therapeutic options include combinations of aztreonam (ATM), which is resistant to hydrolysis by MBLs, plus ceftazidime/avibactam (CZA) or meropenem/vaborbactam (M/V) for coverage of relevant SBLs. However, these selections add a level of complexity to clinical management compared with administration of a single antibiotic as monotherapy.

Methods. Minimum inhibitory concentrations (MICs) of WCK 5222 (ceftepime/zidebactam), ATM, CZA, and M/V were determined with Liofilchem MIC Test Strips against SBL- and MBL-positive CRE (N = 15). The gradient diffusion strip (GDS) cross-method was used to assess the activities of CZA+ATM and M/V+ATM. Additive interactions were defined as fractional inhibitory concentration indices ≤ 1 would be predicted based upon the known genotypic profiles; thus, the relative activities of the combination regimens were compared with the “zone of hope” (ZOH) test. The size of the ZOH (the zone of inhibited growth) was quantitated by multiplying the observed length of inhibited growth (in mm) adjacent to each GDS from the point of intersection. The Mann–Whitney rank-sum test was used to assess differences.

Results. All isolates (N = 15) contained one MBL and ≥1 SBL, and were resistant to ATM, CZA, and M/V with the exception of one isolate intermediate to M/V (MIC = 8 mg/L). WCK 5222 MIC (range) was 1 (0.19–2) mg/L. The median (inter-quartile range) ZOH product for CZA+ATM and M/V+ATM was 75.4 (62.8–93.7) and 23.5 (14.1–60.4), respectively (P = 0.002). In strains that produced OXA-type carbapenemases (n = 6), the median ZOH product for CZA+ATM and M/V+ATM was 78.1 and 20.4, respectively (P = 0.002). In strains with ≥2 anaerobes (i.e., the MBL), the median ZOH product for CZA+ATM and M/V+ATM was 73.8 and 25.6, respectively (P = 0.052).

Conclusion. WCK 5222 displayed potent in vitro activity against SBL- and MBL-positive CRE, warranting further pre-clinical in vivo evaluation, as a monotherapy option. When considering the co-expression of SBL and MBL, CZA+ATM appears to offer enhanced coverage compared with M/V+ATM.

Disclosures. All authors: No reported disclosures.

686. Evaluation of Contezolid Activity to Anaerobic and Gram-positive-cocci Isolates from a Phase 3 Acute Bacterial Skin and Skin Structure Infection Clinical Trial (MRX-1-06)
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Background. Contezolid (MRX-1) is an oxazolidinone in development for the treatment of acute bacterial skin and skin structure infections (ABSSSI). In this study, in vitro susceptibility (S) for Contezolid and comparator agents for Gram-positive (GP) and anaerobic isolates from Phase 3 ABSSSI clinical trials were determined.

Methods. 313 isolates were collected from 65 participated sites and sent to a central lab for MIC testing. Clinical isolates included 34 anaerobic (15 Finegolda magna, 8 Actinomyces spp., 4 Prevotella spp., 3 Propionibacterium avidum, 2 Peptostreptococcus spp., 1 Veillonella spp. and 1 Bacteroides fragilis), 187 S. aureus (59.7%), 12 S. pyogenes, 5 Enterococcus, and 75 other Gram-positive organisms. Broth microdilution was used to determine the MIC of selected broth microdilution regimens of ceftriaxone and other comparators to facultative isolates. Agar dilution was carried out for the remaining 9 strains with a single carbapenem resistance. YU253911's activity was measured against a collection of 200 previously described (whole-genome sequencing) Acinetobacter isolates including 98 carbapenem-resistant A. baumannii strains. YU253911 antimicrobial activity was also evaluated in combination with clinically relevant broad-spectrum combinations that achieve the desired antibacterial activity.

Results. In vitro susceptibility of eravacycline was determined by broth microdilution against a collection of 200 previously described (whole-genome sequencing) Acinetobacter isolates. YU253911 potency was similar with an MIC50 of 1 μg/mL (Genetic analysis showed β-lactamase genes, including OXA-23 and other carbapenemases, were common in both YU253911-resistant and susceptible strains.

Conclusion. YU253911 demonstrates promising in vitro potency against a collection of Acinetobacter isolates and compares favorably to β-lactam antibiotic. Understanding interactions with PBP agents and β-lactamase inhibitors is being explored as well as further studies on the mechanism of resistance.

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688. In vitro Activity of Eravacycline, a New Tetracycline Analog, and Comparators Against the Six Most Commonly Isolated Ribotypes of Clostridioides difficile
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Background. Eravacycline is a novel, tetracycline class antibacterial indicated for the treatment of complicated intra-abdominal infections in adults. In clinical trials, patients given eravacycline had a low likelihood of developing Clostridioides difficile infection (CDI). We hypothesized this was likely due, in part, to the in vitro susceptibility of eravacycline to C. difficile. The purpose of this study was to test the in vitro susceptibility of eravacycline vs. comparators on contemporary clinical isolates representing common ribotypes, including isolates with decreased susceptibility to metronidazole and vancomycin.

Methods. Two hundred and thirty-four isolates from our biobank were selected from the six most common ribotypes (F001, F002, F014-020, F027, F106, and F255). Minimum inhibitory concentrations (MIC) at 24 hours were measured according to CLSI guidelines for eravacycline, vancomycin, metronidazole and fidaxomicin. MIC results were tabulated and are presented as the geometric mean by ribotype.

Results. Geometric MIC results are shown in Table 1. Eravacycline was the most potent antimicrobial tested followed by fidaxomicin, metronidazole, and vancomycin. Results were consistent amongst all ribotypes, including isolates with reduced susceptibility to vancomycin and metronidazole.

Conclusion. Eravacycline displayed potent in vitro activity against a large collection of C. difficile isolates. These data provide insight into why patients given eravacycline had a low likelihood of developing CDI and support further research to better understand the use of eravacycline to prevent or potentially treat patients with CDI.

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