in vivo* efficacy in a preclinical model of melioidosis.

Methods. The antibacterial activity of SUL alone or in combination with DUR (fixed at 4 mg/L) against *B. pseudomallei* (*n* = 30) and *B. mallei* (*N* = 28) was determined following CLSI guidelines. *In vivo* efficacy was tested in an acute murine model of melioidosis in which 4 × 10^4 cfu *Bp* K96423 (SUL-DUR MIC = 1 mg/L) was administered intranasally to BalbC mice. SUL-DUR (100/200 or 400/200 mg/kg) was administered q4h subcutaneously 4 hours post-challenge for 6 days and murine survival was monitored for 45 days. Doxycycline (DOX) and ciprofloxacin (CIP) were dosed as positive controls at 40 mg/kg q12 h for 6 days.

Results. The addition of DUR effectively lowered the SUL $MIC_{50/90}$ from 8/16 to 0.25/0.5 mg/L vs. *B. pseudomallei* and from 8/8 to 1/2 mg/L for *B. mallei.* All untreated mice in the melioidosis model succumbed to infection within 3 days of challenge. 60% survival was observed for both dose arms of SUL-DUR as compared with 40% survival observed for both CIP and DOX.

Conclusion. Preliminary preclinical data demonstrating robust *in vitro* and *in vivo* antibacterial activity of SUL-DUR against *Burkholderia* spp. suggests this combination may be an effective new therapy for the treatment of these challenging pathogens.

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710. *In Vitro* Activity and Performance of Available Susceptibility Testing Methods for Eravacycline Against Carbapenem-Resistant Enterobacteriaceae (CRE)

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