Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Antimicrobial Susceptibility Profiles and Associated Clinical Risk Factors of Stenotrophomonas maltophilia Isolated from Blood Cultures: A Retrospective Study

Rong Sun
Nanjing Drum Tower Hospital
Yandan Liu
Department of Clinical Laboratory, Tianjin Academy of Traditional Chinese Medicine Affiliated Hospital, Tianjin, PR China
Chang Liu
Nanjing Drum Tower Hospital
Xiaoli Cao
cao-xiao-li@163.com

Nanjing Drum Tower Hospital
Qianjin Sun
Department of Clinical Laboratory, Xuzhou Hospital of Traditional Chinese Medicine, Xuzhou, Jiangsu, PR China.
Shuo Gao
Nanjing Drum Tower Hospital

Research Article

Keywords: Stenotrophomonas maltophilia, blood culture isolates, antimicrobial susceptibility, cefiderocol, aztreonam-avibactam, risk factors, pulmonary infection

Posted Date: August 10th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10280429/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Purpose

Stenotrophomonas maltophilia is intrinsically resistant to multiple antimicrobial agents, resulting in limited therapeutic options when it is recovered from blood cultures. This study aimed to evaluate the antimicrobial susceptibility profiles of *S. maltophilia* bloodstream isolates, with particular attention to cefiderocol and aztreonam-avibactam, and to identify clinical factors associated with poor prognosis, ICU admission, and pulmonary infection among patients with S. maltophilia-positive blood cultures.

Patients and Methods:

This retrospective study included 90 *S. maltophilia* isolates recovered from blood cultures at Nanjing Drum Tower Hospital, China, between January 2012 and December 2025. Antimicrobial susceptibility testing of the 90 isolates was performed using the broth microdilution method. linical data were available for 86 corresponding patients and were collected from electronic medical records. Patients were stratified according to prognosis, ICU admission, and the presence of pulmonary infection. Univariate and multivariate logistic regression analyses were performed to identify independent associated factors.

Results

Among the 90 isolates, aztreonam-avibactam showed favorable *in vitro* activity, with MIC_{50}/MIC_{90} values of 8/8 μg/mL, and 94.4% of isolates were inhibited at concentrations of ≤ 8 μg/mL. Cefiderocol also demonstrated strong activity, with MIC_{50}/MIC_{90} values of 0.25/1 μg/mL, and 93.3% of isolates were inhibited at concentrations of ≤ 1 μg/mL. Among conventional agents, trimethoprim-sulfamethoxazole showed the highest susceptibility rate (98.9%), followed by levofloxacin (90.0%) and minocycline (45.6%). Complete baseline data were available for 86 patients, of whom 44 (51.2%) had a poor prognosis. Multivariate analysis showed that pulmonary infection was independently associated with poor prognosis (OR = 4.800; 95% CI: 1.645–14.002; P = 0.004) and ICU admission (OR = 4.004; 95% CI: 1.391–11.756; P = 0.010). Male sex (OR = 3.369; 95% CI: 1.038–10.935; P = 0.043) and antifungal agent use (OR = 3.043; 95% CI: 1.004–9.225; P = 0.049) were independently associated with pulmonary infection.

Conclusion

Cefiderocol and aztreonam-avibactam exhibited promising *in vitro* activity against *S. maltophilia* blood culture isolates, while trimethoprim-sulfamethoxazole remained the most active conventional agent. Pulmonary infection was strongly associated with poor prognosis and ICU admission, suggesting that

respiratory involvement may represent an important clinical warning sign among patients with S. maltophilia-positive blood cultures.

Introduction

Stenotrophomonas maltophilia is a non-fermenting Gram-negative bacillus that is increasingly recognized as an opportunistic pathogen associated with healthcare-associated infections[1]. Although it is widely distributed in water and other natural environments, *S. maltophilia* can also persist in healthcare settings, including on medical devices and hospital surfaces and in dialysis fluids and other moist reservoirs[2]. In patients with severe underlying disease, impaired immunity, exposure to invasive medical devices, or prolonged antimicrobial therapy, colonization by *S. maltophilia* may precede to clinically significant infection[3]. Its intrinsic resistance to multiple antimicrobial classes, together with its ability to acquire additional resistance mechanisms, has made this organism an increasing concern in clinical practice.

Data from the 2023 CHINET National Bacterial Resistance Surveillance Program showed that *S. maltophilia* accounted for 11.6% of non-fermenting Gram-negative bacilli, ranking after *Pseudomonas aeruginosa* and the *Acinetobacter baumannii* complex[4]. The reported global incidence density of *S. maltophilia* infection is approximately 4.2 cases per 10,000 hospitalization-days and is substantially higher in intensive care units (ICUs), reaching 12.7 cases per 10,000 hospitalization-days[5]. Clinically significant infections predominantly occur in critically ill or immunocompromised patients and are frequently associated with unfavorable outcomes[6]. Geze et al. reported mortality rates ranging from 29.6% to 65.1% among patients with *S. maltophilia* infection [7]. Previous studies have identified ICU admission, septic shock, mechanical ventilation, indwelling central venous catheters, neutropenia, malignancy, and inappropriate antimicrobial therapy as potential factors associated with increased mortality[7, 8].

The treatment of *S. maltophilia* infection remains challenging because of its intrinsic resistance to many commonly used antimicrobial agents, including several β-lactams, cephalosporins, and carbapenems[9]. Trimethoprim-sulfamethoxazole (TMP-SMX) remains one of the principal therapeutic options, whereas minocycline (MIN) and levofloxacin (LVX) are frequently used as alternative agents [5]. However, TMP-SMX may be poorly tolerated, particularly in patients at risk of hepatic dysfunction, renal impairment, or bone marrow suppression[10, 11]. In addition, emerging resistance to established agents has further narrowed the available therapeutic options[9]. Newer agents, including cefiderocol (FDC) and aztreonam-avibactam (ATM-AVI), have attracted increasing attention because of their potential activity against multidrug-resistant Gram-negative organisms[12]. Nevertheless, data regarding their *in vitro* activity against S. maltophilia, particularly isolates recovered from blood cultures, remain limited.

Recovery of S. maltophilia from blood cultures is clinically important because it may indicate invasive infection and is generally less likely to represent colonization than isolation from nonsterile sites, although interpretation requires careful clinical correlation. Given the potential association between S. maltophilia-positive blood cultures and adverse clinical outcomes, as well as the uncertainty surrounding

optimal antimicrobial therapy, further microbiological and clinical data are needed to inform clinical decision-making. In this retrospective study, we evaluated 90 S. maltophilia isolates recovered from blood cultures at our hospital and analyzed the corresponding clinical data available for 86 patients. We first characterized the antimicrobial susceptibility profiles of these blood culture isolates, with particular emphasis on ATM-AVI and FDC, and subsequently investigated clinical factors associated with poor prognosis, ICU admission, and pulmonary infection among the corresponding patients.

Materials and Methods

Study Design, Setting, and Isolate Collection

This retrospective study comprised two related components: a microbiological analysis of 90 *S. maltophilia* isolates recovered from blood cultures and a clinical analysis of 86 corresponding hospitalized patients with available clinical data. The isolates were collected at Nanjing Drum Tower Hospital, a tertiary Grade A hospital in Jiangsu Province, China, between January 2012 and December 2025.

Eligible isolates were identified through the hospital microbiology laboratory database on the basis of positive blood culture results for *S. maltophilia*. The corresponding electronic medical records were independently reviewed by two infectious disease specialists to verify patient eligibility and collect relevant clinical information. Any disagreement was resolved through consultation with a third senior clinician. Clinical analyses were conducted at the patient level. All collected data were anonymized and independently checked to ensure accuracy and consistency.

This study was approved by the Ethics Committee of Nanjing Drum Tower Hospital (approval No. 2023 – 390). The requirement for informed consent was waived because of the retrospective nature of the study and the use of anonymized clinical data. The study was conducted in accordance with the Declaration of Helsinki.

Antimicrobial susceptibility testing

All *S. maltophilia* bloodstream isolates were identified using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS). The minimum inhibitory concentrations (MICs) of TMP-SMX, LVX, MIN, FDC, and ATM-AVI were determined using the broth microdilution method.

Susceptibility categories were interpreted according to the 2025 edition of the Clinical and Laboratory Standards Institute (CLSI) M100 guidelines where species-specific interpretive criteria were available[13]. For antimicrobial agents without established CLSI breakpoints for *S. maltophilia*, MIC distributions, MIC_{50} and MIC_{90} values, and the proportions of isolates with MICs at selected thresholds were reported without assigning susceptibility categories. *P. aeruginosa* ATCC 27853 was used as the quality-control strain.

Clinical Data Collection

Clinical data for the 86 corresponding patients were extracted from electronic medical records using a standardized case report form. The collected variables included demographic characteristics, underlying diseases, clinical manifestations, pulmonary infection, previous hospitalization, invasive procedures, antimicrobial exposure, ICU admission, and clinical outcomes.

Patients were categorized according to prognosis, ICU admission, and the presence or absence of pulmonary infection. Logistic regression analyses were performed to identify clinical factors associated with poor prognosis, ICU admission, and pulmonary infection among patients with S. maltophilia-positive blood cultures.

Definitions

An *S. maltophilia*-positive blood culture was defined as the recovery of *S. maltophilia* from at least one blood culture specimen during hospitalization[14]. Because microbiological isolation from blood alone does not necessarily establish the clinical diagnosis of bloodstream infection in every case, clinical analyses were based on patients with *S. maltophilia*-positive blood cultures rather than on a uniformly confirmed bloodstream infection cohort.

Pulmonary infection was diagnosed on the basis of compatible clinical manifestations, pulmonary imaging findings, microbiological evidence, and the assessment of the treating physicians[15]. ICU admission was defined as admission to an intensive care unit during the hospitalization in which the S. maltophilia-positive blood culture was obtained[16]. Poor prognosis was defined according to the predefined clinical outcome criteria of this study and should include the specific outcome used in the final analysis, such as in-hospital death, 28-day mortality, or clinical deterioration[17].

Statistical analysis

Categorical variables were summarized as numbers and percentages and compared using the chi-square test or Fisher's exact test, as appropriate. Normally distributed continuous variables were expressed as means with standard deviations and compared using Student's t test. Non-normally distributed continuous variables were expressed as medians with interquartile ranges and compared using the Mann–Whitney U test. Univariate logistic regression analyses were initially performed to identify variables potentially associated with poor prognosis, ICU admission, and pulmonary infection. Variables with a P value of < 0.10 in the univariate analyses were entered into the corresponding multivariate logistic regression models. Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were calculated to identify independently associated factors. All tests were two-sided, and a P value of < 0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA).

Results

In Vitro Antimicrobial Susceptibility of S. maltophilia Blood Culture Isolates

A total of 90 isolates of *S.maltophilia* were tested for *in vitro* antimicrobial activity against five antibacterial agents. The results showed that the $MIC_{50/90}$ of ATM-AVI were both 8 μg/mL, and 94.4% of isolates were inhibited at concentrations ≤ 8 μg/mL. The $MIC_{50/90}$ of FDC was 0.25/1 μg/mL, with an inhibition rate of 93.3% at concentrations ≤ 1 μg/mL. For LVX, the $MIC_{50/90}$ was 0.125/2 μg/mL; the susceptibility rate was 90.0%, while 1.1% of isolates showed intermediate phenotype and 8.9% were resistant. The $MIC_{50/90}$ of minocycline was 2/4 μg/mL, with a susceptibility rate of 45.6%, intermediate rate of 42.2% and resistance rate of 12.2%. TMP-SMX showed an MIC_{50}/MIC_{90} of 0.125/1 μg/mL, accompanied by a susceptibility rate of 98.9% and a resistance rate of 1.1% (Fig. 1, Fig. 2 and Fig. 3).

Clinical Characteristics of the Corresponding Patients

Clinical data were available for 86 patients corresponding to the 90 *S. maltophilia* blood culture isolates. These 86 patients were included in the analyses of demographic characteristics, clinical features, and associated factors. Among these patients, 50 (58.1%) were aged 60 years or older, and 57 (66.3%) were male. Thirty-two patients (37.2%) had a hospital stay of ≥ 30 days, and 44 patients (51.2%) were classified as having a poor prognosis. Hypertension was themost common underlying disease, observed in 39 patients (45.3%), followed bymalignancy in 36 patients (41.9%). Pulmonary infection was present in 36 patients (41.9%). Surgical intervention was the most frequently recorded invasive procedure, occurring in 65 patients (75.6%), followed by indwelling urinary catheterization in 46 patients (53.5%) and endotracheal intubation in 45 patients (52.3%). Antimicrobial exposure was common in this cohort. Carbapenems were the most frequently used antimicrobial agents, administered to 66 patients (76.7%), followed by cephalosporins in 64 patients (74.4%), quinolones and glycopeptides in 40 patients each (46.5%), and antifungal agents in 25 patients (29.1%). The baseline characteristics of the patients are summarized in Table 1 and Fig. 4.

Table 1
Univariate and multivariate logistic regression analyses of risk factors associated with poor prognosis

	Total	Group with favorable prognosis	Group with poor prognosis	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 42)	(n = 44)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Patient demography							
Gender Male	57 (66.3%)	26 (61.9%)	31 (70.5%)	0.402	1.467 (0.598–3.604)		
Age ≥ 60 years	50 (58.1%)	27 (64.3%)	23 (52.3%)	0.259	0.608 (0.256–1.445)		
Admission to Intensive care unit (ICU)	33 (38.4%)	8 (19.0%)	25 (56.8%)	< 0.001	5.592 (2.111–14.813)	0.163	2.745 (0.664–11.348)
Prior hospitalization	31 (36.0%)	13 (31.0%)	18 (40.9%)	0.336	1.544 (0.635–3.754)		
Underlying diseases and acquired infections							
Hypertension	39 (45.3%)	19 (45.2%)	20 (45.5%)	0.984	1.009 (0.431–2.359)		
Diabetes mellitus	26 (31.3%)	13 (31.0%)	13 (29.5%)	0.887	0.935 (0.373–2.349)		
Cardiovascular and cerebrovascular diseases	34 (39.5%)	13 (31.0%)	21 (47.7%)	0.112	2.037 (0.843–4.920)		
Malignancy	36 (41.9%)	17 (40.5%)	19 (43.2%)	0.799	1.118 (0.474–2.635)		
Autoimmune disorder	6 (6.9%)	2 (4.8%)	4 (9.1%)	0.431	2.000 (0.346–11.544)		
Pulmonary infection	36 (41.9%)	9 (21.4%)	27 (61.4%)	< 0.001	5.824 (2.242–15.129)	0.004	4.800 (1.645–14.002)
Abdominal infection	8 (9.3%)	4 (9.5%)	4 (9.1%)	0.945	0.950 (0.222–4.071)		

	Total	Group with favorable prognosis	Group with poor prognosis	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 42)	(n = 44)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Urinary tract infection	6 (6.9%)	4 (9.5%)	2 (4.5%)	0.365	0.452 (0.078–2.612)		
Infection of biliary tract	3 (3.5%)	2 (4.8%)	1 (2.3%)	0.529	0.465 (0.041–5.330)		
Abnormalities in Organ Function and Immune Nutrition							
Multifunctional organ insufficiency	23 (26.7%)	6 (14.3%)	17 (38.6%)	0.110	3.778 (1.314–10.861)		
Hypoproteinemia	36 (39.5%)	14 (33.3%)	22 (50.0%)	0.117	2.000 (0.836–4.785)		
Hospitalization							
Hospitalization duration (≥ 30 days)	32 (37.2%)	17 (40.5%)	15 (34.1%)	0.540	0.761 (0.317–1.827)		
Surgery	65 (75.6%)	31 (73.8%)	34 (77.3%)	0.709	1.206 (0.451–3.231)		
Trachea cannula	45 (52.3%)	15 (35.7%)	30 (68.3%)	0.003	3.857 (1.576–9.439)	0.999	1.001 (0.243–4.120)
Indwelling urinary catheter	46 (53.5%)	29 (69.0%)	17 (38.6%)	0.286	1.589 (0.677–3.729)		
Hormonotherapy	61 (70.9%)	18 (42.9%)	43 (97.7%)	0.006	3.964 (1.440-10.917)	0.081	3.181 (0.868–11.659)
Blood Purification Therapy	26 (30.2%)	9 (21.4%)	17 (38.6%)	0.082	2.309 (0.889–5.998)	0.576	1.396 (0.434–4.487)
History of blood transfusion	23 (26.7%)	8 (19.0%)	15 (34.1%)	0.115	2.198 (0.816–5.921)		
Transplantation of organ	5 (5.8%)	4 (9.5%)	1 (2.3%)	0.151	0.221 (0.024–2.064)		

	Total	Group with favorable prognosis	Group with poor prognosis	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 42)	(n = 44)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Antimicrobial drug exposure							
Carbopenems	66 (76.7%)	29 (69.0%)	37 (84.1%)	0.099	2.369 (0.838-6.700)	0.835	1.158 (0.291–4.609)
Fluoroquinolones	40 (46.5%)	20 (47.6%)	20 (45.5%)	0.841	0.917 (0.393–2.140)		
Cephalosporins	64 (74.4%)	30 (71.4%)	34 (77.3%)	0.535	1.360 (0.514–3.596)		
Glycopeptides	40 (46.5%)	19 (45.2%)	21 (47.7%)	0.817	1.105 (0.473–2.581)		
Antifungal agents	25 (29.1%)	10 (23.8%)	15 (34.1%)	0.294	1.655 (0.643–4.258)		

Factors Associated with Poor Prognosis

Risk factor analysis was performed to identify variables associated with poor prognosis in patients with *S. maltophilia*-positive blood cultures. In the univariate analysis, ICU admission, pulmonary infection, endotracheal intubation, corticosteroid therapy, blood purification therapy, and carbapenem use were identified as potential factors associated with poor prognosis. These variables were subsequently included in the multivariate logistic regression model to adjust for potential confounding effects.

Multivariate analysis showed that pulmonary infection was the only independent factor associated with poor prognosis in patients with *S. maltophilia* -positive blood cultures (OR = 4.800; 95% CI: 1.645–14.002; P = 0.004). This finding suggests that patients with concomitant pulmonary infection had a significantly increased risk of poor clinical outcomes. The results are shown in Table 1.

Factors Associated with ICU Admission

To further explore clinical factors related to ICU admission, patients were stratified according to whether they were admitted to the ICU during the course of *S. maltophilia* -positive blood cultures. In the univariate analysis, pulmonary infection, corticosteroid therapy, blood purification therapy, carbapenem use, and antifungal agent use were identified as potential factors associated with ICU admission. These

variables were then entered into the multivariate logistic regression model. After adjustment for potential confounders, pulmonary infection remained independently associated with ICU admission in patients with *S. maltophilia*-positive blood cultures (OR = 4.004; 95% CI: 1.391–11.756; P = 0.010). This result indicates that patients with concomitant pulmonary infection were more likely to require ICU care. The results are shown in Table 2.

Table 2
Univariate and multivariate logistic regression analyses of risk factors associated with ICU admission

	Total	Non-ICU group	ICU group	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 53)	(n = 33)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Patient demography							
Gender Male	57 (66.3%)	33 (62.3%)	24 (72.7%)	0.318	1.616 (0.627–4.163)		
Age ≥ 60 years	50 (58.1%)	31 (58.5%)	19 (57.6%)	0.933	0.963 (0.399–2.323)		
Prior hospitalization	31 (36.0%)	19 (35.8%)	12 (36.4%)	0.961	1.023 (0.414–2.527)		
Underlying diseases and acquired infections							
Hypertension	39 (45.3%)	25 (47.2%)	14 (42.4%)	0.667	0.825 (0.344–1.982)		
Diabetes mellitus	26 (30.2%)	15 (28.3%)	11 (33.3%)	0.621	1.267 (0.495–3.238)		
Malignancy	36 (41.9%)	22 (41.5%)	14 (42.4%)	0.933	1.038 (0.430–2.504)		
Cardiovascular and cerebrovascular diseases	34 (39.5%)	19 (35.8%)	15 (45.5%)	0.376	1.491 (0.615–3.616)		
Autoimmune disorder	6 (7.0%)	3 (5.7%)	3 (9.1%)	0.544	1.667 (0316-8.793)		
Urinary tract infection	6 (7.0%)	5 (9.4%)	1 (3.0%)	0.257	0.300 (0.033–2.689)		
Pulmonary infection	36 (41.9%)	15 (28.3%)	21 (63.6%)	0.001	4.433 (1.754–11.208)	0.010	4.004 (1.391–11.756)
Abdominal infection	8 (9.3%)	4 (7.5%)	4 (12.1%)	0.478	1.690 (0.392–7.276)		

	Total	Non-ICU group	ICU group	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 53)	(n = 33)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Infection of biliary tract	3 (3.5%)	2 (3.8%)	1 (3.0%)	0.855	0.797 (0.069–9.150)		
Abnormalities in Organ Function and Immune Nutrition							
Multifunctional organ insufficiency	23 (26.7%)	11 (20.8%)	12 (36.4%)	0.112	2.182 (0.826–5.764)		
Hypoproteinemia	36 (41.9%)	20 (37.7%)	16 (48.5%)	0.326	1.553 (0.644–3.744)		
Hospitalization							
Hospitalization duration (≥ 30 days)	32 (37.2%)	18 (34.0%)	14 (42.4%)	0.430	1.433 (0.586–3.504)		
Surgery	65 (75.6%)	37 (69.8%)	28 (84.8%)	0.114	2.422 (0.792–7.405)		
Hormonotherapy	61 (70.9%)	32 (60.4%)	29 (87.9%)	0.006	4.758 (1.460-15.506)	0.079	3.379 (0.869–13.134)
Blood Purification Therapy	26 (30.2%)	11 (20.8%)	15 (45.5%)	0.015	3.182 (1.226–8.261)	0.105	2.502 (0.826–7.577)
History of blood transfusion	23 (26.7%)	11 (20.8%)	12 (36.4%)	0.112	2.182 (0.826–5.764)		
Transplantation of organ	5 (5.8%)	3 (5.7%)	2 (6.1%)	0.939	1.075 (0.170–6.801)		
Antimicrobial drug exposure							
Cephalosporins	64 (74.4%)	38 (71.7%)	26 (78.8%)	0.464	1.466 (0.525–4.093)		
Carbopenems	66 (76.7%)	36 (67.9%)	30 (90.9%)	0.014	4.722 (1.262–17.669)	0.180	2.912 (0.611–13.889)

	Total	Non-ICU group	ICU group	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 53)	(n = 33)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Fluoroquinolones	40 (46.5%)	23 (43.4%)	17 (51.5%)	0.463	1.386 (0.579–3.316)		
Glycopeptides	40 (46.5%)	23 (43.4%)	17 (51.5%)	0.463	1.386 (0.579–3.316)		
Antifungal agents	25 (29.1%)	10 (18.9%)	15 (45.5%)	0.008	3.583 (1.357–9.463)	0.096	2.596 (0.845–7.968)

Factors Associated with Pulmonary Infection

Risk factor analysis was also performed to identify variables associated with pulmonary infection among patients with *S. maltophilia* -positive blood cultures. In the univariate analysis, male sex, cardiovascular and cerebrovascular diseases, multiple organ dysfunction syndrome, endotracheal intubation, history of blood transfusion, and antifungal agent use were identified as potential factors associated with pulmonary infection. Further multivariate logistic regression analysis showed that male sex (OR = 3.369; 95% CI: 1.038–10.935; P = 0.043) and antifungal agent use (OR = 3.043; 95% CI: 1.004–9.225; P = 0.049) were independently associated with pulmonary infection in patients with *S. maltophilia* -positive blood cultures. The results are shown in Table 3.

Table 3
Univariate and multivariate logistic regression analyses of risk factors associated with pulmonary infection

	Total	Non-pulmonary infection group	Pulmonary infection group	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 50)	(n = 36)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Patient demography							
Gender Male	57 (66.3%)	29 (58.0%)	28 (77.8%)	0.056	2.534 (0.965–6.658)	0.043	3.369 (1.038–10.935)
Age ≥ 60 years	50 (58.1%)	31 (62.0%)	19 (52.8%)	0.392	0.685 (0.287–1.633)		
Prior hospitalization	31 (36.0%)	19 (38.0%)	12 (33.3%)	0.657	0.816 (0.332–2.002)		
Underlying diseases and acquired infections							
Hypertension	39 (45.3%)	22 (44.0%)	17 (47.2%)	0.767	1.139 (0.482–2.692)		
Diabetes mellitus	26 (30.2%)	12 (24.0%)	14 (38.9%)	0.138	2.015 (0.793–5.122)		
Malignancy	36 (41.9%)	22 (44.0%)	14 (38.9%)	0.636	0.810 (0.339–1.938)		
Cardiovascular and cerebrovascular diseases	34 (39.5%)	14 (28.0%)	20 (55.6%)	0.010	3.214 (1.304–7.920)	0.108	2.520 (0.817–7.771)
Autoimmune disorder	6 (7.0%)	3 (6.0%)	3 (8.3%)	0.675	1.424 (0.271–7.498)		
Urinary tract infection	6 (7.0%)	5 (10.0%)	1 (2.8%)	0.195	0.257 (0.029–2.302)		
Abdominal infection	8 (9.3%)	6 (12.0%)	2 (5.6%)	0.310	0.431 (0.082–2.273)		

	Total	Non-pulmonary infection group	Pulmonary infection group	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 50)	(n = 36)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Infection of biliary tract	3 (3.5%)	2 (4.0%)	1 (2.8%)	0.761	0.686 (0.060–7.865)		
Abnormalities in Organ Function and Immune Nutrition							
Multifunctional organ insufficiency	23 (26.7%)	8 (16.0%)	15 (41.7%)	0.008	3.750 (1.372–10.248)	0.174	2.400 (0.680–8.478)
Hypoproteinemia	36 (41.9%)	19 (38.0%)	17 (47.2%)	0.392	1.460 (0.613–3.479)		
Hospitalization							
Hospitalization duration (≥ 30 days)	32 (37.2%)	15 (30.0%)	17 (47.2%)	0.103	2.088 (0.856–5.089)		
Surgery	65 (75.6%)	40 (80.0%)	25 (69.4%)	0.261	0.568 (0.211–1.531)		
Indwelling venous catheter	68 (79.1%)	39 (78.0%)	29 (80.6%)	0.774	1.168 (0.404–3.381)		
Trachea cannula	45 (52.3%)	21 (42.0%)	24 (66.7%)	0.024	2.762 (1.132–6.739)	0.414	1.431 (0.551–4.258)
Indwelling urinary catheter	46 (53.5%)	27 (54.0%)	19 (52.8%)	0.911	0.952 (0.403–2.247)		
Hormonotherapy	61 (70.9%)	34 (68.0%)	27 (75.0%)	0.481	1.412 (0.540–3.688)		
History of blood transfusion	23 (26.7%)	9 (18.0%)	14 (38.9%)	0.031	2.899 (1.083–7.760)	0.062	3.263 (0.941–11.317)
Blood Purification Therapy	26 (31.0%)	12 (24.0%)	14 (38.9%)	0.138	2.015 (0.793–5.122)		

	Total	Non-pulmonary infection group	Pulmonary infection group	Univariate analysis		Multivariate analysis	
	(n = 86)	(n = 50)	(n = 36)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Transplantation of organ	5 (5.8%)	4 (8.0%)	1 (2.8%)	0.307	0.329 (0.035–3.071)		
Antimicrobial drug exposure							
Cephalosporins	64 (74.4%)	36 (72.0%)	28 (77.8%)	0.545	1.361 (0.501–3.697)		
Carbopenems	66 (76.7%)	38 (76.0%)	28 (77.8%)	0.847	1.105 (0.399–3.062)		
Fluoroquinolones	40 (46.5%)	25 (50.0%)	15 (41.7%)	0.445	0.714 (0.301–1.695)		
Glycopeptides	40 (46.5%)	22 (44.0%)	18 (50.0%)	0.582	1.273 (0.539–3.006)		
Antifungal agents	25 (29.1%)	9 (18.0%)	16 (44.4%)	0.008	3.644 (1.373–9.672)	0.049	3.043 (1.004–9.225)

Discussion

This study combined a microbiological analysis of 90 *S. maltophilia* isolates recovered from blood cultures with a clinical analysis of 86 corresponding patients. The principal findings were twofold. First, FDC and ATM-AVI demonstrated favorable *in vitro* activity, whereas TMP-SMX remained the most active conventional agent tested. Second, among patients with *S. maltophilia*-positive blood cultures, concomitant pulmonary infection was independently associated with poor prognosis and ICU admission, while male sex and antifungal agent use were independently associated with pulmonary infection. These findings provide complementary microbiological and clinical information that may assist in antimicrobial selection and recognition of patients with potentially unfavorable clinical profiles.

The antimicrobial susceptibility findings are clinically relevant because *S. maltophilia* is intrinsically resistant to many agents commonly used to treat Gram-negative bacterial infections. This resistance is mediated by multiple mechanisms, including production of the L1 metallo-β-lactamase and L2 serine β-lactamase, multidrug efflux pumps, reduced outer-membrane permeability, and biofilm formation[18, 19].

Consequently, empirical antimicrobial selection can be difficult, particularly in patients with severe illness or limited therapeutic options. The favorable *in vitro* activity of FDC and ATM-AVI observed in the present study suggests that these newer agents warrant further investigation as potential therapeutic options[20]. However, microbiological activity alone does not establish clinical effectiveness, and these findings should be interpreted in conjunction with pharmacokinetic, pharmacodynamic, safety, and clinical outcome data[21].

TMP-SMX remains the cornerstone of treatment for *S. maltophilia* infection, and the high susceptibility rate observed in our study supports its continued clinical value[1]. However, the usefulness of TMP-SMX should not be evaluated solely by *in vitro* activity. Patients with *S. maltophilia* -positive blood cultures often have advanced age, severe underlying diseases, impaired immunity, prolonged hospitalization, invasive devices, and extensive previous antimicrobial exposure. In such patients, adverse effects associated with TMP-SMX, including renal dysfunction, hepatic injury, hyperkalemia, and bone marrow suppression, may restrict its use[10, 11, 22]. Therefore, although TMP-SMX remains an important therapeutic option when the isolate is susceptible and the patient can tolerate treatment, alternative active agents are needed for patients with contraindications, intolerance, clinically significant drug interactions, or resistant isolates.

FDC demonstrated potent *in vitro* activity against the blood culture isolates included in this study. As a siderophore cephalosporin, FDC exploits bacterial iron-transport pathways to facilitate entry into the periplasmic space[23, 24]. This mechanism may partially overcome resistance related to reduced outer-membrane permeability and some efflux systems[25]. Its relative stability against several β-lactamases may also contribute to its activity against multidrug-resistant Gram-negative organisms[20, 23, 26]. These properties are particularly relevant to *S. maltophilia*, against which most conventional β-lactams and carbapenems have limited activity[27]. Our findings therefore support FDC as a promising *in vitro* option when established agents such as TMP-SMX, LVX, or MIN cannot be used. Nevertheless, the role of FDC in patients with *S. maltophilia*-positive blood cultures remains uncertain. *In vitro* activity may not translate directly into clinical success, especially in critically ill patients with organ dysfunction, high bacterial burden, inadequate source control, or altered drug exposure.

ATM-AVI also demonstrated favorable *in vitro* activity. This finding is mechanistically plausible because the combination targets an important resistance pattern of *S. maltophilia*. Aztreonam is relatively stable against hydrolysis by metallo-β-lactamases such as L1, whereas avibactam inhibits serine β-lactamases such as L2[28]. Since *S. maltophilia* produces both L1 and L2 β-lactamases, aztreonam alone may be insufficient, but the addition of avibactam may restore activity by protecting aztreonam from L2-mediated hydrolysis[1, 29]. This provides a rational explanation for the activity of ATM-AVI against *S. maltophilia*. However, as with FDC, caution is needed when interpreting ATM-AVI results. Formal clinical breakpoints for *S. maltophilia* may be limited or unavailable for newer agents, and inhibition concentrations should not be overinterpreted as definitive clinical susceptibility. Further studies are required to determine whether the favorable *in vitro* activity of ATM-AVI can translate into improved clinical outcomes.

Among the other conventional agents, LVX retained activity against most isolates, suggesting that it may remain a useful alternative in selected patients. However, fluoroquinolone resistance can emerge during treatment, particularly in the setting of prolonged antimicrobial exposure, high bacterial burden, or selection of resistant subpopulations[30]. LVX should therefore be used on the basis of isolate-specific susceptibility results rather than assumed empirical activity[31]. MIN showed a more heterogeneous susceptibility pattern, including a relatively large proportion of isolates categorized as intermediate according to the interpretive criteria applied in this study. This pattern may be related to efflux systems, acquired tetracycline resistance determinants, isolate-specific resistance mechanisms, and changes in breakpoint interpretation[32]. These findings do not support the empirical use of MIN for severe infection without susceptibility confirmation. When MIN is considered, disease severity, infection source, achievable drug exposure, and the availability of other active agents should also be evaluated[33].

The clinical characteristics of the corresponding patient cohort reflected a population with substantial healthcare exposure and underlying vulnerability. Many patients were older adults and had malignancy, prolonged hospitalization, invasive procedures, pulmonary infection, or previous exposure to broad-spectrum antimicrobial agents. Carbapenem and cephalosporin exposure was particularly common. These findings are consistent with the opportunistic behavior of *S. maltophilia*, which is frequently recovered in patients with impaired host defenses and prolonged healthcare contact[30]. Broad-spectrum antimicrobial exposure may suppress competing microorganisms and create ecological conditions that favor colonization or infection by intrinsically resistant organisms[34]. These observations underscore the importance of antimicrobial stewardship, both to optimize treatment and to reduce the selection pressure that facilitates the emergence of resistant opportunistic pathogens[30].

Pulmonary infection was independently associated with poor prognosis in the multivariate analysis. Patients with concomitant pulmonary infection had higher odds of an unfavorable outcome than those without pulmonary infection. Respiratory involvement may reflect greater overall illness severity, impaired gas exchange, a higher burden of comorbid disease, exposure to invasive airway procedures, or increased vulnerability to secondary infection[16, 27, 35]. It may also indicate a potential respiratory source of the positive blood culture in some patients[1]. However, the present observational analysis cannot establish that pulmonary infection directly caused the unfavorable outcome. Moreover, pulmonary infection was diagnosed using clinical, imaging, microbiological, and physician-assessment criteria and did not necessarily establish *S. maltophilia* as the causative pulmonary pathogen in every patient[36]. Pulmonary infection should therefore be interpreted primarily as an important clinical marker requiring careful respiratory evaluation and close monitoring.

Pulmonary infection was also independently associated with ICU admission. This finding may indicate that patients with respiratory involvement were more likely to require intensive monitoring, respiratory support, or management of organ dysfunction[37]. Nevertheless, ICU admission is influenced by disease severity, institutional admission practices, bed availability, goals of care, and the timing of clinical deterioration[38]. In addition, the retrospective design may not fully establish whether pulmonary infection preceded ICU admission in every case. Thus, pulmonary infection should not be interpreted as

a proven causal determinant of ICU admission. Rather, its presence may help identify patients with a more complex or severe clinical course who could benefit from early assessment for respiratory compromise and other organ dysfunction[37, 39, 40].

Male sex was independently associated with pulmonary infection in this cohort. This association should be interpreted cautiously. The study did not comprehensively capture several potential confounders, including smoking history, chronic pulmonary disease, occupational exposure, aspiration risk, baseline pulmonary function, and differences in healthcare exposure. Male sex may therefore represent a surrogate marker for unmeasured respiratory or behavioral factors rather than a direct biological determinant of pulmonary infection. Additional studies incorporating detailed pulmonary and exposure-related variables are required to clarify this association. Antifungal agent use was also independently associated with pulmonary infection. A direct causal interpretation is unlikely. Antifungal agents are commonly administered to patients with prolonged hospitalization, immunosuppression, malignancy, exposure to broad-spectrum antibiotics, invasive procedures, or suspected fungal infection[41]. Antifungal use may therefore reflect underlying disease severity, diagnostic uncertainty, extensive antimicrobial pressure, or a high-risk host profile. Confounding by indication and uncertainty regarding the temporal relationship between antifungal exposure and pulmonary infection cannot be excluded. This result should consequently be regarded as an association that generates hypotheses rather than evidence that antifungal therapy itself increases the likelihood of pulmonary infection.

This study has several limitations. First, it was a single-center retrospective study, and the findings may reflect local patient characteristics, antimicrobial use patterns, and microbiological epidemiology, thereby limiting generalizability. Second, although 90 blood culture isolates were included in the microbiological analysis, complete clinical data were available for only 86 corresponding patients, which may have introduced selection bias. The inclusion of more than one isolate from some patients may also have influenced the isolate-level susceptibility distribution and should be considered when interpreting the microbiological results. Third, the retrospective design precluded causal inference, and residual confounding could not be excluded. The temporal relationships among pulmonary infection, ICU admission, antimicrobial or antifungal exposure, and clinical outcomes were not fully established. Fourth, several potentially important variables were unavailable or incompletely analyzed, including validated illness-severity scores, the timing and appropriateness of active antimicrobial therapy, source control, smoking history, chronic pulmonary disease, baseline respiratory function, and detailed organ-support requirements. Fifth, the definition of pulmonary infection did not necessarily confirm that *S. maltophilia* was the causative respiratory pathogen in every case. Sixth, the study relied on phenotypic antimicrobial susceptibility testing and did not include resistance-gene analysis, strain typing, virulence profiling, or assessment of β-lactamase and efflux-pump expression. Finally, treatment regimens were not directly linked to clinical outcomes. The present study therefore cannot determine the comparative clinical efficacy of TMP-SMX, FDC, ATM-AVI, LVX, MIN, or combination therapy. Prospective multicenter studies integrating microbiological, molecular, pharmacological, treatment, and outcome data are needed to validate these findings.

Conclusion

In conclusion, TMP-SMX remained highly active against S. maltophilia blood culture isolates, while FDC and ATM-AVI showed promising in vitro activity as potential alternative agents. Pulmonary infection was independently associated with poor prognosis and ICU admission among patients with S. maltophilia-positive blood cultures, whereas the associations of male sex and antifungal use with pulmonary infection require further validation. Multicenter studies integrating antimicrobial susceptibility testing, molecular resistance mechanisms, treatment exposures, and well-defined clinical outcomes are needed to inform individualized management strategies for patients with *S. maltophilia*-positive blood cultures.

Declarations

This study was approved by the Ethics Committee of Nanjing Drum Tower Hospital, the Affiliated Hospital of Nanjing University Medical School (approval No. 2023-390). As this was an anonymous retrospective analysis, the ethics committee waived the requirement for informed consent. All research procedures were carried out in compliance with relevant guidelines and regulations.

Author Contributions

Rong Sun,Yandan Liu and Chang Liu served as co-first authors and completed the main research work and manuscript drafting. Shuo Gao, Qianjin Sun and Xiaoli Cao acted as corresponding authors, responsible for study design, result revision andproject supervision. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests

Funding

This work was supported by the National Natural Science Foundation of China (81902124).

Data Availability

All data used in this study are described and cited within the manuscript.

References

1. Kouroupis D, Zarras C, Zarfeiadou M, Sanos C, Iosifidis E, Michailidou C et al. Epidemiological and Clinical Characteristics of Stenotrophomonas maltophilia Isolates from Hospitalized Medical Patients; An Emerging Pathogen in the Non-Critically Ill. Trop Med Infect Dis. 2025;10(9).

2. Singh P, Ahmed MN, Srivastava AK, Thakur AK, Parveen R, Puraswani M, et al. Multicenter epidemiology of Stenotrophomonas maltophilia bloodstream infections in Indian ICUs: building digital surveillance network. Front Microbiol. 2025;16:1725629.
3. Sameni F, Dadashi A, Hajikhani B, Eshkalak MP, van Belkum A, Hashemi A, et al. Insights into antibiotic resistance and prevalence of Stenotrophomonas maltophilia isolated from clinical samples in Iran: A systematic review. New Microbes New Infect. 2025;68:101664.
4. Qin X, Ding L, Hao M, Li P, Hu F, Wang M. Antimicrobial resistance of clinical bacterial isolates in China: current status and trends. JAC Antimicrob Resist. 2024;6(2):dlae052.
5. Bostanghadiri N, Sholeh M, Navidifar T, Dadgar-Zankbar L, Elahi Z, van Belkum A, et al. Global mapping of antibiotic resistance rates among clinical isolates of Stenotrophomonas maltophilia: a systematic review and meta-analysis. Ann Clin Microbiol Antimicrob. 2024;23(1):26.
6. Hafiz TA, Aldawood E, Albloshi A, Alghamdi SS, Mubaraki MA, Alyami AS et al. Stenotrophomonas maltophilia Epidemiology, Resistance Characteristics, and Clinical Outcomes: Understanding of the Recent Three Years' Trends. Microorganisms. 2022;10(12).
7. Gezer Y, Tayşi MR, Tarakçı A, Gökçe Ö, Danacı G, Altunışık Toplu S, et al. Evaluation of clinical outcomes and risk factors associated with mortality in patients with Stenotrophomonas maltophilia bloodstream infection: a multicenter study. BMC Infect Dis. 2024;24(1):1387.
8. Huang C, Lin L, Kuo S. Risk factors for mortality in Stenotrophomonas maltophilia bacteremia - a meta-analysis. Infect Dis (Lond). 2024;56(5):335–47.
9. Mojica MF, Humphries R, Lipuma JJ, Mathers AJ, Rao GG, Shelburne SA, et al. Clinical challenges treating Stenotrophomonas maltophilia infections: an update. JAC Antimicrob Resist. 2022;4(3):dlac040.
10. Sawalha K, Sobash PT, Kamoga GR. A Rare Cause of Drug-Induced Pancytopenia: Trimethoprim-Sulfamethoxazole-Induced Pancytopenia. Clin Pract. 2021;11(2):358–62.
11. Chahal J, Junko G. Trimethoprim-Sulfamethoxazole-Induced Pancytopenia: A Case Report. Cureus. 2024;16(12):e75113.
12. Dan MO, Tălăpan D. Friends or foes? Novel antimicrobials tackling MDR/XDR Gram-negative bacteria: a systematic review. Front Microbiol. 2024;15:1385475.
13. Schuetz AN, Ferrell A, Hindler JA, Humphries R, Bobenchik AM. Overview of changes in the Clinical and Laboratory Standards Institute Performance Standards for Antimicrobial Susceptibility Testing: M100 32nd and 33rd editions. J Clin Microbiol. 2025;63(9):e0162323.
14. Sezen AI, Ozdemir YE, Yeşilbağ Z, Borcak D, Canbolat Ünlü E, Bayrak Erdem F, et al. Seven-year evaluation of Stenotrophomonas maltophilia bacteremia in a university-affiliated hospital. J Infect Dev Ctries. 2025;19(4):498–503.
15. Lee YH, Lee J, Yu B, Lee WK, Choi SH, Park JE, et al. Risk factors for mortality in intensive care unit patients with Stenotrophomonas maltophilia pneumonia in South Korea. Acute Crit Care. 2023;38(4):442–51.

16. Zappulo E, Schiano Moriello N, Paolillo R, Zumbo G, Pinchera B, Catania MR et al. What Drives Mortality in S. maltophilia Bloodstream Infections? An Outcome-Focused Cohort Study of Treatment Strategies and Resistance Profiles. Microorganisms. 2025;14(1).
17. Guo W, Lin Q, Li J, Feng X, Zhen S, Mi Y, et al. Stenotrophomonas maltophilia bacteremia in adult patients with hematological diseases: clinical characteristics and risk factors for 28-day mortality. Microbiol Spectr. 2025;13(1):e0101124.
18. Brooke JS. Advances in the Microbiology of Stenotrophomonas maltophilia. Clin Microbiol Rev. 2021;34(3):e0003019.
19. Sameni F, Hajikhani B, Hashemi A, Owlia P, Niakan M, Dadashi M. The Relationship between the Biofilm Genes and Antibiotic Resistance in Stenotrophomonas maltophilia. Int J Microbiol. 2023;2023:8873948.
20. Závora J, Adámková V, Studená A, Kroneislová G. In Vitro Activity of Cefiderocol and Aztreonam/Avibactam Against Gram-Negative Non-Fermenting Bacteria: A New Strategy Against Highly Antibiotic-Resistant Infectious Agents. Antibiot (Basel). 2025;14(8).
21. Barrasa H, Morán MA, Fernández-Ciriza L, Isla A, Solinís MÁ, Canut-Blasco A et al. Optimizing Antibiotic Therapy for Stenotrophomonas maltophilia Infections in Critically Ill Patients: A Pharmacokinetic/Pharmacodynamic Approach. Antibiot (Basel). 2024;13(6).
22. Yi S, Ye M, Liu P, Chen K, Yuan Y, Li L. A Mortality Prediction Nomogram for Stenotrophomonas maltophilia Bloodstream Infection. Infect Drug Resist. 2025;18:5129–37.
23. Zhanel GG, Golden AR, Zelenitsky S, Wiebe K, Lawrence CK, Adam HJ, et al. Cefiderocol: A Siderophore Cephalosporin with Activity Against Carbapenem-Resistant and Multidrug-Resistant Gram-Negative Bacilli. Drugs. 2019;79(3):271–89.
24. Simner PJ, Patel R. Cefiderocol Antimicrobial Susceptibility Testing Considerations: the Achilles' Heel of the Trojan Horse. J Clin Microbiol. 2020;59(1).
25. Bilal M, El Tabei L, Büsker S, Krauss C, Fuhr U, Taubert M. Clinical Pharmacokinetics and Pharmacodynamics of Cefiderocol. Clin Pharmacokinet. 2021;60(12):1495–508.
26. Méndez-Sotelo BJ, Delgado-Beltrán M, Hernández-Durán M, Colín-Castro CA, Esquivel-Bautista J, Ortega-Oliva SA, et al. In vitro activity of ceftazidime/avibactam, cefiderocol, meropenem/vaborbactam and imipenem/relebactam against clinical strains of the Stenotrophomonas maltophilia complex. PLoS ONE. 2024;19(4):e0298577.
27. Bryan A, Shields-Pruiett M, Berger H, Fang FC, Penewit K, Waalkes A, et al. In vitro susceptibility of Stenotrophomonas maltophilia and other carbapenem non-susceptible Gram-negative rods in the Pacific Northwest to cefiderocol and aztreonam-avibactam. JAC Antimicrob Resist. 2026;8(3):dlag105.
28. Sader HS, Duncan LR, Arends S, Carvalhaes CG, Castanheira M. Antimicrobial Activity of Aztreonam-Avibactam and Comparator Agents When Tested against a Large Collection of Contemporary Stenotrophomonas maltophilia Isolates from Medical Centers Worldwide. Antimicrob Agents Chemother. 2020;64(11).

29. Verschelden G, Noeparast M, Stoefs A, Van Honacker E, Vandoorslaer K, Vandervore L, et al. Aztreonam-avibactam synergy, a validation and comparison of diagnostic tools. Front Microbiol. 2023;14:1322180.
30. Maraolo AE, Licciardi F, Gentile I, Saracino A, Belati A, Bavaro DF. Stenotrophomonas maltophilia Infections: A Systematic Review and Meta-Analysis of Comparative Efficacy of Available Treatments, with Critical Assessment of Novel Therapeutic Options. Antibiot (Basel). 2023;12(5).
31. Brooke JS. Stenotrophomonas maltophilia: an emerging global opportunistic pathogen. Clin Microbiol Rev. 2012;25(1):2–41.
32. Bakthavatchalam YD, Manoharan Y, Shankar A, Gunasekaran K, Walia K, Veeraraghavan B. Understanding the rationale and clinical impact of the revised CLSI 2024 minocycline susceptibility breakpoints against Stenotrophomonas maltophilia. Eur J Clin Microbiol Infect Dis. 2024;43(12):2453–7.
33. Mojica MF, Rutter JD, Taracila M, Abriata LA, Fouts DE, Papp-Wallace KM, et al. Population Structure, Molecular Epidemiology, and β-Lactamase Diversity among Stenotrophomonas maltophilia Isolates in the United States. mBio. 2019;10(4):e00405–19.
34. Bi H, Lin Q, Sun Q, Lv Y, Tian Z, Fu X, et al. Predictors of mortality and progression from colonization to Stenotrophomonas maltophilia bloodstream infection in hematologic disorders: a single-center retrospective study. Eur J Clin Microbiol Infect Dis. 2025;44(11):2609–21.
35. Signorini L, Moioli G, Calza S, Van Hauwermeiren E, Lorenzotti S, Del Fabro G, et al. Epidemiological and Clinical Characterization of Superinfections in Critically Ill Coronavirus Disease 2019 Patients. Crit Care Explor. 2021;3(6):e0430.
36. Nair AP, Sasi S, Al Maslamani M, Al-Khal A, Chacko K, Deshmukh A, et al. Clinical and Epidemiological Characteristics of Stenotrophomonas maltophilia Associated Lower Respiratory Tract Infections in Qatar: A Retrospective Study. Cureus. 2022;14(3):e23263.
37. Wang N, Tang C, Wang L. Risk Factors for Acquired Stenotrophomonas maltophilia Pneumonia in Intensive Care Unit: A Systematic Review and Meta-Analysis. Front Med (Lausanne). 2021;8:808391.
38. Carbonell N, Oltra MR, Clari MÁ. Stenotrophomonas maltophilia: The Landscape in Critically Ill Patients and Optimising Management Approaches. Antibiot (Basel). 2024;13(7).
39. Aisenberg G, Rolston KV, Dickey BF, Kontoyiannis DP, Raad II, Safdar A. Stenotrophomonas maltophilia pneumonia in cancer patients without traditional risk factors for infection, 1997–2004. Eur J Clin Microbiol Infect Dis. 2007;26(1):13–20.
40. Kunz Coyne AJ, Lucas K, Gray R, May E. Propensity-Matched Comparison of Timely vs Delayed Antibiotic Therapy in Stenotrophomonas maltophilia Pneumonia. Open Forum Infect Dis. 2025;12(8):ofaf469.
41. Pham HT, Castelino RL, Kiser TH, Truong-Nguyen KH, Tran MH. Empirical versus pre-emptive antifungal therapies for invasive fungal infections in critically ill patients. BMC Infect Dis. 2025;25(1):395.

Figures

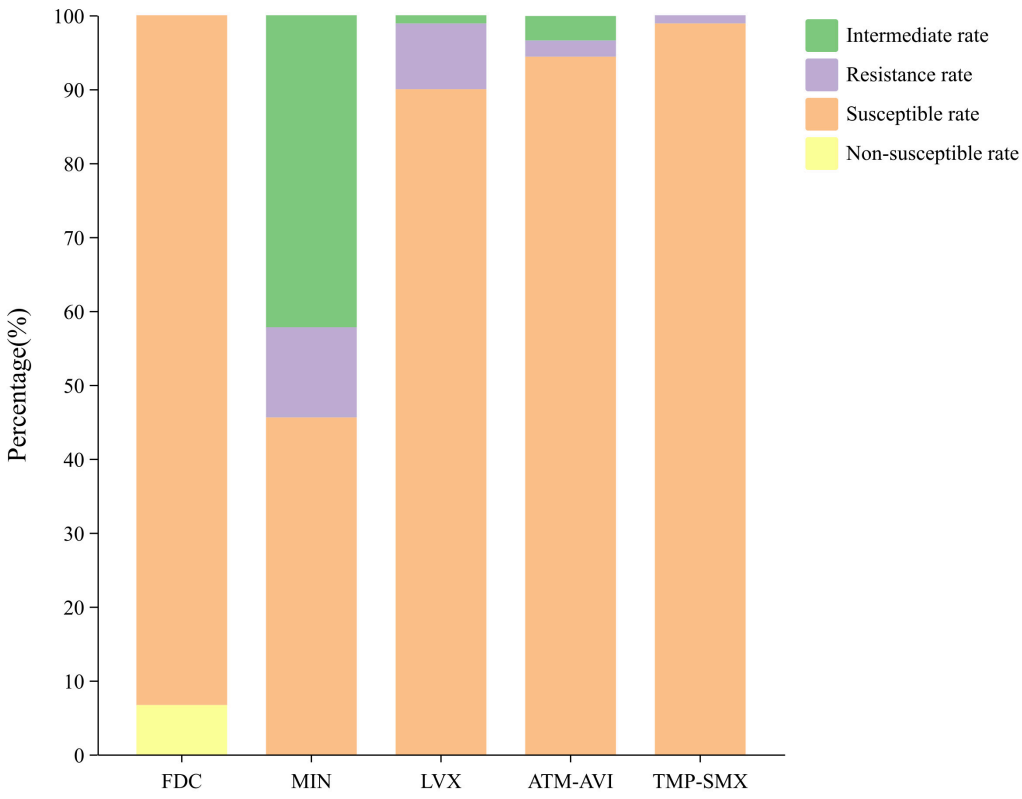


Figure 1

Distribution of predisposing factors among patients with *S. maltophilia* infection The x-axis represents the number of patients with each predisposing factor, and the y-axis lists individual predisposing items. Green bars denote underlying diseases and concomitant infections, orange bars represent prior antimicrobial exposure, and blue bars indicate invasive medical procedures.

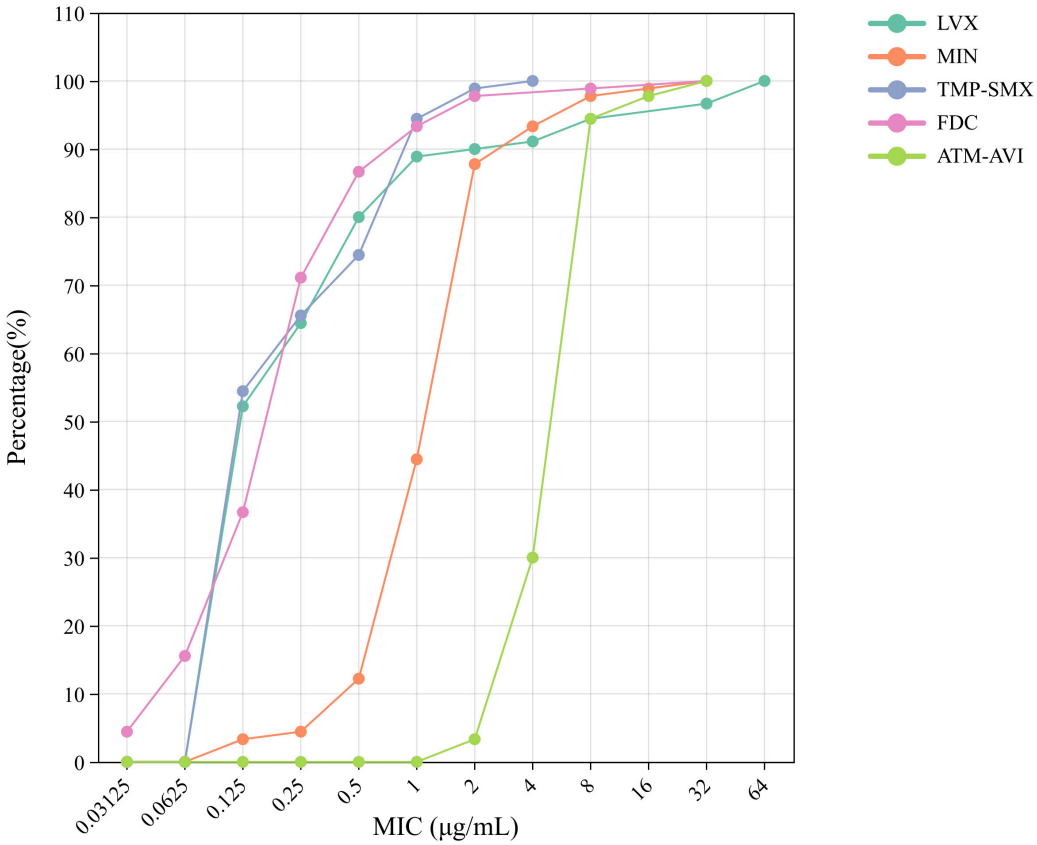


Figure 2

Susceptible, intermediate and resistant percentage of *S. maltophilia* isolates to five tested antimicrobial agents.

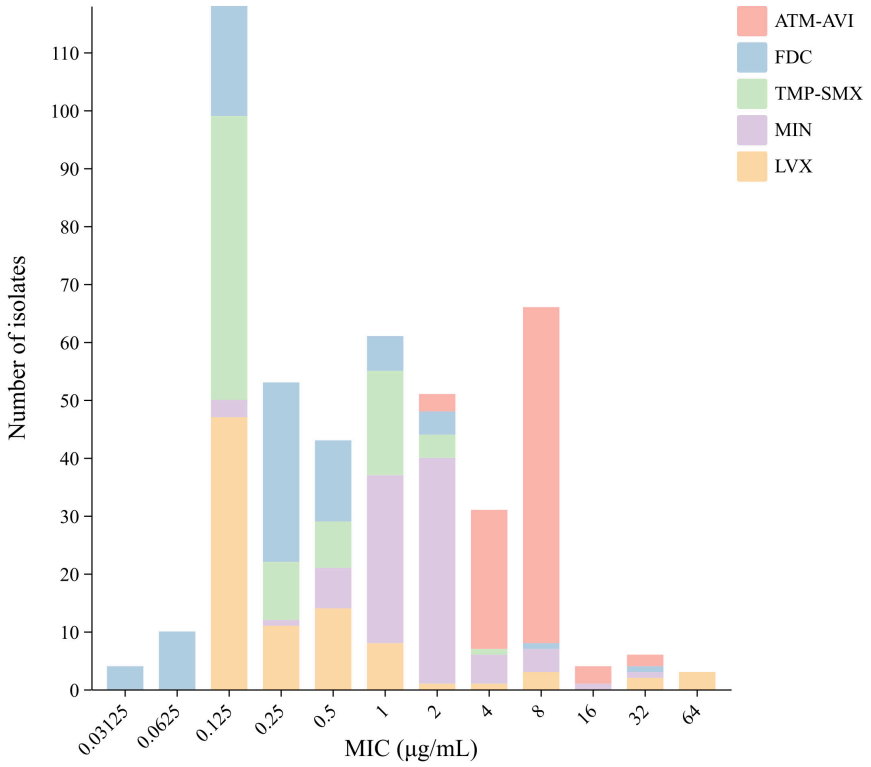


Figure 3

Cumulative MIC distribution curves of five antimicrobial agents against clinical *Stenotrophomonas maltophilia* isolates

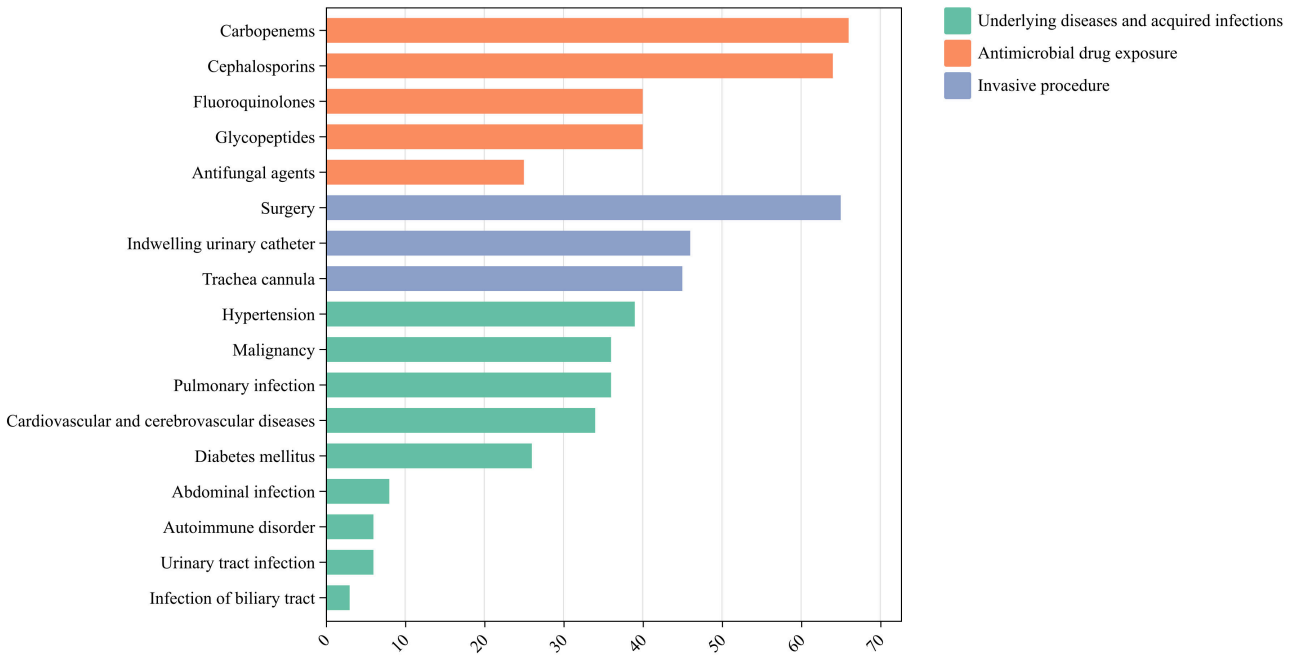


Figure 4

Distribution of clinical *Stenotrophomonas maltophilia* isolate counts across serial MIC gradients for five antimicrobial agents