Journal of Molecular Science
Journal of Molecular Science
Volume 35 Issue 3, Year of Publication 2025, Page 600-605
DoI-10.004687/1000-9035.2025.082

Journal of Molecular Science
www.jmolecularsci.com ISSN:1000-9035

Decoding the Clinical Relevance of Stenotrophomonas Maltophilia: A Silent Menace.

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Article Information
Received: 18-09-2025
Revised: 25-09-2025
Accepted: 02-10-2025
Published: 08-10-2025

Keywords
Hospital acquired pneumonia, nosocomial infection, Stenotrophomonas maltophilia, ventilator associated pneumonia.

ABSTRACT

Objectives: *Stenotrophomonas maltophilia* is an emerging multidrug-resistant pathogen causing nosocomial infections. Our study aimed to analyze the isolation rate, clinical profile, antibiotic susceptibility, and TMP-SMX resistance genes from clinical samples. **Materials and Methods:** A prospective study was conducted from March 2024 - March 2025. Clinical samples with Gram-negative, non-lactose fermenting bacilli were screened for *S. maltophilia*. It was identified using conventional biochemical tests and MALDI-TOF. Antimicrobial susceptibility was performed by VITEK 2. Clinical details were recorded directly from patient case file. 26 isolates underwent PCR for sul1 and dfrA genes. **Results:** 42 isolates (1.13%) were isolated from 3703 non-lactose fermenting colonies. The mean age was 55-years with a 76% male preponderance. Most isolates were from exudate samples (43%), respiratory samples (29%), and blood (19%). 31% percent were ICU-associated. Statistically significant risk factors were CKD, ventilator requirement and tracheostomy. Infections were monomicrobial in 48% of samples. Susceptibility was highest with levofloxacin (95%), minocycline (93%), TMP-SMX (93%), and ceftazidime-avibactam (93%). Sul1 gene was detected in 3 (11.5%) isolates and dfrA in 1 (3.8%) isolate, respectively. Among the 38 patients, 25 (65.8%) recovered, 7 (18.4%) succumbed to infection, and 4 (10.5%) left against medical advice. Mortality was noted more in monomicrobial infections (6/7 deaths). **Conclusion:** Our study suggests that with the increasing isolation of *Stenotrophomonas maltophilia* as a significant pathogen, clinicians should consider including trimethoprim-sulfamethoxazole or other anti-*Stenotrophomonas* drugs (Minocycline, Levofloxacin, Ceftazidime-avibactam) as one of the empirical antibiotic therapies for patients with risk factors such as chronic kidney disease, tracheostomy, or mechanical ventilation, rather than relying solely just on cephalosporins or meropenem as empirical drugs. Sul1 gene continues to be an important factor in developing resistance against TMP-SMX and must be closely watched for any change in trends.

INTRODUCTION:

Stenotrophomonas maltophilia has transitioned from a relatively insignificant environmental bacteria to a formidable opponent in clinical microbiology. This aerobic, non-fermenting, Gram-negative bacilli has developed increasing attention as an opportunistic pathogen globally [1,2,3]. While historically it was considered a low-virulence colonizer, its escalating frequency of isolation from clinical specimens and its undeniable association with significant morbidity and mortality, have firmly established its status as a "newly emerging pathogen of concern" [1,2,3]. The World Health Organization (WHO) has recognized *S. maltophilia* as an important, multidrug-resistant organism within hospital environments [4].

Its ubiquitous presence in natural environments, including soil, water, and plants, coupled with its remarkable ability to colonize hospital equipment and water systems, renders it a persistent and pervasive threat [1,2,3]. This environmental adaptability, combined with an expanding population of immunocompromised patients and the widespread, often indiscriminate use of broad-spectrum antibiotics, has significantly fueled its increasing prevalence and complicated its clinical management [1,2,3]. Understanding the multifaceted nature of *S. maltophilia* from its intricate pathogenic mechanisms and formidable resistance profiles is paramount for clinical microbiologists and infectious disease specialists striving to combat this challenging adversary.

MATERIALS AND METHODS:

This study was approved by the Institutional Ethics Committee and was conducted over a period of 1 year from March 2024 – 2025.

INSTITUTIONAL ETHICS COMMITTEE APPROVAL:

REF: CSP-MED/24/JAN/97/03

STUDY DESIGN: Prospective study from March 2024 – March 2025.

INCLUSION CRITERIA:

1. All clinical samples sent to Laboratory.
2. Sputum samples with pure growth of non-fermenter gram negative bacilli only.
3. Bronchial wash and Endotracheal secretions with non-lactose fermenter colony counts of $>10^5$ cfu/ml
4. Urine sample - $>10^5$ cfu/ml

EXCLUSION CRITERIA:

1. Patients diagnosed with respiratory infections prior to admission.
2. Sputum samples with polymicrobial growth in culture media.
3. Bronchial wash and ET secretions with a colony count $<10^5$ cfu/ml.

METHODOLOGY:

The samples received at the clinical microbiology laboratory, in the course of routine patient care were processed. The demographic details and laboratory parameters were obtained from the hospital IP manager and detailed history was collected during the course in the hospital.

Microscopy:

Gram stain was performed on all samples and presence of Gram-negative bacilli was noted. The significance of Gram stain was noted in relation to the cells and presence of Gram-negative bacilli.

Culture:

All the samples were cultured on to Blood agar, Chocolate agar, MacConkey agar and incubated at 37°C for 24 hours. Plates were routinely examined for growth for 2 days. Growth of non-lactose fermenting colonies on MacConkey agar was taken up for this study. Samples like urine, endotracheal secretion and bronchoalveolar lavage, were processed as per significant colony count as mentioned below.

- $\geq 10^5$ cfu/ml - significant for urine.
- $>10^5$ cfu/ml - significant for Endotracheal secretion.
- $\geq 10^4$ cfu/ml - significant for Bronchoalveolar secretion.
- Pure growth on Sputum sample was considered significant when there was <10 squamous epithelial cells on microscopy according to bartlett's scoring and confluent growth was present.
- In some samples such as urine where patients' history showed clinical significance, a lower colony count of 10^4 cfu/ml was also included as part of the study.

Conventional tests to identify *Stenotrophomonas maltophilia*:

All non-lactose fermenters which grew on MacConkey agar, were processed using conventional biochemicals such as Indole, triple sugar iron agar, Simmon's citrate, Christensen's urea agar, mannitol motility medium. Oxidase test was performed on all isolates to differentiate it from *Pseudomonas* species. All biochemical tests were performed using reference from Koneman.

Automated identification system:

All the isolates were also subjected to identification by Matrix Assisted Laser Desorption Ionization – Time of Flight after incubation of 24 hours with growth of NLF colonies. (MALDI-TOF) (bioMérieux, India).

Antibiotic susceptibility testing:

Antibiotic susceptibility testing (AST) was performed using VITEK®2 AST-GN10 card. Quality control was performed using *Pseudomonas aeruginosa* ATCC 27853.

Molecular detection of trimethoprim sulfamethoxazole antibiotic resistant genes in *Stenotrophomonas maltophilia:*

TMP-SXM resistant genes specific primers used:

1. Sul1 – Forward: GACGATTGCGGTTCTT3
2. Sul2 – Reverse: CCGATTCAGCTTTTGAAGG3
3. dfrA – Forward: CACTTGTAATGGCACGGAAA
4. dfrA – Reverse: ACCCCTTCATGGTGAAATGA

Table 1A. Master Mix preparation for PCR

Components	Volume Per reaction	Number of vials	Volume Per vials
Probe PCR Master Mix	10µl	1	250µl
Forward and Reverse Primer Probes	5µl	1	125µl
DNA Template	10µl	1	150µl
PCR grade water	-	1	4ml

Table 1B. PCR Cycling Conditions

	STEP	TIME	TEMP
	Taq enzyme activation / Hold	15min	95ºC
35 cycles	Denaturation	20sec	95ºC
	Annealing	20sec	60ºC
	Extension	20sec	72ºC

RESULTS:

A total of 36503 samples was received in the clinical microbiology laboratory; 3703 samples grew non-lactose fermenting colonies (on MacConkey agar) out of which 42 clinical isolates of *Stenotrophomonas maltophilia* were isolated.

Table 2. Clinical Sample Distribution of *Stenotrophomonas Maltophilia*

Sample Type	Number of Isolates (n)	Percentage (%)
Pus	11	26.5%
Endotracheal Secretion	11	26.5%
Blood	7	16.5%
Wound	5	12.5%
Urine	3	7%
Tissue	2	5%
Bronchial Wash	1	2%
Permanent catheter	1	2%
Central Line tip	1	2%
Total	**42**	**100%**

During the study period, **42 clinical isolates** of *Stenotrophomonas maltophilia* were isolated from various samples like respiratory secretions, blood, pus, tissue, sterile body fluids, urine, etc.

Analysis of Clinical Profile of Patients with *Stenotrophomonas Maltophilia* Infection:

Demographic details of patients:

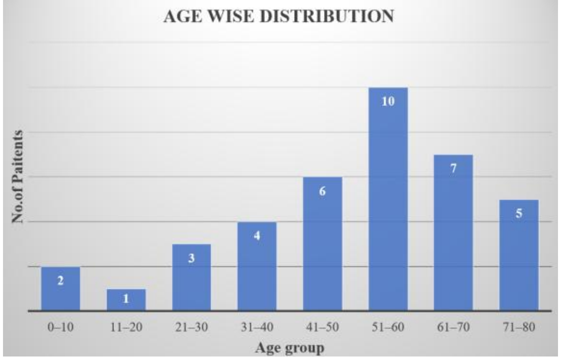

Figure 1. Age-wise distribution of patients with *Stenotrophomonas maltophilia* infection

- Among the 38 patients, the mean age of patients overall was 55 years old.
- The youngest affected patient was 7 months old child, and eldest affected patient was 84 years old.
- Mean age of ICU patients was 52.2 years old and Mean age of non-ICU patients was 57.23 years old.
- Gender-wise distribution: Males = 76% (n = 29), Females = 24% (n = 9).

Table 3. Antibiotic susceptibility results of 42 *S. maltophilia* isolates

Antibiotic	Susceptible (n, %)	Resistant (n, %)
Levofloxacin	40 (95%)	2 (5%)
Tigecycline	40 (95%)	2 (5%)
Minocycline	39 (93%)	3 (7%)
TMP-SMX	39 (93%)	3 (7%)
Ceftazidime–avibactam	39 (93%)	3 (7%)
Cefoperazone–sulbactam	32 (76%)	9 (21%)
Cefepime	29 (69%)	13 (31%)
Chloramphenicol	26 (62%)	16 (38%)
Ceftazidime	23 (55%)	19 (45%)

Table 4. Analysis of 26 isolates for TMP-SMX resistant genes.

- 3 isolates (11.5%) were positive for sul1 gene only.
- 1 isolate (3.8%) was positive for dfrA gene only.
- None had a combination of both.

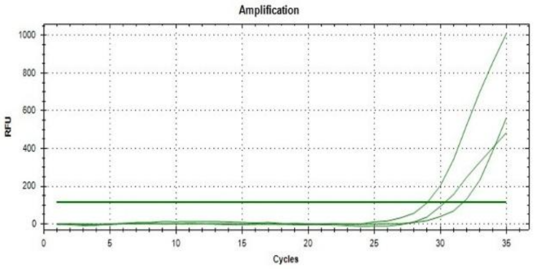

Figure 2A showing Real-Time PCR graph of sul1 gene (n = 3)

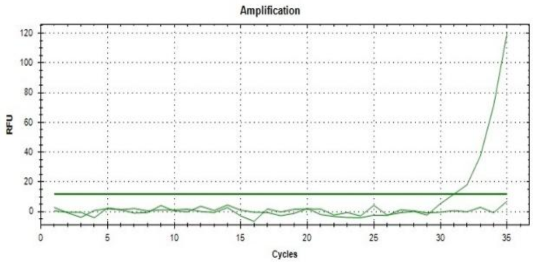

Figure 2B showing Real-Time PCR graph of dfra gene (n = 1)

Table 4. Patient comorbidities and risk factors:

Comorbidity / Risk Factor	**No. of Patients**	**Percentage (%)**
Surgical Procedures	22	52%
Diabetes Mellitus	18	48%
Hypertension	14	38%
Ventilator Use	12	32%
Trauma	12	31%
Chronic Kidney Disease	8	21%
Tracheostomy	8	19%
Coronary Artery Disease	5	14%
Chronic Lung Disease (COPD, TB, etc.)	3	8%
Myocardial Infarction	3	9.5%
Malignancy	2	5%

POLYMICROBIAL AND MONOMICROBIAL INFECTIONS.

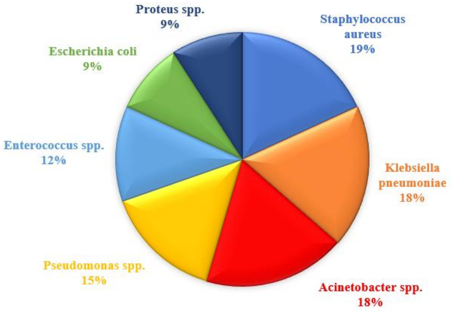

Figure 3. Polymicrobial growth of *S.maltophilia* in our study

- **Polymicrobial infections** were noted in **52%** (n = 20) of cases, with *S. maltophilia* isolated alongside other pathogens such as *Staphylococcus aureus* (19%), and *Acinetobacter* spp (18%), *Klebsiella pneumoniae (18%)*, *Pseudomonas spp.* (15%), *Enterococcus* spp (12%), *Escherichia coli* (9%) *Proteus spp* (9%).
- **Monomicrobial infections** were **48%** (n = 18).

Table 5. Chi-square test and odds ratio among polymicrobial and monomicrobial infections

Risk Factor	**Polymicrobial (n = 20)**	**Monomicrobial (n = 18)**	**OR (95% CI)**	**p-value**
ICU admission	5 / 20 (25.0%)	6 / 18 (33.3%)	**1.50 (0.37 – 6.14)**	0.836
Diabetes mellitus	9 / 20 (45.0%)	9 / 18 (50.0%)	**1.22 (0.34 – 4.38)**	1
Chronic kidney disease	0 / 20 (0.0%)	7 / 18 (38.9%)	**25.45 (1.32 – 492.17)**	**0.008**
Malignancy	1 / 20 (5.0%)	1 / 18 (5.6%)	1.12 (0.06 – 19.28)	1
Tracheostomy	1 / 20 (5.0%)	6 / 18 (33.3%)	**9.50 (1.01 – 88.97)**	**0.067**
Surgery	13 / 20 (65.0%)	7 / 18 (38.9%)	0.34 (0.09 – 1.28)	0.199
Ventilator requirement	3 / 20 (15.0%)	9 / 18 (50.0%)	**5.67 (1.22 – 26.33)**	**0.049**
Duration of stay (days)	14.2 ± 10.6	13.5 ± 12.3	–	0.9

PATIENT OUTCOMES:

Outcomes were known for most patients:

- **68% (n = 25)** recovered and were discharged.
- **18% (n = 7)** died during hospitalization.
- **17% (n = 4)** left the hospital against medical advice (AMA).
- **4% (n = 2)** were Out Patients and we were unable to follow up.
- Overall, there was 18% mortality rate (n = 7).

DISCUSSION:

Over the study period, we **isolated 42 *S. maltophilia* strains** from 3,703 Non-Lactose Fermenter culture growths (1.13%). Similar percentage of isolation rate at 1.6% was found in a South-Indian study by Jacob A et al (2021) [5] but higher rates of isolation at 3.9% was found in a study by Srivastava et al (2023) [6]. The most frequent sources were from exudate samples (48%), followed by respiratory specimens (28.5%), bloodstream infections (17%), and others 7% (urine, central line tip, permanent catheter tip) (table 2). Unlike out study, Varshini et al (2022) had isolated *S. maltophilia* more from blood (44%), followed by respiratory samples (32%) and least in exudates (24%) [7]. Said MS et al (2023) have mentioned *S. maltophilia's* capacity to cause wound/soft tissue infections at 7.8% respectively [8]. About 31% of our patients had a history of trauma and 52% underwent surgical procedures (e.g. wound debridement, shunt insertion in pediatric case, ORIF Implants etc.) due to which we had a higher isolation rate from exudate samples. *S. maltophilia* infection was seen in all age groups with the youngest person in our study a 7-month-old child and the oldest was 84 years old but the mean age of infected patients was 55 years, with a male preponderance (76% male) (Figure 1). This aligns with other reports that *S. maltophilia* infections are more common in males and patients in extremes of ages [3,6,7].

A challenge with *S. maltophilia* is its notorious drug resistance profile. It is intrinsically resistant to many broad-spectrum antibiotics, including most β-lactams and aminoglycosides [1,9]. A North Indian analysis by Singh R (2024) et al found TMP-SMX and levofloxacin to have a susceptibility of 84% with even higher susceptibility to minocycline at 94.8% [10]. Similarly in our study, the highest susceptibility of more than 90% was seen with TMP-SMX, Ceftazidime-avibactam, Minocycline and Levofloxacin reflecting their continued efficacy. Cefoperazone-sulbactam was 76% susceptible and cefepime was 69% susceptible. Chloramphenicol had only moderate activity with 62% susceptibility and only 55% of isolates were susceptible to ceftazidime. (table 3). Another study by Banar M et al (2023) also presented similar trends as ours [11].
26 isolates were analysed for the detection of TMP-SMX resistant genes **sul1** and **dfrA** by Real-time PCR. We chose to analyse samples in which multi-drug resistance in *S.maltophilia* was reported (n = 26). Results showed three isolates to have sul1 genes and one isolate to have dfrA gene. None had a combination of sul1 and dfrA together. The three isolates found with sul1 gene were from ET secretions and one dfrA gene was from Bronchial wash but the VITEK 2 AST report of the bronchial wash showed TMP-SMX to be susceptible. Some studies have showed the presence of sul1 + dfrA combination or efflux pump is necessary for acquiring resistance to TMP-SMX [12,13] but in our study, analysis showed strains with sul1 gene in combination with other resistant mechanisms such as efflux pumps were more commonly found and proves that the presence of dfrA gene alone is unable to contribute to the acquired resistance of TMP-SMX by *S.maltophilia*

In many of our cases, initial empirical antibiotics were ineffective against *S. maltophilia.* For example, some patients in our study were started on piperacillin-tazobactam or carbapenems empirically before culture results, the drugs to which *S. maltophilia* typically exhibits resistance. Once *S. maltophilia* was identified, therapy was adjusted in most cases to cover the organism. Antibiotic therapy for **9/38** patients was switched from 3rd generation cephalosporins and meropenem to TMP-SMX reflecting its status as the drug of choice for *S. maltophilia* infections. In cases with resistance to TMP-SMX, fluoroquinolones like levofloxacin were used as alternatives, and a few patients received minocycline as well. All 7 deceased patients in our study had received multiple antibiotics like 3rd generation cephalosporins and meropenem instead of recommended anti-Stenotrophomonas antibiotics.
In General, the majority of our patients had underlying illnesses or predisposing conditions (table 4). Approximately 62% had at least one co-morbidity, and 38% had multiple comorbidities. The majority had a history of surgery, followed by Type 2 Diabetes Mellitus, hypertension, chronic kidney disease (CKD), and chronic heart diseases. We also documented 13% of the patients (n = 5) had pre-existing chronic lung diseases like COPD and old pulmonary tuberculosis. This profile mirrors the risk factors reported in several studies such as Jacob A et al and Varshini et al [5,7]. Several other studies such as Singh R et al, Gupta et al and Sumida et al noted that diabetes, renal impairment, and malignancy were common in *S. maltophilia* infected patients, similar to our study [3,10,14]. These chronic diseases likely impair the immune defenses and create a situation that predisposes patients to opportunistic infections.

Well-known hospital-based risk factors include prolonged hospitalization, ICU care, invasive devices (central lines, endotracheal tubes, urinary catheters, hemodialysis water), recent surgeries, and prior broad-spectrum antibiotic use [1,2,3]. It has been reported as the third most common non-fermenting gram-negative bacillus in healthcare associated infections, after *Pseudomonas aeruginosa* and *Acinetobacter* spp [11,15]. 20 out of 38 (52%) patients in our study had prolonged hospital stays of more than 10 days which exposes them a range of nosocomial infections. About 41% (n = 5 out of 12) of the ICU patients required mechanical ventilation with a **median of 10 days on ventilator**. In our study, risk factors such as CKD (p-value = 0.008), patients requiring active ventilator support (0.049) and with tracheostomy (0.06) were statistically significant (table 5) and **odds ratio** had strongest associations with CKD, Tracheostomy, and active ventilator requirement.

It is difficult to ascertain if *S. maltophilia* isolates are pathogens especially in cases with associated underlying comorbid conditions. The pathogenicity of *S. maltophilia* can be ruled-in based on monomicrobial growth in specimens indicating true pathogenicity. A notable finding in our study is the rate of **monomicrobial infection** at 48% (n = 12) and polymicrobial infection at 52% (n = 20) (Figure 3). Jacob A *et al* [5] had a slightly lesser range of polymicrobial findings of 38.7%, this maybe because of our inclusion of more exudative samples. Amongst the monomicrobial infections, highest isolation rate was observed in ET samples (44%), followed by blood samples (22%). Literatures have mentioned Co-pathogens like *Pseudomonas* *aeruginosa, Klebsiella pneumoniae* or *Acinetobacter* can contribute to severity of sepsis and complicate therapy [5,7]. But in our study, patients who succumbed (6/7 deaths) mostly had monomicrobial infection (except one with concurrent *Chrysobacterium*). Four monomicrobial isolates were from ET samples, two from Blood and one from bronchial wash.

The mortality rate in our study was 18% (n = 7). A meta-analysis of 19 studies with 1248 patients reported approximately 40% mortality in patients with Bacteremia [11], and a recent Chinese study on *S. maltophilia* bacteremia by Jian J *et al* found a 37.3% mortality rate [19]. In contrast, Varshini et al [7] an ICU-focused study noted only 6% mortality, suggesting that outcomes can vary widely depending on antibiotic policies and severity of underlying diseases in the patients. Despite most patients being very ill, 25 patients (65%) recovered and were discharged, 7 patients (19%) died in hospital, 4 patients (10%) took discharge against medical advice (AMA) and 2 were Out Patients (5%).

CONCLUSION:

Our study suggests that with the increasing isolation of *Stenotrophomonas maltophilia* as a significant pathogen, clinicians should consider including trimethoprim-sulfamethoxazole or other anti-*Stenotrophomonas* drugs (Minocycline, Levofloxacin, Ceftazidime-avibactam) as one of the empirical antibiotic therapies for patients with risk factors such as chronic kidney disease, tracheostomy, or mechanical ventilation, rather than relying solely just on cephalosporins or meropenem as empirical drugs. Sul1 gene continues to be an important factor in developing resistance against TMP-SMX and must be closely watched for any change in trends.

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