(CAV), cefepime (CEF), ciprofloxacin (CIP), colistin (CL), meropenem (MR), ceftolozane/tazobactam (C/T), tigecycline (TG), and trimethoprim/sulfamethoxazole (T/S).

Results. CFDC MIC_{90}s as mg/L were: *S. maltophilia* [50 isolates] 0.25, *E. coli* (ESBL−) [50 isolates] 0.5, *E. coli* (ESBL+) [51 isolates] 2.0, *K. pneumoniae* (ESBL− and +) [60 isolates] 0.5; *K. pneumoniae* (CRE) [22 isolates] 2.0; *P. aeruginosa* (MDR) [32 isolates] 1.0; *E. cloacae* [27 isolates] 4.0; *Achromobacter* spp. [15 isolates] 0.12. CFDC inhibited *P. agglomerans*, *Burkholderia* spp., *Sphingomonas* spp., *Ochrobactrum* spp. at ≤1 mg/L [23 total isolates] and *Elizabethkingia* spp. and *R. radiobacter* at ≤8 mg/L [11 total isolates]. Among comparator agents, only T/S had consistent activity against *S. maltophilia*. For *E. coli* (ESBL− and +) MR, TG, CAV, CL were most active. For *K. pneumoniae* (ESBL−and +) MR, CAV were most active. For *K. pneumoniae* (CRE) and *P. aeruginosa* (MDR), none of the comparators had significant activity. For *E. cloacae*, MR, A, CAV, TG were most active. Among the uncommon organisms, MR and TG had the greatest activity.

Conclusion. Although susceptibility breakpoints have yet to be determined, CFDC has significant activity (≤4 mg/L) against most problematic Gram-negative organisms causing infections in CP based on available pharmacokinetic/pharmacodynamic data. In particular, its activity against *S. maltophilia* was superior to the comparators. Also, it was the most active agent against *P. aeruginosa* (MDR) and *K. pneumoniae* (CRE). Based on our results, CFDC warrants clinical evaluation for the treatment of blood stream infections caused by GNB in CP.

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1376. Antifungal Activity of Cerium Nitrate Against Fungal Isolates Associated with Combat-Related Injuries Including Burns

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Background. Fungal infections are a critical cause of morbidity and mortality in burn patients. In addition to debridement and systemic antifungal therapy, various topical adjuncts have been used, and topical burn care is a key component of infection prevention and treatment. Cerium nitrate (CN) has been used in combination with silver sulfadiazine (SS) in burn care. Previous studies showed that CN had bacteriostatic activity, and suggested anti-biofilm activity against *Candida* biofilms. In this study, we evaluated the *in vitro* activity of CN against fungal isolates associated with combat-related injuries.

Methods. The efficacy of CN was evaluated against 14 mold (three *Aspergillus* spp., two *Fusarium* spp., five different mucormycetes, two *Bipolaris* spp., one *Alternaria* spp., one *Exophiala* spp.) and 21 *Candida* spp. isolates collected as part of the Trauma Infectious Disease Outcomes Study. Fungicidal activity of various concentrations of CN (2.2%, 1%, 0.5% and 0.2%) was determined using an established time-kill assay. Standard conidia/cell suspensions were prepared according to Clinical and Laboratory Standards Institute guidelines and then exposed to the CN solutions for 24 hours. At different times (0, 5, 15, 30 minutes, 1, 1.5, 3, 6, 12, and 24 hours) aliquots were plated and incubated at 35ºC. Colony forming unit (CFU) counts were determined after 24 hours incubation or after an appropriate time for slow growing molds.

Results. All mold isolates had persistent growth at 24 hours with most having no significant change in colony counts over the 24-hour period. The only exception was *Mucor circinelloides*, which appeared to have a time-dependent reduction in CFUs at 24 hours for all CN concentrations. *Exophiala* did not grow as well in CN solutions compared with the control (mean 65 vs. 28.2 CFUs with a difference of mean 37.4 CFUs, $P = 0.0001$), but this was not time or concentration dependent. All yeast species showed a time-dependent killing after 6–12 hours.

Conclusion. CN demonstrated time-dependent killing of the yeasts. However, very little activity was observed against the tested molds. Since CN is often used in combination with SS there might be a synergistic effect against molds. Further research will evaluate higher concentrations of CN and its toxicity for cells and tissue.

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1377. Omadacycline *In Vitro* Activity Against a Molecularly Characterized Collection of Clinical Isolates with Known Tetracycline Resistance Mechanisms

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Background. Omadacycline is a novel aminomethylcycline that recently completed Phase 3 clinical trials for the treatment of acute bacterial skin and skin structure infections (ABSSSIs) and community-acquired bacterial pneumonia (CABP). This study evaluated the activity of omadacycline against a broad collection of recent (2016) clinical isolates with molecularly characterized tetracycline resistance mechanisms.

Methods. A total of 177 Gram-positive and -negative clinical isolates were identified as carrying acquired tetracycline resistance genes and were included in this study. Isolates were previously subjected to next-generation sequencing followed by screening of known tetracycline resistance mechanisms. Susceptibility testing and interpretation were performed according to CLSI methods.

Results. Omadacycline demonstrated MIC_{50} values of 0.06–0.12 µg/mL against Gram-positive isolates carrying tet genes. Similar MIC results (0.06–0.12 µg/mL) were obtained against Gram-positive organisms carrying tet(K), tet(L)/tet(M) or tet(M). Omadacycline ($MIC_{50/90}$, 0.12/0.25 µg/mL) and tigecycline ($MIC_{50/90}$, 0.06/0.25 µg/mL) showed similar MIC results when tested against *Staphylococcus aureus* carrying tet(K). While tetracycline was less active (0.0–78.6% susceptible) against Tet(K)-producing *S. aureus,* doxycycline ($MIC_{50/90}$, 0.5/0.5 µg/mL; 100.0% susceptible) was active *in vitro*. Omadacycline (MIC_{90}, 0.25–2 µg/mL) and tigecycline (MIC_{90}, 0.12–1 µg/mL) showed potent MIC results against Gram-positive isolates carrying tet(L) and/or tet(M). Tetracycline and doxycycline had MIC_{90} values of ≥8 µg/mL. Omadacycline (MIC_{90} 4–32 µg/mL) and tigecycline (MIC_{90} 0.5–2 µg/mL) were active against Gram-negative isolates harboring tet(A), tet(B) or tet(D) or a combination of tet. Tetracycline ($MIC_{50/90}$, >16/>16 µg/mL) and doxycycline ($MIC_{50/90}$, >8/>8 µg/mL) had elevated MIC_{50} and MIC_{90} results against these isolates.

Conclusion. Results presented here indicate that omadacycline is not adversely affected by tet genes present in contemporary Gram-positive and -negative clinical isolates, a characteristic that differs from the legacy tetracycline agents.

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1378. Evaluation of the *In vitro* Activity of Meropenem-Vaborbactam Against Carbapenem-Resistant *Enterobacteriaceae*, Including Isolates Resistant to Ceftazidime–Avibactam

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Background. Meropenem-vaborbactam (M-V) is a novel antibiotic for treatment of carbapenem-resistant *Enterobacteriaceae* (CRE) infections. Our objective was to determine the *in vitro* activity of meropenem-vaborbactam against genetically-diverse CRE isolates, including those that have developed resistance to Ceftazidime–Avibactam (C-A).

Methods. Minimum inhibitory concentrations (MICs) were determined for meropenem (MER), M-V, and C-A by reference broth microdilution (BMD) methods in triplicate. Vaborbactam and avibactam were tested at fixed concentrations of 8 and 4 µg/mL, respectively. Quality control strains were used and within expected ranges. Polymerase chain reaction (PCR) with DNA sequencing was used to detect resistance determinants, including *Klebsiella pneumoniae* carbapenemase (KPC) subtypes and porin mutations.

Results. A total of 117 CRE isolates were tested, including *K. pneumoniae* (*Kp*; $n = 83$), *E. cloacae* ($n = 17$), *E. coli* ($n = 10$), and *E. aerogenes* ($n = 7$). Seventy-nine percent harbored bla_{KPC}. KPC subtypes included KPC-2 ($n = 32$), KPC-3 ($n = 41$), KPC-3 variants ($n = 16$), and KPC [not typed] ($n = 4$, all *E. coli*). Among 74 *K. pneumoniae*, 95% had a premature stop codon in *ompk35* and *ompK36* genotypes included wild type ($n = 48$), IS5 insertion ($n = 13$), 135–136 DG duplication ($n = 9$), and other mutations ($n = 4$). The median (range) MICs for MER, C-A, and M-V were 8 (0.06 to ≥128), 1 (0.25 to ≥512), and 0.03 (0.015−−16), respectively. Corresponding rates of susceptibility were 23, 84, and 98%, respectively. Fifty-three percent and 95% of C-A-resistant isolates were susceptible to MER and M-V, respectively. Among *Kp*, C-A MICs did not vary by KPC subtype or porin genotype. On the other hand, median M-V MICs were higher among KPC-2 than KPC-3 *Kp* (0.12 vs. 0.03; $P = 0.002$), and among *Kp* with *ompK36* porin mutations compared with wild type (0.25 vs. 0.03; $P < 0.001$). Among *Kp* with KPC-3 variants ($n = 16$), the median M-V MIC was 0.03 (0.015−−2); 100% were M-V susceptible. Median M-V MICs did not vary by CRE species. Only two isolates were M-V resistant, both were *E. cloacae* that did not harbor bla_{KPC}.

Conclusion. M-V demonstrates high rates of *in vitro* susceptibility against diverse CRE isolates, including those that are resistant to C-A. As this agent is introduced into the clinic, it will be important to identify *K. pneumoniae* isolates harboring KPC-2 with *ompK36* porin mutations that demonstrate higher MICs.

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