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Clinical characteristics, antimicrobial susceptibility, and 30-day mortality among patients with Acinetobacter junii isolates: a multicenter retrospective cohort study

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Research Article

Keywords: Acinetobacter junii, antimicrobial resistance, clinical characteristics, mortality, multidrug resistance, non-baumannii Acinetobacter

Posted Date: November 20th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11440088/v1

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Additional Declarations: No competing interests reported.

Clinical characteristics, antimicrobial susceptibility, and 30-day mortality among patients with *Acinetobacter junii* isolates: a multicenter retrospective cohort study

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Abstract

Background

To describe the clinical characteristics and 30-day mortality among hospitalized patients with *Acinetobacter junii* isolates and to characterize the antimicrobial susceptibility and multidrug resistance (MDR) of these isolates.

Methods

We conducted a multicenter retrospective cohort study at three tertiary hospitals in China from 2020 to 2024. All *A. junii* isolates were identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry. We summarized clinical characteristics and antimicrobial susceptibility profiles. Multivariable logistic regression was used to identify factors associated with MDR and 30-day mortality.

Results

Among 432 patients with *A. junii* isolates, respiratory specimens accounted for 255 isolates (59.0%), and 202 patients (46.8%) were classified as having infection. Resistance exceeded 30% for cefotaxime, ceftazidime, trimethoprim-sulfamethoxazole, and gentamicin. MDR was present in 169 isolates (39.1%), and 90 isolates (20.8%) were carbapenem resistant. Male sex, clinical infection, prior antimicrobial exposure, central venous catheterization, and urine or wound, drain, or catheter specimen sources were independently associated with MDR. Forty-three patients (10.0%) died within 30 days. An Acute Physiology and Chronic Health Evaluation II score greater than 10 was strongly associated with 30-day mortality.

Conclusions

A. junii was recovered most often from respiratory specimens, and its clinical significance varied by specimen source. MDR was common, whereas 30-day mortality was associated mainly with acute illness severity.

Clinical trial number: Not applicable.

Keywords: *Acinetobacter junii*; antimicrobial resistance; clinical characteristics; mortality; multidrug resistance; non-baumannii *Acinetobacter*

Introduction

Acinetobacter species are Gram-negative bacteria found in environmental and healthcare settings. *Acinetobacter baumannii* has received the greatest attention because of its role in healthcare-associated infections and antimicrobial resistance [1]. Conventional phenotypic methods do not reliably distinguish closely related *Acinetobacter species*, and misidentification can obscure species-specific resistance patterns [2]. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) has improved species-level identification and increased recognition of non-baumannii *Acinetobacter*, including *Acinetobacter junii* [3-5].

A. junii has been recovered from humans, animals, water, soil, and hospital environments [6]. It can cause bacteremia, empyema, peritonitis, keratitis, and other invasive infections, especially in patients with recent antimicrobial exposure, malignancy, or intravascular devices [7,8]. Healthcare-associated transmission of a *blaOXA-58-carrying* strain between preterm infants has been documented [9]. Colistin-resistant isolates and strains carrying *blaOXA-58* or *blaOXA-24/40* have also been reported [10, 11], highlighting the diversity of resistance phenotypes and genetic determinants in *A. junii*.

Clinical evidence on *A. junii* remains limited. Most reports have focused on bacteremia, other sterile-site infections, outbreaks, or individual cases. Few multicenter studies

111 have evaluated the clinical significance of isolates from different specimen types
112 together with co-detected organisms, antimicrobial susceptibility, multidrug resistance
113 (MDR), and short-term outcomes. We therefore evaluated five years of *A. junii*
114 isolation at three tertiary hospitals to describe clinical characteristics and antimicrobial
115 susceptibility and to identify factors associated with MDR and 30-day mortality.
116
117 **Methods**
118 **Study design and population**
119 We conducted a multicenter retrospective cohort study at three tertiary hospitals in
120 Jiangsu, China: The First Affiliated Hospital with Nanjing Medical University (Center
121 A), The Affiliated Hospital of Xuzhou Medical University (Center B), and The Third
122 Affiliated Hospital of Soochow University (Center C). We screened microbiology
123 records for hospitalized patients from whom *A. junii* was recovered between January
124 1, 2020, and December 31, 2024. We excluded repeat isolates and records lacking the
125 required clinical or susceptibility data. Only the first isolate from each patient was
126 included.
127
128 The Ethics Committee of The First Affiliated Hospital with Nanjing Medical
129 University approved the study (2024-SR-226) and waived informed consent because
130 the analysis used retrospective, de-identified clinical and microbiologic data.
131 **Clinical data and definitions**

We collected de-identified demographic, clinical, microbiologic, treatment, and outcome data from the three centers. Variables included age, sex, body mass index, hospital department, onset category, specimen source, primary diagnostic category, clinical assessment, comorbidities, invasive devices, severity scores, laboratory measurements, prior antimicrobial exposure, co-detected organisms, length of stay, and 30-day vital status. Comorbidity was summarized with the Charlson Comorbidity Index (CCI) [12]. Acute illness severity was assessed with the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, and organ dysfunction with the Sequential Organ Failure Assessment (SOFA) score [13,14].

The index date was the date on which the index specimen was collected. An episode was classified as community-onset if it was present on admission or if the index specimen was collected within 48 hours of admission; all other episodes were classified as hospital-onset. Prior antimicrobial exposure was defined as receipt of any systemic antibacterial agent during the 30 days before the index date. Two attending physicians independently reviewed each medical record and classified the isolate as infection, colonization, or of indeterminate significance. A third attending physician resolved disagreements. The outcome was all-cause mortality within 30 days after the index date.

Specimens were grouped as respiratory, urine, sterile fluid or blood, and wound, drain, or catheter specimens. Respiratory specimens included sputum, bronchoalveolar

lavage fluid, and throat swabs. Sterile fluid or blood included blood, cerebrospinal fluid, pleural fluid, ascites, and bile. Wound, drain, or catheter specimens included skin or wound secretions, postoperative drainage, ear or conjunctival secretions, and related device material. Co-detection was defined as recovery of at least one additional organism from the index specimen.

Microbiologic identification and susceptibility testing

All laboratories identified *A. junii* to the species level using MALDI-TOF MS with the VITEK MS system (bioMérieux, Marcy-l'Étoile, France). Routine antimicrobial susceptibility testing was performed using the VITEK 2 Compact system (bioMérieux). The analytic data set contained final susceptible (S), intermediate (I), or resistant (R) interpretations for 21 agents. Available minimum inhibitory concentrations and inhibition-zone diameters were reviewed according to the *Acinetobacter* criteria and reporting rules in Clinical and Laboratory Standards Institute (CLSI) M100, 36th edition [15]. Carbapenem resistance was defined as resistance to imipenem or meropenem. MDR was defined as nonsusceptibility to at least one agent in at least three antimicrobial categories, based on the international framework for *Acinetobacter* spp. [16]. For agents with standard S, I, and R categories, intermediate and resistant results were classified as nonsusceptible. Because CLSI does not define a susceptible category for polymyxin B or colistin against *Acinetobacter* spp., only results classified as resistant were considered nonsusceptible for MDR classification [16,17].

Statistical analysis

Continuous variables were summarized as medians and interquartile ranges (IQRs), and categorical variables as counts and percentages. The infection group was compared with the colonization or indeterminate group using the Mann-Whitney U test for continuous variables and the chi-square test or Fisher exact test for categorical variables. Annual MDR and carbapenem-resistance estimates were presented with 95% Wilson confidence intervals (CIs).

Candidate factors were examined in separate univariable logistic regression models for MDR and 30-day mortality. Variables with a univariable P value < 0.20 or prespecified clinical importance were considered for multivariable analysis. When several variables represented the same clinical domain, one was selected for the multivariable model. Results are reported as adjusted odds ratios (aORs) with 95% CIs. All tests were two-sided, and $P < 0.05$ indicated statistical significance. Analyses were performed using Python version 3.11.

Results

Cohort characteristics

The microbiology database contained 505 records of *A. junii* isolation during the study period. After excluding 11 repeat isolates and 62 records that lacked required information, the final cohort comprised 432 patients and 432 index isolates: 192 from Center A, 149 from Center B, and 91 from Center C. Annual patient counts were 35,

76, 96, 114, and 111 from 2020 through 2024, respectively. Respiratory specimens were the most common source each year. Supplementary Figure S1 shows annual distributions by specimen source, center, hospital department, and diagnostic category.

The median age was 67 years (IQR, 56-76), 247 patients (57.2%) were aged 65 years or older, and 283 (65.5%) were male. Twelve patients were younger than 18 years, including four neonates. A total of 145 patients (33.6%) were in an intensive care unit at isolation, and 115 episodes (26.6%) were community-onset. An APACHE II score >10 was present in 139 patients (32.2%). Central venous and urinary catheters were present in 194 (44.9%) and 255 (59.0%) patients, respectively. Prior antimicrobial exposure was documented in 263 patients (60.9%) (Table 1).

Specimen distribution and clinical assessment

Respiratory specimens accounted for 255 isolates (59.0%), followed by urine (90, 20.8%), sterile fluid or blood (60, 13.9%), and wound, drain, or catheter specimens (27, 6.3%). Clinical assessment classified 202 patients (46.8%) as having infection and 230 (53.2%) as having colonization or indeterminate significance. Respiratory specimens contributed the largest number of infections, but only 109 of 255 respiratory isolates (42.7%) were classified as infection. By contrast, 54 of 60 isolates from sterile fluid or blood (90.0%) were classified as infection (Figure 1 and Supplementary Table S1).

Compared with the colonization or indeterminate group, the infection group had higher proportions of APACHE II scores >10 (38.1% vs 27.0%), SOFA scores ≥2 (52.3% vs 41.3%), prior antimicrobial exposure (65.8% vs 56.5%), carbapenem resistance (28.2% vs 14.3%), MDR (46.0% vs 33.0%), and community-onset episodes (31.2% vs 22.6%). Co-detection was less frequent in the infection group (13.4% vs 42.2%). Routine laboratory measurements did not differ significantly between groups (Table 1).

Co-detected organisms

Among 432 patients, 124 (28.7%) had at least one co-detected organism, yielding 134 co-isolate entries. The annual count was 11 in 2020 and 38 in 2024 (Figure 2A). *Klebsiella pneumoniae* was the most frequent named co-isolate (24 entries, 17.9%), followed by *Staphylococcus aureus* (14, 10.4%) and *A. baumannii* (10, 7.5%). Less frequent organisms accounted for 58 entries (43.3%). *K. pneumoniae* was the most common named co-isolate in respiratory specimens, whereas *S. aureus* was most common in wound, drain, or catheter specimens (Figure 2B-C and Supplementary Table S2).

Antimicrobial susceptibility

Resistance varied across the 21 agents tested. Resistance exceeded 30% for cefotaxime (41/118, 34.7%), ceftazidime (143/421, 34.0%), trimethoprim-sulfamethoxazole (131/413, 31.7%), and gentamicin (49/161, 30.4%).

Tigecycline (7/260, 2.7%), amikacin (19/426, 4.5%), and minocycline (24/357, 6.7%) had the lowest laboratory-reported resistance proportions. Carbapenem resistance was present in 90 of 432 isolates (20.8%). Polymyxin B resistance was reported in 13 of 193 tested isolates (6.7%), and colistin resistance in 10 of 54 (18.5%) (Figure 3A).

Resistance patterns varied by specimen source. Wound, drain, or catheter isolates had the highest resistance to ceftazidime (13/27, 48.1%) and trimethoprim-sulfamethoxazole (12/26, 46.2%). Among sterile fluid or blood isolates, resistance was highest to ceftriaxone (46.7%) (Figure 3B). Testing coverage exceeded 95% for cephalosporins, carbapenems, aminoglycosides, and fluoroquinolones. By antimicrobial class, resistance was highest to cephalosporins (160/431, 37.1%), followed by folate pathway inhibitors (131/413, 31.7%) and aminoglycosides (129/432, 29.9%) (Supplementary Figure S2).

Multidrug resistance

A total of 169 isolates (39.1%) met the MDR definition. The annual MDR proportion declined from 45.7% in 2020 to 32.3% in 2022 and then increased to 45.9% in 2024. Carbapenem resistance declined overall from 31.4% in 2020 to 16.2% in 2024. Neither phenotype showed a consistent upward trend (Supplementary Figure S3). MDR was most frequent among wound, drain, or catheter isolates (51.9%) and least frequent among respiratory isolates (36.9%) (Supplementary Table S1). Univariable associations are shown in Supplementary Figure S4. In multivariable analysis, MDR

was associated most strongly with wound, drain, or catheter specimens (aOR 3.79, 95% CI 1.52-9.47) and urine specimens (aOR 3.30, 95% CI 1.73-6.28), with respiratory specimens as the reference. Clinical infection (aOR 2.32, 95% CI 1.43-3.77), prior antimicrobial exposure (aOR 1.99, 95% CI 1.22-3.26), central venous catheterization (aOR 1.66, 95% CI 1.00-2.76), and male sex (aOR 1.58, 95% CI 1.01-2.48) were also independently associated with MDR (Figure 4).

Thirty-day mortality

Forty-three patients died within 30 days (10.0%). Deaths occurred in 35 patients with respiratory isolates, seven with sterile fluid or blood isolates, and one with a urine isolate; no deaths occurred in the wound, drain, or catheter group (Supplementary Table S1). Mortality was 12.4% in the infection group and 7.8% in the colonization or indeterminate group (P = 0.115). In univariable analyses, shock, an APACHE II score greater than 10, a SOFA score of at least 2, mechanical ventilation, central venous catheterization, and urinary catheterization were associated with mortality (Supplementary Figure S4). In multivariable analysis, an APACHE II score greater than 10 was strongly associated with 30-day mortality (aOR 26.11, 95% CI 8.97-75.97). MDR and co-detection were not independently associated with mortality (Figure 4).

Discussion

In this multicenter cohort of 432 patients, *A. junii* was isolated most often from respiratory specimens, but its clinical significance varied substantially by specimen source. Respiratory isolates frequently represented colonization or indeterminate significance, whereas most isolates from blood or other sterile fluids were associated with infection. MDR was common and was associated with prior antimicrobial exposure, central venous catheterization, clinical infection, and specimen source. Thirty-day mortality was associated primarily with acute illness severity.

Accurate species identification is essential for studying uncommon pathogens. Conventional microbiologic methods remain necessary for organism recovery and susceptibility testing but do not reliably distinguish closely related *Acinetobacter species* [2,5]. MALDI-TOF MS has enabled routine identification of rare bacteria that previously required additional phenotypic or molecular testing [3]. MALDI-TOF MS also improves species identification within *Acinetobacter*. For example, isolates initially classified as the *A. baumannii* complex have been reassigned to *A. junii*, *A. johnsonii*, and *A. tandoii* [5]. All three centers in our study used MALDI-TOF MS, providing a consistent approach to species identification.

Respiratory specimens were the most common source throughout the study period, consistent with hospital studies of non-baumannii *Acinetobacter* [18]. Frequent respiratory sampling in critically ill patients may partly explain this distribution. *Acinetobacter species* can also be recovered from the upper airways of adults without

307 invasive disease [19]. These factors may contribute to the predominance of respiratory
308 isolates in hospitalized patients.
309
310 Clinical significance differed most clearly between sterile and nonsterile sites. Ninety
311 percent of sterile fluid or blood isolates were classified as infection, consistent with
312 earlier *A. junii* series of invasive disease [7,8]. In contrast, only 42.7% of respiratory
313 isolates and 34.4% of urine isolates were classified as infection. Isolates from
314 nonsterile sites should therefore be interpreted in the context of clinical symptoms,
315 imaging, specimen quality, and other microbiologic findings.
316
317 Co-detection was approximately three times more common in the colonization or
318 indeterminate group than in the infection group. This pattern likely reflects the
319 predominance of nonsterile specimens, especially respiratory samples, in which
320 multiple organisms may be recovered. *K. pneumoniae* was the most frequent named
321 co-isolate overall and in respiratory specimens, whereas *S. aureus* was most frequent
322 in wound, drain, or catheter specimens. The contribution of each organism should
323 therefore be interpreted in relation to specimen source and clinical syndrome.
324
325 Our findings differ from the generally susceptible profiles described in earlier *A. junii*
326 studies. Reports of sterile-site infection and bacteremia showed broad susceptibility,
327 and more than 95% of isolates in the 2000-2010 bacteremia cohort were susceptible to
328 carbapenems, fluoroquinolones, and amikacin [7,8]. A recent case series also reported

broad susceptibility, particularly to carbapenems, aminoglycosides, and fluoroquinolones [20]. In our cohort, resistance exceeded 30% for cefotaxime, ceftazidime, trimethoprim-sulfamethoxazole, and gentamicin, and 20.8% of isolates were carbapenem resistant. Resistance to amikacin, minocycline, and tigecycline was below 10%. Colistin resistance was also observed, consistent with a previous report [10].

Several factors may explain these differences. Earlier cohorts were largely restricted to bacteremia or other sterile-site infections, whereas our cohort included a broader hospitalized population and was dominated by respiratory isolates. Prior antimicrobial exposure was also common. Laboratory methods also differed across studies. One recent series used VITEK 2 Compact with conventional biochemical tests but without MALDI-TOF MS or molecular confirmation [20]. Automated phenotypic systems can misidentify closely related *Acinetobacter species* [2,5]. Reports of *A. junii* carrying *blaOXA-58* or *blaOXA-24/40* also show that acquired carbapenem resistance occurs [9,11]. Broad susceptibility should therefore not be assumed. Treatment should be guided by susceptibility results when the isolate is clinically significant.

MDR was present in 39.1% of isolates and was associated with specimen source and several clinical factors. Earlier species-specific estimates came from studies with only a few *A. junii* isolates [21,22], which limits direct comparison. Urine and wound, drain, or catheter sources remained associated with MDR after adjustment, with respiratory

specimens as the reference. Male sex, clinical infection, central venous catheterization, and prior antimicrobial exposure were also independently associated with MDR. Antimicrobial exposure and invasive procedures are established risk factors for resistant *A. baumannii* infection [23], and earlier *A. junii* series reported frequent recent antimicrobial use and central venous access [7,8]. These factors may help identify patients at increased risk while susceptibility results are pending. Empirical therapy should be reviewed once the results are available.

Thirty-day mortality was 10.0% in our cohort, and short-term mortality was associated mainly with acute illness severity. This rate was similar to that reported in earlier sterile-site and bacteremia cohorts [7,8]. An APACHE II score greater than 10 was the only factor independently associated with mortality, whereas MDR, clinical infection, and co-detection were not. Recent studies of non-baumannii *Acinetobacter* bacteremia also identified illness severity and host factors as important predictors of short-term mortality [24,25]. These findings suggest that although antimicrobial resistance remains important for treatment selection, acute physiologic severity was the main correlate of short-term outcome in this cohort.

Neither MDR nor carbapenem resistance showed a consistent increase over the five-year study period. The year-to-year variation should be interpreted cautiously, particularly given the limited number of isolates in some years. Longer surveillance is needed to determine whether these resistance patterns change over time.

This study has several limitations. First, the retrospective design depended on the completeness and accuracy of medical records. Second, genomic analysis was not performed, so resistance mechanisms and possible transmission between patients could not be assessed. Finally, the study was limited to three tertiary hospitals in one province, and the findings may not generalize to other settings. Prospective multicenter studies with standardized clinical assessment, treatment data, susceptibility testing, and genomic analysis are needed.

In conclusion, *A. junii* was recovered most often from respiratory specimens, but fewer than half of respiratory isolates were classified as infection. Sterile fluid or blood isolates were usually clinically significant. MDR and carbapenem resistance were common. Male sex, clinical infection, prior antimicrobial exposure, central venous catheterization, and specimen source were independently associated with MDR. Thirty-day mortality was associated mainly with acute illness severity.

Abbreviations

MDR

Multidrug resistance

MALDI-TOF MS

Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry

CCI

Charlson Comorbidity Index

APACHE II

Acute illness severity was assessed with the Acute Physiology and Chronic Health Evaluation II

SOFA

Sequential Organ Failure Assessment

S

Susceptible

I

Intermediate

R

Resistant

CLSI

Clinical and Laboratory Standards Institute

IQRs

Interquartile ranges

CIs

Confidence intervals

aORs

Adjusted odds ratios

Declarations

Ethical approval and consent to participate

The Ethics Committee of The First Affiliated Hospital with Nanjing Medical University approved the study (2024-SR-226) and waived informed consent because the study used retrospective, de-identified clinical and microbiologic data. The study was conducted in accordance with the Declaration of Helsinki.

Data availability

Additional information needed to reanalyze the data reported in this study is available from the corresponding author upon reasonable request.

Declaration of competing interest

The authors declare no competing interests.

Funding

This study was funded by the Qing Lan Project for Academic Leaders in Jiangsu Colleges and Universities (2024-14), the Jiangsu Provincial Health Commission Medical Research Project General Program (M2025048), and the First Affiliated Hospital with Nanjing Medical University Clinical Capacity Enhancement Project (JSPH-MB-2024-6).

Author contributions

YZ, QQ, and WL contributed equally and share first authorship. YZ contributed to study conception, data curation, statistical analysis, investigation, software

development, visualization, and drafting the manuscript. QQ contributed to funding acquisition, methodology, and project administration. WL contributed to methodology, project administration, and validation. NZ and YL contributed to data curation. HK and CF contributed resources. WX contributed to project administration, supervision, and critical revision of the manuscript. WS contributed to funding acquisition, supervision, and critical revision of the manuscript. All authors reviewed and approved the final version of the manuscript.

Figures and legends

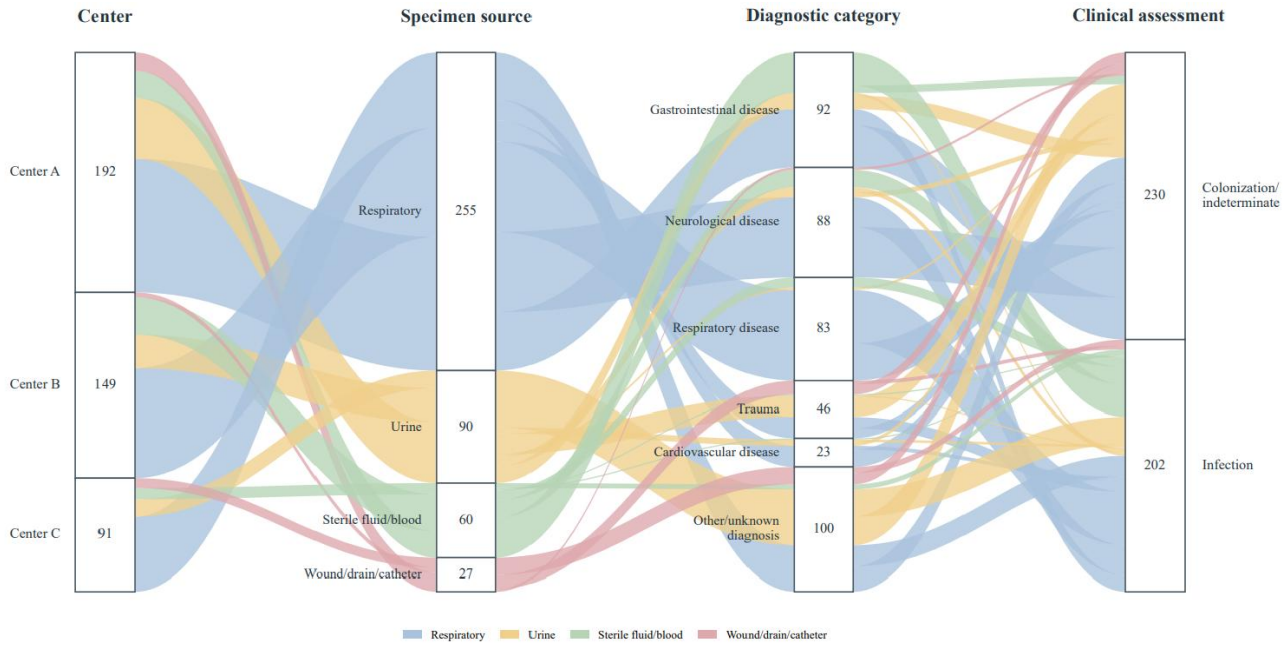


Figure 1. Sankey diagram of patient distribution. Patient distribution by center, specimen source, diagnostic category, and clinical assessment. Band width is proportional to the number of patients, and colors denote specimen source.

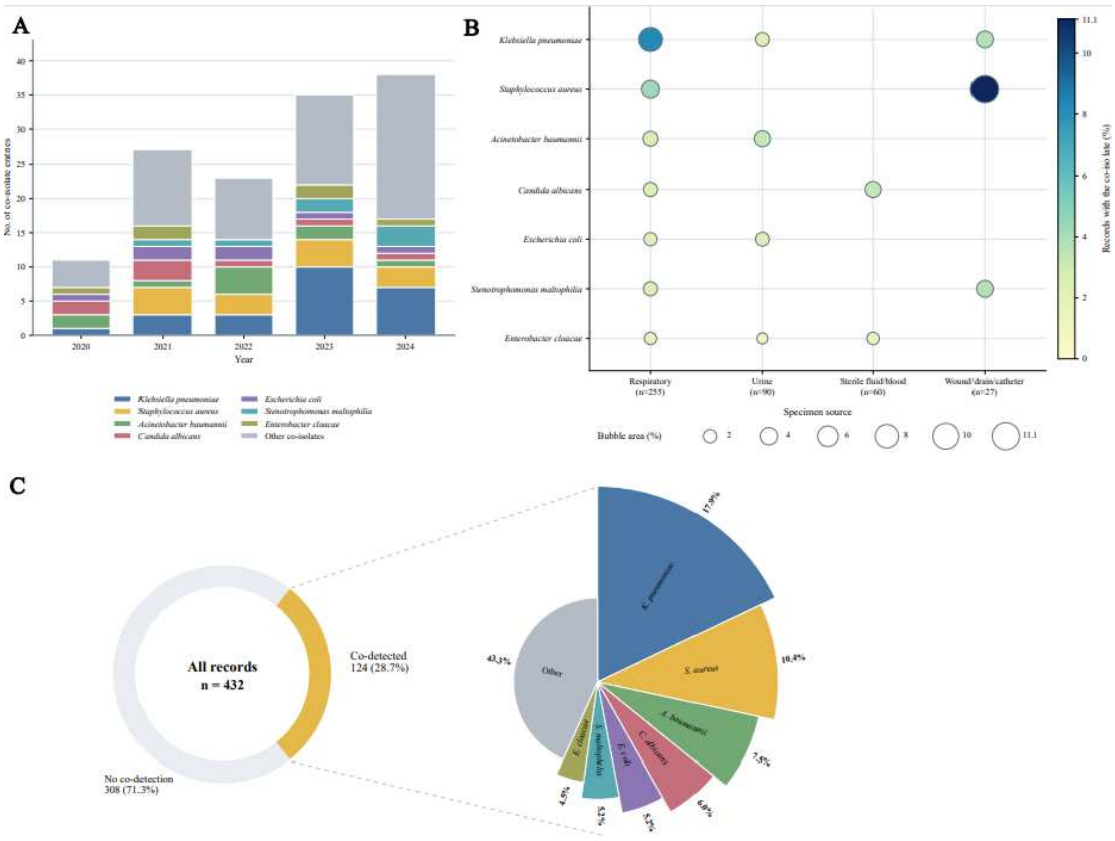
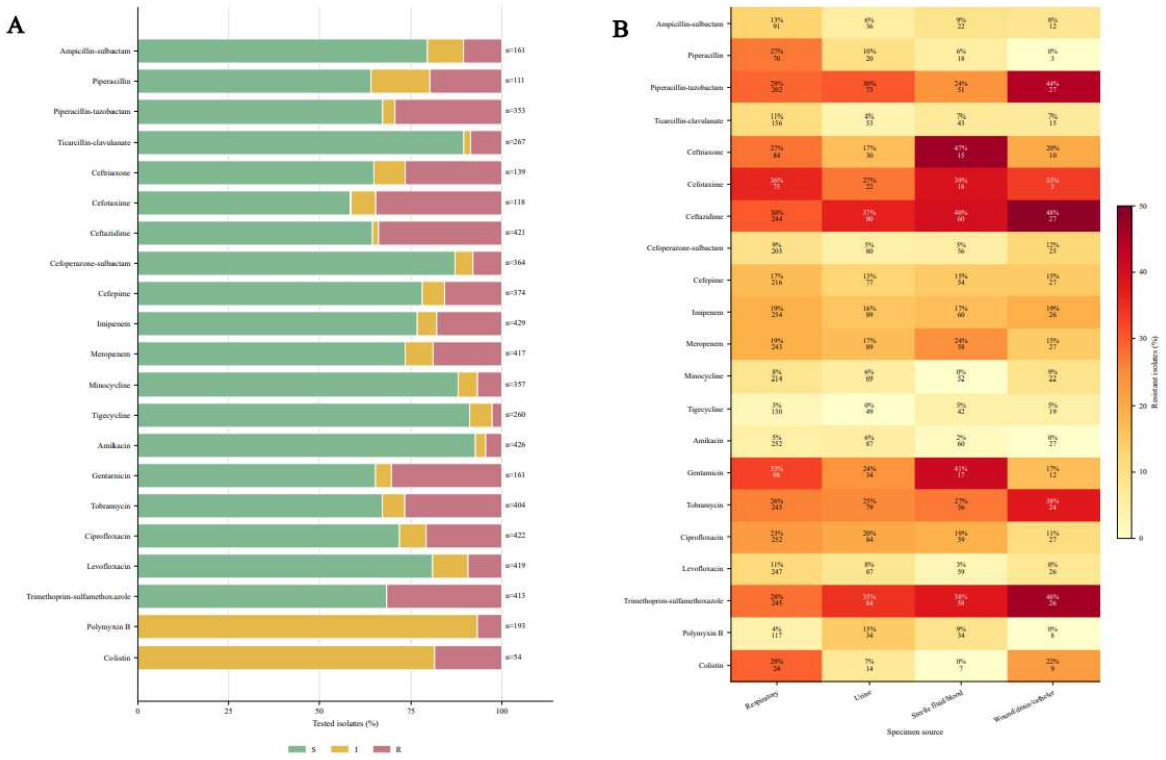


Figure 2. Co-detected organisms. (A) Number of co-isolate entries by year. (B) Percentage of records containing each of the seven most frequent named co-isolates within each specimen group; bubble area and color represent the percentage. (C) Proportion of patients with co-detection and distribution of the 134 co-isolate entries. Less frequent organisms are grouped as other co-isolates.



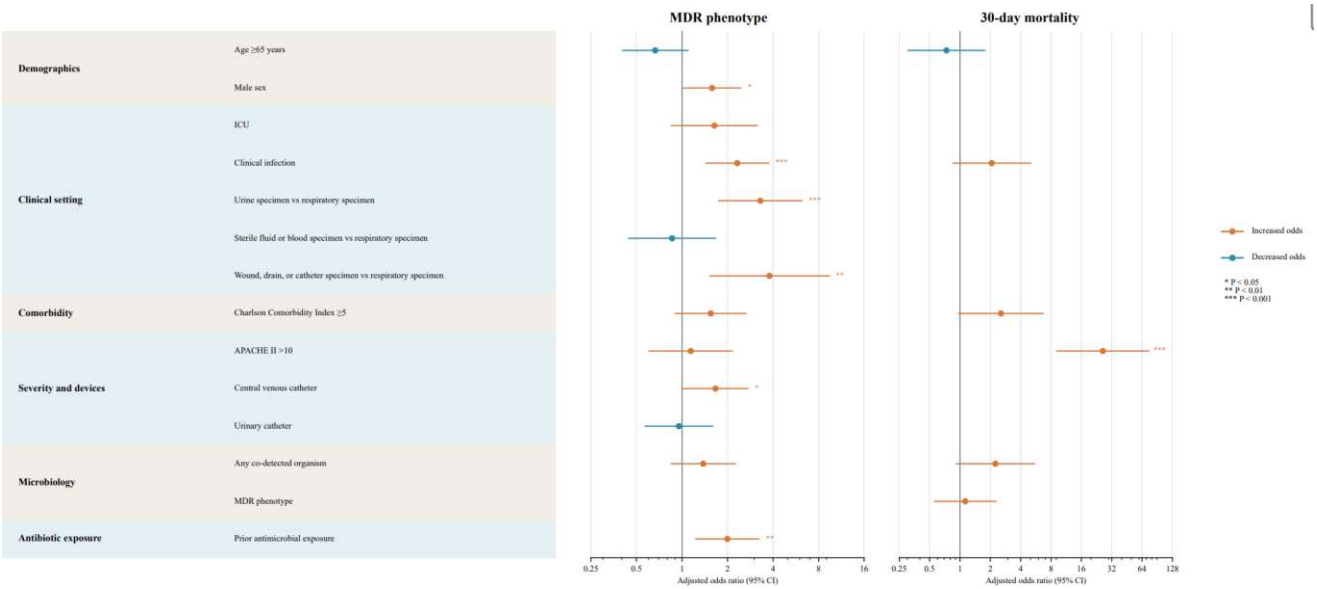
456

457 **Figure 3. Antimicrobial susceptibility of *Acinetobacter junii* isolates.** (A)

458 Laboratory-reported susceptible (S), intermediate (I), and resistant (R) categories for 21

459 agents; labels show the number tested. (B) Resistance by specimen source; cells show

460 the percentage resistant and number tested.



461

Figure 4. Multivariable associations with MDR and 30-day mortality. Points and horizontal lines show adjusted odds ratios and 95% confidence intervals. Respiratory specimens are the reference category for specimen-source comparisons. Asterisks indicate $P < 0.05$ (*), $P < 0.01$ (**), and $P < 0.001$ (***). MDR, multidrug resistance; ICU, intensive care unit; APACHE II, Acute Physiology and Chronic Health Evaluation II; CI, confidence interval.

Table

Table 1. Patient Characteristics by Clinical Assessment.

Domain	Characteristic	Overall (n=432)	Infection (n=202)	Colonization or indeterminate (n=230)	*P* value
Demographics	Age, years	67 (56-76)	67 (57-76)	67 (56-75)	0.967
	Age ≥65 years	247 (57.2%)	117 (57.9%)	130 (56.5%)	0.769
	Male sex	283 (65.5%)	124 (61.4%)	159 (69.1%)	0.091
	BMI, kg/m²	23.1 (20.8-25.9)	23.2 (20.7-25.9)	23.1 (20.8-25.8)	0.799
Clinical setting	Length of stay, days	16 (10-26)	16 (10-24)	15 (9-27)	0.471
	ICU	145 (33.6%)	72 (35.6%)	73 (31.7%)	0.391
Site of acquisition	Community-onset	115 (26.6%)	63 (31.2%)	52 (22.6%)	0.044
	Hospital-onset	317 (73.4%)	139 (68.8%)	178 (77.4%)	
Comorbidities	CCI	5 (4-7)	6 (4-7)	5 (3-7)	0.351
	CCI ≥5	278 (64.4%)	134 (66.3%)	144 (62.6%)	0.420
	Malignancy	135 (31.2%)	63 (31.2%)	72 (31.3%)	0.979
	Neurologic comorbidity	128 (29.6%)	67 (33.2%)	61 (26.5%)	0.131
	Chronic kidney disease	24 (5.6%)	15 (7.4%)	9 (3.9%)	0.112

	Diabetes mellitus	90 (20.8%)	44 (21.8%)	46 (20.0%)	0.649
	Chronic pulmonary disease	51 (11.8%)	27 (13.4%)	24 (10.4%)	0.346
Severity and invasive support	Shock	28 (6.5%)	16 (7.9%)	12 (5.2%)	0.255
	APACHE II score	8 (5-14)	8 (6-15)	7 (5-11.8)	0.005
	APACHE II >10	139 (32.2%)	77 (38.1%)	62 (27.0%)	0.013
	SOFA score	1 (0-4)	2 (0-5)	1 (0-4)	0.004
	SOFA ≥2	199 (46.4%)	104 (52.3%)	95 (41.3%)	0.023
	Mechanical ventilation	106 (24.5%)	53 (26.2%)	53 (23.0%)	0.441
	Central venous catheter	194 (44.9%)	92 (45.5%)	102 (44.3%)	0.803
	Urinary catheter	255 (59.0%)	112 (55.4%)	143 (62.2%)	0.156
Microbiology, exposure, and outcomes	Any co-detected organism	124 (28.7%)	27 (13.4%)	97 (42.2%)	<0.001
	Prior antimicrobial exposure within 30 days	263 (60.9%)	133 (65.8%)	130 (56.5%)	0.048
	Carbapenem-resistant phenotype	90 (20.8%)	57 (28.2%)	33 (14.3%)	<0.001
	MDR phenotype	169 (39.1%)	93 (46.0%)	76 (33.0%)	0.006
	30-day mortality	43 (10.0%)	25 (12.4%)	18 (7.8%)	0.115
Laboratory measurements	WBC, $\times 10^9$/L	8.5 (6.3-11.4)	8.3 (6.1-10.9)	8.6 (6.4-11.6)	0.321
	Hb, g/L	107.5 (95-125)	105 (94-123)	111.5 (96-128)	0.169
	PLT, $\times 10^9$/L	203 (145.8-267)	215 (143.5-267)	196.5 (148.2-264.8)	0.518
	ALT, U/L	22 (14-39.7)	24 (15-40)	20.9 (13.4-39.4)	0.248
	ALP, U/L	79 (63-107)	80 (64-108)	78 (61-107)	0.480
	ALB, g/L	34.5 (31.1-38)	34.2 (30.5-37.4)	35 (31.4-38.4)	0.098
	TBIL, μmol/L	11.2 (7.6-17)	10.2 (7.3-15.7)	11.9 (7.8-18)	0.134

BUN, mmol/L	6 (4.6-8.8)	6 (4.5-9.3)	6 (4.7-8.4)	0.601
SCr, μmol/L	64 (49-83.8)	63.9 (48-85.4)	64.3 (51-83)	0.811
FIB, g/L	3.7 (2.7-5.1)	3.8 (2.5-5.2)	3.6 (2.8-5)	0.920
D-dimer, mg/L	1.8 (0.6-3.5)	1.5 (0.7-3.7)	1.9 (0.6-3.2)	0.846
PCT, ng/mL	0.2 (0.1-0.5)	0.2 (0.1-0.5)	0.2 (0.1-0.6)	0.869
hs-CRP, mg/L	34.2 (9.3-84.4)	36.8 (9.1-86.6)	32.1 (10.7-82)	0.621

Continuous variables are reported as median (interquartile range), and categorical variables as n (%). BMI, body mass index; ICU, intensive care unit; CCI, Charlson Comorbidity Index; APACHE II, Acute Physiology and Chronic Health Evaluation II; SOFA, Sequential Organ Failure Assessment; MDR, multidrug resistance; WBC, white blood cell count; Hb, hemoglobin; PLT, platelet count; ALT, alanine aminotransferase; ALP, alkaline phosphatase; ALB, albumin; TBIL, total bilirubin; BUN, blood urea nitrogen; SCr, serum creatinine; FIB, fibrinogen; PCT, procalcitonin; hs-CRP, high-sensitivity C-reactive protein.

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