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BRIEF REPORT

In vitro interactions of sulbactam/durlobactam in combination with meropenem, ceftazidime/avibactam, piperacillin/tazobactam, cefiderocol and fosfomycin against carbapenem-resistant *Acinetobacter baumannii* (CRAB) clinical isolates

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Abstract
We evaluated in vitro activity of sulbactam/durlobactam in combination with different antimicrobials against Carbapenem-Resistant *Acinetobacter baumannii* (CRAB) clinical isolates with different susceptibility profiles, including sulbactam/durlobactam-resistant strains. The genomes of 13 CRAB clinical isolates were characterized by whole-genome sequencing and synergy testing was performed with MIC Test Strips. Sulbactam/durlobactam, when combined with piperacillin/tazobactam or ceftazidime/avibactam, showed synergistic activity against 53.8% (7/13) of CRAB isolates and restored meropenem MIC values below the clinical breakpoint in 46.2% (6/13) of them. Our results demonstrate that sulbactam-durlobactam in combination with β-lactams exhibited high in vitro synergistic activity against CRAB strains.

Keywords Synergy testing · MIC test strip · Whole-genome · Sulbactam/durlobactam · MTS-SAS™

Introduction

Carbapenem-resistant *Acinetobacter baumannii* (CRAB) is a significant cause of healthcare-associated infections and constitutes a critical public health concern by exhibiting a multidrug-resistant (MDR) phenotype with limited available treatment options [1, 2].

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Since 2023, FDA has approved sulbactam/durlobactam (SUL-DUR) for the treatment of hospital-acquired and ventilator-associated bacterial pneumonia (HAP/VAP) due to CRAB in adults. Sulbactam is a β-lactam antibiotic that targets penicillin binding protein-1 (PBP-1) and PBP-3 and exhibits inhibitory activity against Ambler class A β-lactamases [3]. Durlobactam (formerly ETX2514) is a next-generation diazabicyclooctane β-lactamase inhibitor, with potent activity against serine β-lactamases of classes A, C, and D including carbapenem-hydrolyzing class D β-lactamases produced by *A. baumannii* such as OXA-23, and exhibits intrinsic antibacterial activity through inhibition of PBP2 [4].

Despite its recent approval, the emergence of SUL-DUR-resistant strains has been reported in several countries [5, 6]. To overcome this emergence and reduce the spreading of SUL-DUR-resistant strains, synergy testing could be used as a valuable approach to define novel antimicrobial combination treatments and limit the diffusion of such resistance in CRAB. This study aimed to assess the in vitro synergistic activity of SUL-DUR in combination with different antimicrobials against genome-characterized CRAB isolates.

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Material and methods

The study included clinical CRAB strains isolated from various biological sources and from different patients hospitalized at two Italian hospitals between 2019 and 2022. Species identification was performed by the MALDI-TOF MS assay (Bruker Daltonics, Bremen, Germany). Antimicrobial susceptibility testing was performed using MIC Test Strips (Liofilchem, Roseto, Italy). Cefiderocol susceptibility was confirmed by reference broth microdilution using iron depleted Muller Hinton Broth (Liofilchem, Roseto, Italy). Minimum Inhibitory Concentration (MIC) results were interpreted using EUCAST clinical breakpoints v15.0 (available at: https://www.eucast.org/clinical_breakpoints/), except for sulbactam/durlobactam, which was interpreted according to CLSI breakpoints (susceptible ≤ 4 mg/L; resistant > 16 mg/L; 34th edition of CLSI M100, 2024).

Genomic characterization of CRAB strains was performed as previously described [6]. Briefly, genomes weres equenced using the Illumina MiSeq platform (Illumina, CA, USA) and assemblies were performed using SPAdes v.3.15.3. Antimicrobial resistance genes and MLST analysis were performed using CGE server (https://www.genomicepidemiology.org) and analysis of PBP genes was manually performed by aligning the deduced amino acid sequences of each isolate compared to reference alleles of ATCC17978 *A. baumannii* strain using ClustalO software as previously described [5]. Phylogenomic analysis was performed based on core genome SNPs among CRAB isolates included in this study and clinical strains collected in Italy using Parsnp software using the genome of ATCC17978 strain as reference [5].

Antimicrobial combinations were tested using MIC Test Strip – Synergy Application System (Liofilchem®, Roseto, Italy) by crossing MIC Test Strips at the respective MICs for each isolate and incubated at 37 °C for 24 h. Fractional inhibitory concentration (FIC) index (FICI) was calculated as: FIC of agent A + FIC B, where FIC A is the MIC of the combination/MIC of drug A alone, and FIC B is the MIC of the combination/MIC of drug B alone. The FIC index was interpreted as follows: synergy, FIC ≤ 0.5; indifferent, 0.5 > FIC ≤ 4; antagonism, FIC ≥ 4 [7].

Results

Antimicrobial susceptibility profiles of CRAB included in this study are shown in Table 1. Phenotypic testing showed that all CRAB strains included in the study were resistant to meropenem, while all were susceptible to cefiderocol as determined by both MIC Test Strip and broth microdilution. In addition, 4 out of 13 strains (30.7%) were resistant to SUL-DUR. All strains showed elevated MICs for piperacillin and piperacillin/tazobactam (> 256 µg/ml), ceftazidime/avibactam (48 to > 256 µg/ml), and fosfomycin (32 to > 256 µg/ml).

Clinical and genomic characteristics of the CRAB clinical isolates included in this study are shown in the Table 2. MLST analysis showed that 9 out of 13 (69.2%) CRAB strains belonged to ST195, 2 (15.4%) to ST231, and 2 out of 13 (15.4%) belonged to the ST1837 following Oxford scheme. Also, MLST analysis based on Pasteur scheme is shown in Table 2. Phylogenetic tree demonstrated that the isolates included in this study were related to other CRAB isolated in Italy, while the two $bla_{\text{NDM-1}}$ harboring CRAB segregated separately to other Italian strains (Figure S1 in the Supplementary material).

Table 1 MIC results of CRAB strains included in this study against different antimicrobials

Name	MIC (µg/ml)						
	SUD	FDC[a]	CAZ	MRP	FOS	TZP	PIP
BO403	1.5	0.12	> 256	24	96	> 256	> 256
BO415	1.5	0.25	> 256	24	48	> 256	> 256
BO416	1.5	0.25	> 256	24	128	> 256	> 256
BO423	1.5	0.5	> 256	24	64	> 256	> 256
BO427	1	0.5	> 256	32	64	> 256	> 256
BO428	1.5	0.12	> 256	24	96	> 256	> 256
BO432	2	0.12	> 256	16	128	> 256	> 256
BO440	3	0.5	> 256	24	128	> 256	> 256
BO441	2	0.5	> 256	32	64	> 256	> 256
TO19	> 64	1	> 256	24	> 256	> 256	> 256
TO28	> 64	2	> 256	32	32	> 256	> 256
TO35	12	0.5	48	24	> 256	> 256	> 256
TO48	24	0.5	96	16	> 256	> 256	> 256

[a]FDC MICs obtained using broth microdilution method

SUD, Sulbactam/Durlobactam; *FDC*, Cefiderocol; *CAZ*, Ceftazidime/Avibactam; *MRP*, Meropenem, *FOS*, Fosfomycin; *TZP*, Piperacillin/Tazobactam; *PIP*, Piperacillin

Table 2 Genotypic and clinical characteristics of CRAB strains included in this study

Strain	Date of collection	Ward	Origin	ST (Oxford Scheme)	ST (Pasteur Scheme)	Antimicrobial resistance determinants			PBP mutations				
						Aminoglycosides	β-lactams	Sulfamide	PBP1a	PBP1b	PBP2	PBP3	PBP5
BO403	2020	ICU	LRT	195	2	*armA, aph(3'')-Ib [aadA1], aph(3'')-Ia*	$bla_{\text{OXA-23}}$, $bla_{\text{TEM1-D}}$,$bla_{\text{ADC-25}}$,$bla_{\text{OXA-66}}$		WT	P112S	WT	A515V	N310S
BO415	2020	ICU	LRT	195	2	*armA, aph(3'')-Ib [aadA1]*	$Bla_{\text{OXA-23}}$,$bla_{ADC-25,}$ bla_{OXA-66}	*sul2*	WT	P112S	WT	A515V	N310S
BO416	2020	ICU	LRT	195	2	*armA, aph(3'')-Ib [aadA1], aph(3'')-Ia*	$bla_{\text{OXA-23}}$, $bla_{\text{TEM1-D}}$,$bla_{\text{ADC-25}}$,$bla_{\text{OXA-66}}$	*sul2*	WT	P112S	WT	A515V	N310S
BO423	2020	ICU	BLOOD	195	2	*armA, aph(3'')-Ib [aadA1], aph(3'')-Ia*	$bla_{\text{OXA-23}}$, $bla_{\text{TEM1-D}}$,$bla_{\text{ADC-25}}$,$bla_{\text{OXA-66}}$		WT	P112S	WT	A515V	N310S
BO427	2020	ICU	BLOOD	195	2	*armA, aph(3'')-Ib [aadA1]*	$Bla_{\text{OXA-23}}$,$bla_{\text{ADC-25}}$,$bla_{\text{OXA-66}}$	*sul2*	WT	P112S	WT	A515V	N310S
BO428	2020	ICU	BLOOD	195	2	*armA, aph(3'')-Ib [aadA1], aph(3'')-Ia*	$bla_{\text{OXA-23}}$, $bla_{\text{TEM1-D}}$,$bla_{\text{ADC-25}}$,$bla_{\text{OXA-66}}$		WT	P112S	WT	A515V	N310S
BO432	2020	ICU	LRT	195	2	*armA, aph(3'')-Ib [aadA1], aph(3'')-Ia*	$bla_{\text{OXA-23}}$, $bla_{\text{TEM1-D}}$,$bla_{\text{ADC-25}}$,$bla_{\text{OXA-66}}$		WT	P112S	WT	A515V	N310S
BO440	2020	ICU	LRT	195	2	*armA, aph(3'')-Ib [aadA1], aph(3'')-Ia*	$bla_{\text{OXA-23}}$, $bla_{\text{TEM1-D}}$,$bla_{\text{ADC-25}}$,$bla_{\text{OXA-66}}$		WT	P112S	WT	A515V	N310S
BO441	2020	ICU	LRT	195	2	*armA, aph(3'')-Ib [aadA1], aph(3'')-Ia*	$bla_{\text{OXA-23}}$, $bla_{\text{TEM1-D}}$,$bla_{\text{ADC-25}}$,$bla_{\text{OXA-66}}$		WT	P112S	WT	A515V	N310S
TO19	2022	IM	BLOOD	231	1	*armA, aac(3)-Ia, ant(2'') Ia, aph(3'')-Ib*	$bla_{\text{OXA-23}}$,$bla_{\text{OXA-69}}$, $bla_{\text{ADC-25}}$, $bla_{\text{NDM-1}}$		T38A, A244T, T776A	N511H	P665A	L480I, T511S	WT
TO28	2022	BU	BLOOD	231	1	*armA, aac(3)-Ia, ant(2'')-Ia, aph(3'')-Ia*	$bla_{\text{OXA-23}}$,$bla_{\text{OXA-69}}$, $bla_{\text{ADC-25}}$, $bla_{\text{NDM-1}}$		T38A, A244T, T776A	N511H	P665A	L480I, T511S	WT
TO35	2021	BU	BLOOD	1837	389	*armA, aph(3'')-Ib, aph(6)-Id*	$bla_{\text{OXA-23}}$,$bla_{\text{OXA-66}}$, $bla_{\text{ADC-25}}$	*sul2*	A184V	P112S, G137R	WT	N392T	N329S
TO48	2021	BU	BLOOD	1837	389	*armA, aph(3'')-Ib, aph(6)-Id*	$bla_{\text{OXA-23}}$,$bla_{\text{OXA-66}}$, $bla_{\text{ADC-25}}$	*sul2*	WT	P112S, G137R	WT	N392T	N329S
REF[a]	REF[a]	REF[a]	REF[a]	REF[a]	REF[a]	REF[a]	REF[a]	REF[a]	WT	P112S	WT	H370Y A346V	N329S
REF[b]	REF[b]	REF[b]	REF[b]	REF[b]	REF[b]	REF[b]	REF[b]	REF[b]	T38A, A244T, T776A	P665A	P665A	WT	WT

ST, Sequence TypE; *ICU*, Intensive Care Unit; *LRT*, Low Respiratory Tract; *WT*, Wild Type; *IM*, Internal Medicine; *BU*, Burn Unit; *WT*, Wild Type; *REF[a]*, Reference genome previously published by Iacono et al.[12],; *REF[b]*, Reference genome previously published by Vallenet[13]

Genetic analysis showed that all isolates carried antimicrobial resistance determinants to aminoglycosides and β-lactams. In detail, 100% (13/13), 69.2% (9/13) and 61.5% (8/13) of the isolates harboured respectively *armA, aph(3")-Ib [aadA1] and aph(3")-Ia* genes conferring resistance determinants to different aminoglycosides (i.e. gentamicin, tobramycin, amikacin, streptomycin, etc.), while 100% (13/13) and 15.4% (2/13) carried respectively $bla_{\text{OXA-23}}$/bla_{ADC-25} and $bla_{\text{NDM-1}}$ β-lactamases.

In order to identify the mechanism related to the resistance to SUL-DUR, a deep analysis of the PBP genes, primary targets of sulbactam, was performed. Analysis of PBP genes demonstrated that all isolates carried mutations in PBP1 and PBP-3 against reference genome of ATCC17978 strain (Table 3). In detail, a substitutions (*i.e.* N392T in PBP3) were observed in SUL-DUR-resistant non-NDM-producing strains.

The results of synergy testing are shown in Fig. 1 panel A and Table 3. SUL-DUR in combination with cefiderocol and fosfomycin was indifferent, respectively, against 84.6% (11/13) and 76.9% (10/13) of the strains included in this study, whereas in combination with meropenem was synergistic against 15.4% (2/13) of CRAB clinical isolates. In addition, SUL-DUR in combination with either ceftazidime/avibactam or piperacillin/tazobactam displayed synergistic activity against 53.4% (7/13) of CRAB strains. At the same time, SUL-DUR exhibited synergistic effect in combination with piperacillin alone against 46.2% (6/13) of CRAB strains.

Deeper examination of the synergy results showed that SUL-DUR did not reduced significantly the MICs of ceftazidime/avibactam, fosfomycin, piperacillin/tazobactam, and piperacillin against all clinical isolates (Fig. 1 panel B2, B4, B5 and B6), while it restored the MIC of MRP below the clinical breakpoint in 46.2% (6/13) of the CRAB strains (Fig. 1 panel B3). In particular, SUL-DUR restored the susceptibility to MRP in one out of two SUL-DUR-resistant CRAB, while did not decreased the MIC below the clinical breakpoint for resistance of the meropenem in the second SUL-DUR-resistant isolate and NDM-producing CRAB (Figure S2 in the Supplementary material). At the same time, SUL-DUR in combination with cefiderocol exhibited MIC below the clinical breakpoint against all CRAB strains included in this study (Fig. 1 panel B1).

Discussion

Here, we evaluated the synergistic activity of SUL-DUR with beta-lactams or beta-lactam/beta-lactamase inhibitor combinations against CRAB isolates. In the CRAB collection used in this study, four clinical isolates exhibited resistance to SUL-DUR due to the production of metallo-β-lactamase enzymes (i.e., n = 2 NDM-1 producers) or point mutations within the PBP3 gene (i.e. N392T), confirming the role of this mutation in reduced antimicrobial activity against SUL-DUR [5, 6].

Synergy experiments demonstrated that SUL-DUR, in combination with ceftazidime/avibactam or with piperacillin alone or combined with tazobactam, exhibited synergistic activity against CRAB clinical isolates, including strains resistant to SUL-DUR. However, we observed that SUL-DUR did not reduce the MICs of ceftazidime/avibactam or piperacillin below the resistance breakpoints for all CRAB isolates, despite exhibiting synergistic effects in these combinations. Therefore, although SUL-DUR in combination with ceftazidime/avibactam or with piperacillin/tazobactam demonstrated synergistic activity in vitro, its clinical use is unlikely, as the synergy is insufficient to reduce MICs to clinically treatable levels. In contrast, the combination of

Table 3 FIC results of CRAB strains included in this study against different antimicrobials

Name	Fractional Inhibitory Concentration					
	SUD/FDC	SUD/CAZ	SUD/MRP	SUD/FOS	SUD/TZP	SUD/PIP
BO403	2	0.5	1	1	0.63	0.7
BO415	0.55	0.75	1.33	1	0.42	0.5
BO416	1.5	0.44	0.67	1	0.5	0.5
BO423	1.17	0.36	0.83	1.42	0.7	0.63
BO427	1.5	0.44	0.88	1	0.5	0.63
BO428	1.17	0.88	0.83	1	0.5	0.5
BO432	7.3	0.5	0.44	0.75	0.5	0.5
BO440	3.9	0.38	0.83	0.7	0.42	0.5
BO441	0.88	0.5	0.75	0.88	0.5	0.5
TO19	2	2	2	2	1.13	0.69
TO28	2	2	1.5	2	0.75	1.13
TO35	1	1	0.42	1	0.88	0.88
TO48	1	0.58	0.63	1.42	1	0.58

SUD, Sulbactam/Durlobactam; *FDC*, Cefiderocol; *CAZ*, Ceftazidime/Avibactam; *MRP*, Meropenem, *FOS*, Fosfomycin; *TZP*, Piperacillin/Tazobactam; *PIP*, Piperacillin

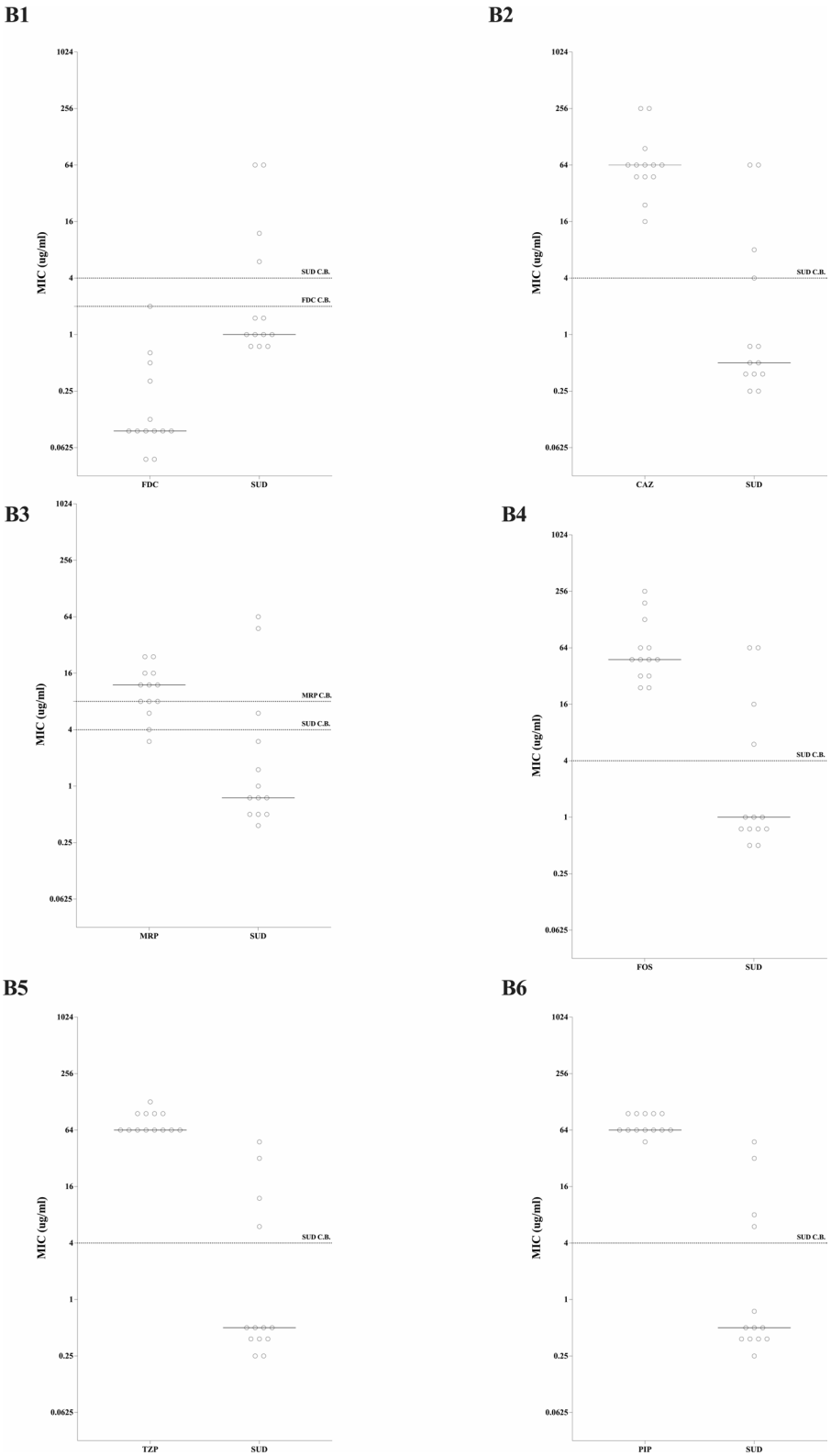

Fig. 1 Synergistic activity of sulbactam/durlobactam (SUL-DUR) in combination with different antimicrobials against Carbapenem-Resistant *Acinetobacter baumannii* (CRAB) Clinical Isolates by showing the cumulative FIC indexes for each combination (Panel A) and the relative reduced MICs of different molecules tested in combination. Panel **A**, FIC indexes (FICs) of SUL-DUR in combinations with FDC, CAZ, MRP, FOS, TZP, and PIP; Panel **B**, MICs reduction of FDC (B1), CAZ (B2), MRP (B3), FOS (B4), PTZP (B5), and PIP (B6) in combination with SUL-DUR. Abbreviations: C.B. Clinical Breakpoint; Cefiderocol (FDC), Ceftazidime/Avibactam (CAZ), Meropenem (MRP), Fosfomycin (FOS), Piperacillin/Tazobactam (TZP), and Piperacillin (PIP)

SUL-DUR with cefiderocol demonstrated high in vitro activity against all CRAB strains included in this study, even though the synergistic interaction was weak. We hypothesized that high synergistic activities observed for SUL-DUR in combination with ceftazidime/avibactam, or piperacillin alone or in combination with tazobactam may be related to the similar target of such molecules (*i.e.* PBP-3). Of note, although cefiderocol displayed a similar target, no synergistic effect has been observed when tested in combination with SUL-DUR reflecting the high antibacterial activities of such molecules alone against CRAB. At the same time, the synergistic activity observed between meropenem and SUL-DUR suggests a cooperative effect between agents that primarily target PBP2, such as meropenem and durlobactam.

Based on these findings, we hypothesized that SUL-DUR in combination with meropenem could represent a promising option for infections caused by CRAB strains by enhancing antimicrobial activity. The simultaneous targeting of different PBPs (PBP1/PBP3 by SUL-DUR and PBP2 by meropenem), together with β-lactamase inhibition by durlobactam, may broaden the in vitro activity against CRAB, including strains resistant to SUL-DUR due to mutations in PBP1 and PBP3. Of note, our results showed that the addition of meropenem restored the susceptibility (*i.e.* 4 µg/ml) to SUL-DUR in one out of two resistant *A. baumanii* strains due to PBP1b-PBP3 mutations, while the MIC of SUL-DUR was reduced to 8 µg/ml in the other SUL-DUR-resistant CRAB. On the other side, SUL-DUR restored the MICs for meropenem below the clinical breakpoints in both SUL-DUR-resistant strains due to PBP mutations, while this association was ineffective against SUL-DUR-resistant NDM-producers. Previous studies have demonstrated synergistic activity of sulbactam/durlobactam combined with imipenem against *Acinetobacter* spp. time–kill experiments [8]. These findings reinforce and contextualize our results, highlighting that the addition of carbapenems to SUL-DUR could be used for the treatment of infections due to CRAB by enhancing the bactericidal activity of these molecules and also by possibly preventing the insurgence of resistance to this novel antimicrobial combination in non-MBL-producing CRAB [9–13].

The study has some limitations, including the relatively small number of strains analyzed and their origin from Italy only. Furthermore, the results obtained require further investigation using time–kill assays to confirm and extend our findings. Further in vivo studies will be necessary to evaluate the combined in vitro effects of SUL-DUR with other molecules and to define the clinical impact of SUL-DUR in combination with these molecules for the treatment of infections due to CRAB.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10096-025-05398-w.

Author contributions All authors contributed to the study conception-design and have read and agreed to the published version of the manuscript. Acquisition of data: GZ and GB, analysis and interpretation of data: RL, GB, CT and PG,; original draft preparation REL, GB, and PG,; manuscript revision and supervision: REL, GZ, GB, CT, and PG.

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Data availability All strains are available upon reasonable request.

Declarations

Competing interests The authors declare no competing interests.

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