Acinetobacter baumannii (AB) is one of the most challenging Gram-negative pathogens, especially in the intensive care unit setting, and causes bloodstream, respiratory tract, and wound infections. These infections have also led to higher rates of morbidity and mortality in critically ill patients. Furthermore, the treatment of AB is complicated by the emergence of multidrug-resistant (MDR) strains. In the past, cephalosporins were considered the drug of choice against AB infections; however, the increasing occurrence of carbapenem-producing isolates has led to a concerning scenario, highlighting the need for alternative agents with novel anti-AB activity.

The MICs of ZID, WCK 5153 and reference drugs - FEP, SUL and MEC were determined by using standard CLSI broth microdilution method for the strains ATCC 19606. BLEs potentiated dependent on PBP3 binding profile with high affinity to MDR AB. Despite being poor inhibitors of OXA-23, BLEs, FEP and SUL demonstrated relatively higher affinity towards PBP2 as potent inhibitors of this carbapenemase as reported previously for β-lactamase.

**RESULTS**

The β-lactams such as FEP, SUL and MEC were determined by using membrane isolations from a reference strain - A. baumannii ATCC 19606. Briefly, membrane preparations were obtained by the protocol previously described. PBPs were visualised and IC50 values determined (Typhoon™), and following protocol.

The WCK enhancers-ZID and WCK 5153 brought about ≥3 log reduction (Table 1). The MICs of standalone FEP and SUL were 32-64 and >2 mg/L respectively, addition of ZID or WCK 5153 (8 µg/mL) significantly reduced MICs of FEP and SUL within 6h (Table 1). MEC demonstrated relatively higher affinity towards PBP2 as potent inhibitors of this carbapenemase as reported previously for β-lactamase.

**DISCLOSURES**

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**REFERENCES**

2. ICAAC. 2010; F1–2139.
4. PBP binding assays
5. β-lactamase inhibition.
6. Table 3.
7. Table 1.
8. Figure 1.
9. Figure 2.
10. Table 2.
11. Table 3.
12. Figure 1.
13. Figure 2.
14. Table 1.
15. Table 2.
16. Table 3.
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19. Table 1.
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