

PERSPECTIVE ARTICLE

Implications of accurate pseudomonad identification for patient care, antimicrobial stewardship, and outbreak investigations

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Abstract

In low-resource clinical microbiology laboratories, the identification of non-fermenting, oxidase-positive Gram-negative bacilli remains a significant challenge. Due to limited diagnostic capabilities, there is a pervasive tendency to report all such organisms simply as *Pseudomonas* species, often implicitly considered *Pseudomonas aeruginosa*. This practice overlooks the vast diversity within the *Pseudomonadaceae* family, which includes numerous genera such as *Burkholderia*, *Stenotrophomonas*, *Achromobacter*, *Ochrobactrum*, *Ralstonia*, and many non-aeruginosa *Pseudomonas* spp. This misidentification poses serious clinical consequences, as intrinsic antibiotic resistance profiles vary dramatically across these species, leading to inappropriate therapy and treatment failure. Furthermore, it curtails effective epidemiological surveillance and source investigation during outbreaks. This paper delineates the scope of this problem, its impact on patient care and public health, and proposes a multifaceted, feasible way forward for laboratories constrained by resources. With this, the author calls for the adoption of stratified, algorithmic approaches using basic biochemical tests and enhanced training, as well as the strategic development of affordable, context-specific diagnostic solutions.

Keywords: Pseudomonads; Gram-negative bacilli; Antimicrobial resistance; Outbreak investigation; Diagnostic stewardship; Antimicrobial stewardship; Clinical microbiology; Low-resource settings

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1. Introduction

The isolation of non-fermenting Gram-negative bacilli (NFGNB) from a clinical specimen often triggers an immediate and presumptive identification of *Pseudomonas aeruginosa* in many microbiology laboratories across low-resource settings. This heuristic, born out of necessity and diagnostic limitation, is a dangerous oversimplification. The term “pseudomonad” encompasses a phylogenetically broad group of organisms that, while sharing some common microbiological characteristics, are remarkably diverse in their pathogenicity, ecological niches, and, most critically, their antimicrobial resistance profiles.¹ The routine conflation of these organisms under the blanket term “*Pseudomonas* species” is not a mere academic oversight; it is a significant clinical and public health failure with tangible consequences for patient outcomes and infection control. This perspective aims to illuminate the profound implications of this misidentification and

chart a pragmatic course for improvement within the constraints of low-resource settings.

2. The taxonomic and clinical spectrum of pseudomonads

The family *Pseudomonadaceae* and related families include a plethora of genera relevant to human medicine. While *P. aeruginosa* is indeed a pre-eminent pathogen, it is merely the most prominent member of a large and varied group.¹ Clinically significant organisms frequently misidentified as *P. aeruginosa* or mistakenly lumped under “*Pseudomonas* spp” include, but are not limited to, members of the *Burkholderia cepacia* complex, *Stenotrophomonas maltophilia*, *Achromobacter xylosoxidans*, *Ralstonia pickettii*, *Ochrobactrum anthropi*, *Chryseobacterium* spp., and a growing list of non-aeruginosa *Pseudomonas* spp. such as *P. putida*, *P. fluorescens*, and *P. stutzeri*. Each of these organisms possesses a unique characteristic different from other organisms in the group. For instance, *S. maltophilia* is intrinsically resistant to carbapenems and possesses inducible resistance mechanisms, whereas *B. cepacia* complex exhibits variable but often broad-spectrum resistance.^{2,3} The ecological habitats also differ vastly, thus necessitating accurate identification. For instance, *B. pseudomallei* is a soil saprophyte in endemic regions, *S. maltophilia* is often associated with water sources and plants, and *P. aeruginosa* is ubiquitous in moist

environments.⁴⁻⁶ Figure 1 presents the negative clinical and public health implications of misidentifying NFGNB.

3. Clinical implications of misidentification

The most immediate and detrimental impact of incorrect identification is the selection of inappropriate antimicrobial therapy. The intrinsic resistance patterns of these bacteria are highly variable; therefore, first-line therapy for one is often entirely ineffective for another. For example, *P. aeruginosa* is typically susceptible to anti-pseudomonal penicillins, cephalosporins such as ceftazidime and cefepime, carbapenems such as imipenem and meropenem, and aminoglycosides such as amikacin and tobramycin.⁷ In sharp contrast, *S. maltophilia* is intrinsically resistant to most beta-lactams, including carbapenems, and is typically treated with trimethoprim-sulfamethoxazole or alternatively with tigecycline.⁸ Similarly, many *Burkholderia* spp. exhibits resistance to aminoglycosides and polymyxins.³ Therefore, a physician receiving a report of “*Pseudomonas* spp.” from a blood culture might empirically prescribe meropenem in cases of serious infection, a rational choice for *P. aeruginosa*. However, treatment failure is imminent if, in fact, the isolate is *S. maltophilia*, risking the patient’s life and contributing to unnecessary carbapenem selective pressure. This scenario, while hypothetical, represents a daily reality in settings where differentiation is not performed, directly

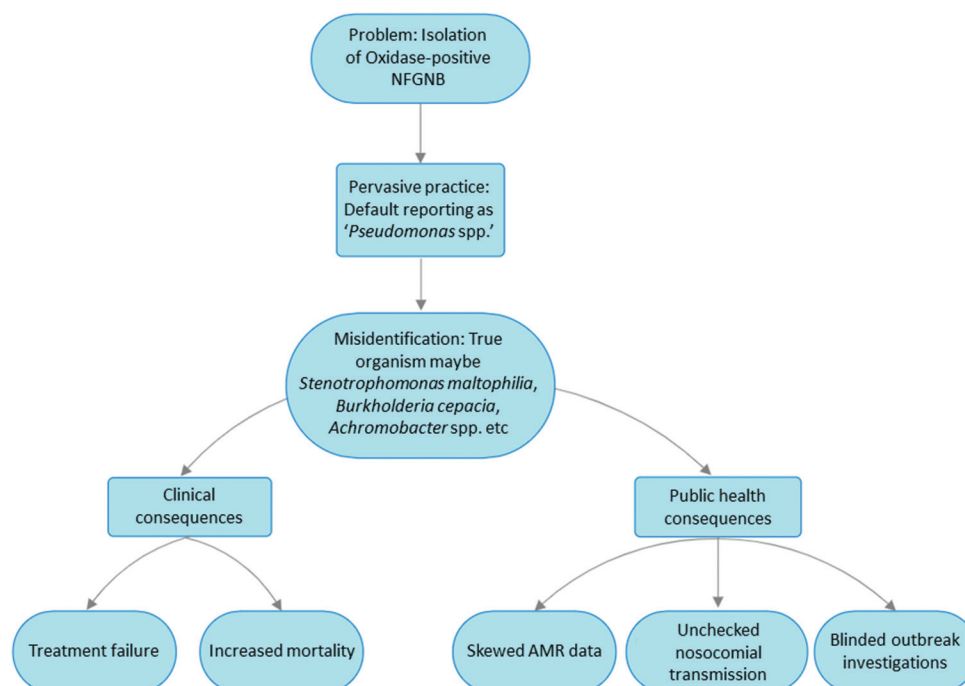


Figure 1. Negative implications of misidentifying non-fermenting Gram-negative bacilli
Abbreviations: AMR: Antimicrobial resistance; NFGNB: Non-fermenting Gram-negative bacilli.

contributing to poor clinical outcomes and the silent spread of antimicrobial resistance. The key differentiating features and intrinsic resistance patterns of common NFGNB are tabulated in Table 1.

4. Epidemiological and public health implications

Beyond the individual patient, the failure to accurately identify pseudomonads cripples effective infection prevention and control, particularly during outbreak investigations. The source of a hospital-onset outbreak is directly linked to the organism's natural reservoir. An outbreak of *P. aeruginosa* might be traced to contaminated water taps, sinks, or respiratory equipment.⁹ An outbreak of *B. cepacia* complex, historically associated with contaminated disinfectants and nebulizer solutions, requires investigating pharmaceutical and aqueous sources.¹⁰ An outbreak of *Elizabethkingia* spp., often linked to contaminated water systems and colonized faucets, demands a different investigative approach.¹¹ Without accurate identification, public health and infection prevention teams are unable to implement targeted interventions, nor can they definitively declare an outbreak over. This lack of resolution leaves healthcare facilities perpetually vulnerable to unsolved transmission cycles, putting the most immunocompromised patients at continuous risk.

Inaccurate laboratory identification of NFGNB not only compromises individual patient care but also distorts antimicrobial resistance surveillance data, especially in resource-constrained settings.^{12,13} Mislabeling diverse NFGNB as *Pseudomonas* spp. leads to erroneous resistance prevalence estimates, skewing national

and global antimicrobial resistance repositories and hindering evidence-based policy decisions. Furthermore, surveillance systems reliant on species-specific resistance profiles cannot detect emerging resistance mechanisms in understudied genera, allowing silent dissemination of resistant strains. From an epidemiological standpoint, outbreak investigations depend on precise species identification to trace sources and transmission pathways; failure to distinguish *S. maltophilia* or *B. cepacia* complex from *P. aeruginosa* can lead to misdirected infection control efforts and prolonged transmission. Incorporating diagnostic stewardship principles and selecting the right tests for the right patient at the right time have been shown to enhance identification accuracy, optimize antimicrobial use, and strengthen surveillance credibility in low- and middle-income countries (LMICs).¹²⁻¹⁵

5. Environmental reservoirs and nosocomial transmission pathways

Healthcare environments are replete with niches that facilitate the persistence and proliferation of NFGNB, such as *P. aeruginosa*, *Acinetobacter baumannii*, and *S. maltophilia*. Studies employing whole-genome sequencing have revealed that components of hospital plumbing, particularly sink drains, p-traps, and aerators, frequently harbor *P. aeruginosa* populations, which periodically seed surrounding surfaces and equipment. Although only a minority of nosocomial bloodstream infections, at approximately 15%, have been definitively traced back to these environmental isolates, the continual colonization of water systems underscores their role as persistent reservoirs that sustain endemic transmission within healthcare facilities.¹⁶

Table 1. Common NFGNB mistaken for *P. aeruginosa*: Key differentiators and intrinsic resistance patterns

Organism	Key differentiating features	Classic intrinsic resistance signals	First-line therapeutic options (empirical)
<i>Pseudomonas aeruginosa</i>	Growth at 42°C, acetamide +, pyocyanin pigment +, and motile	Variable, but often susceptible to anti-pseudomonals	Anti-pseudomonal penicillin/cephalosporin (e.g., piperacillin-tazobactam, ceftazidime), carbapenems, and aminoglycosides
<i>Stenotrophomonas maltophilia</i>	No growth at 42°C, oxidase –, lysine decarboxylase +, and non-motile	Carbapenems, aminoglycosides, and many beta-lactams	Trimethoprim-sulfamethoxazole, tigecycline, and levofloxacin
<i>Burkholderia cepacia</i> complex	No growth at 42°C, oxidase +/-variable, often slow lactose fermenter, and motile	Aminoglycosides, polymyxins	Trimethoprim-sulfamethoxazole, ceftazidime, and meropenem
<i>Achromobacter xylosoxidans</i>	No growth at 42°C, oxidase +, motile, and oxidative in OF test	Aminoglycosides, many cephalosporins	Trimethoprim-sulfamethoxazole, meropenem, and ticarcillin-clavulanate
<i>Chryseobacterium/Elizabethkingia</i> spp.	No growth at 42°C, oxidase +, produces yellow pigment, and non-motile	Aminoglycosides, colistin, and many beta-lactams	Piperacillin-tazobactam, rifampin, and fluoroquinolones
<i>Ralstonia pickettii</i>	No growth at 42°C, oxidase +, motile, and oxidative in OF test	Polymyxins, aminoglycosides (variable)	Trimethoprim-sulfamethoxazole and fluoroquinolones

Note: The “+” and “–” signs indicate “positive” and “negative”, respectively.

Abbreviations: OF: Oxidation-Fermentation; NFGNB: Non-fermenting Gram-negative bacilli.

Transmission from these environmental reservoirs occurs through multiple, often overlapping, pathways. Direct contact between healthcare workers' hands or patients and contaminated surfaces or medical devices enables rapid pathogen transmission, especially when NFGNB biofilms protect organisms from routine cleaning and disinfectant exposure. Device-associated infections, most notably those involving indwelling urinary catheters, central venous catheters, and ventilator circuits, are frequently initiated by contact with biofilm-laden water or surfaces, with the favorable microenvironment of biofilms further shielding bacteria from host defenses and antimicrobial therapy. In addition, routine water usage and cleaning activities can induce splash and aerosolization of contaminated water or biofilm fragments, dispersing bacteria onto benchtops, bedside rails, and even into ambient air.¹⁷ Mathematical modeling of intensive care unit outbreaks has estimated that environmental transmission routes, particularly those linked to plumbing biofilms, contribute to more than 80% of metallo- β -lactamase-producing *P. aeruginosa* cases, surpassing direct patient-to-patient transmission in importance.¹⁸ These findings highlight the necessity of integrating environmental control measures, such as periodic disassembly and targeted disinfection of plumbing fixtures, adoption of splash-resistant sink designs, and deployment of antimicrobial surface materials, into comprehensive infection prevention strategies to effectively interrupt nosocomial transmission chains.

A small percentage of the human population harbors *Pseudomonas* spp. as commensal microorganisms residing in the false nasal passages and digestive tract. While commonly recognized as opportunistic pathogens, some *Pseudomonas* spp. exist harmlessly within these niches under normal conditions, contributing to the microbial ecosystem balance. Their presence in these sites highlights the dual role of *Pseudomonas* as both commensal and pathogen, which has implications for colonization dynamics and infection risk assessments.¹

6. Challenges in low-resource laboratories

The persistence of this problem is not due to a lack of awareness but a consequence of severe systemic and operational constraints. Microbiology laboratories in LMICs face a triumvirate of challenges, namely financial limitations, infrastructural deficits, and human resource constraints.¹⁹ Automated identification systems, such as matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) or molecular sequencing, are prohibitively expensive to purchase, maintain, and sustain due to the high cost of reagents and databases. Even simpler commercial biochemical kit

systems represent a recurring cost that many laboratories are unable to afford. Furthermore, stable electricity, continuous internet access for database updates, and consistent supply chains for reagents are often unreliable. Perhaps most critically, there is a shortage of specialized microbiologists and trained technologists who can perform and interpret the nuanced biochemical tests required for manual differentiation. This perfect storm of constraints forces laboratories to rely on outdated, inadequate algorithms that stop at the genus level or, worse, at a presumptive identification.

In addition, a critical barrier to effective diagnostic stewardship lies in the divergent perspectives of laboratory management versus diagnostic management, the latter being clinician-led and driven by clinician-centric terminology and workflows. While laboratory management traditionally prioritizes technical accuracy and standardized reporting language, diagnostic management emphasizes the clinician's experience with the ordering system and seeks to shape clinical reasoning before a test is even selected. This mismatch can lead to confusion, delayed or inappropriate test ordering, and underutilization of nuanced diagnostic options, ultimately perpetuating suboptimal patient care and antimicrobial stewardship practices. To bridge this gap, future efforts should focus on the co-design of laboratory information systems and electronic test-ordering interfaces through collaborative human-factors research involving both clinicians and laboratory personnel, coupled with joint training workshops that align terminology and decision support prompts with clinical thought processes. Such initiatives would foster shared mental models, streamline test selection, and support proactive diagnostic decision-making at the point of care.

7. Pragmatic strategies for improved identification

Addressing this deep-seated issue requires innovative, multi-level, and context-specific strategies that move beyond expensive technology.

First, diagnostic stewardship must be embraced. This involves the implementation of simple, low-cost, algorithmic approaches using a core set of classic biochemical tests. Key tests include growth at 42°C (a key characteristic of *P. aeruginosa*), oxidation-fermentation tests, motility, and the use of simple media such as acetamide for *P. aeruginosa* identification. A structured diagnostic algorithm can significantly improve differentiation using resources typically already available. Through [Figure 2](#), a simplistic diagnostic algorithm for the identification of NFGNB incorporating key low-cost differentiation tests is proposed.

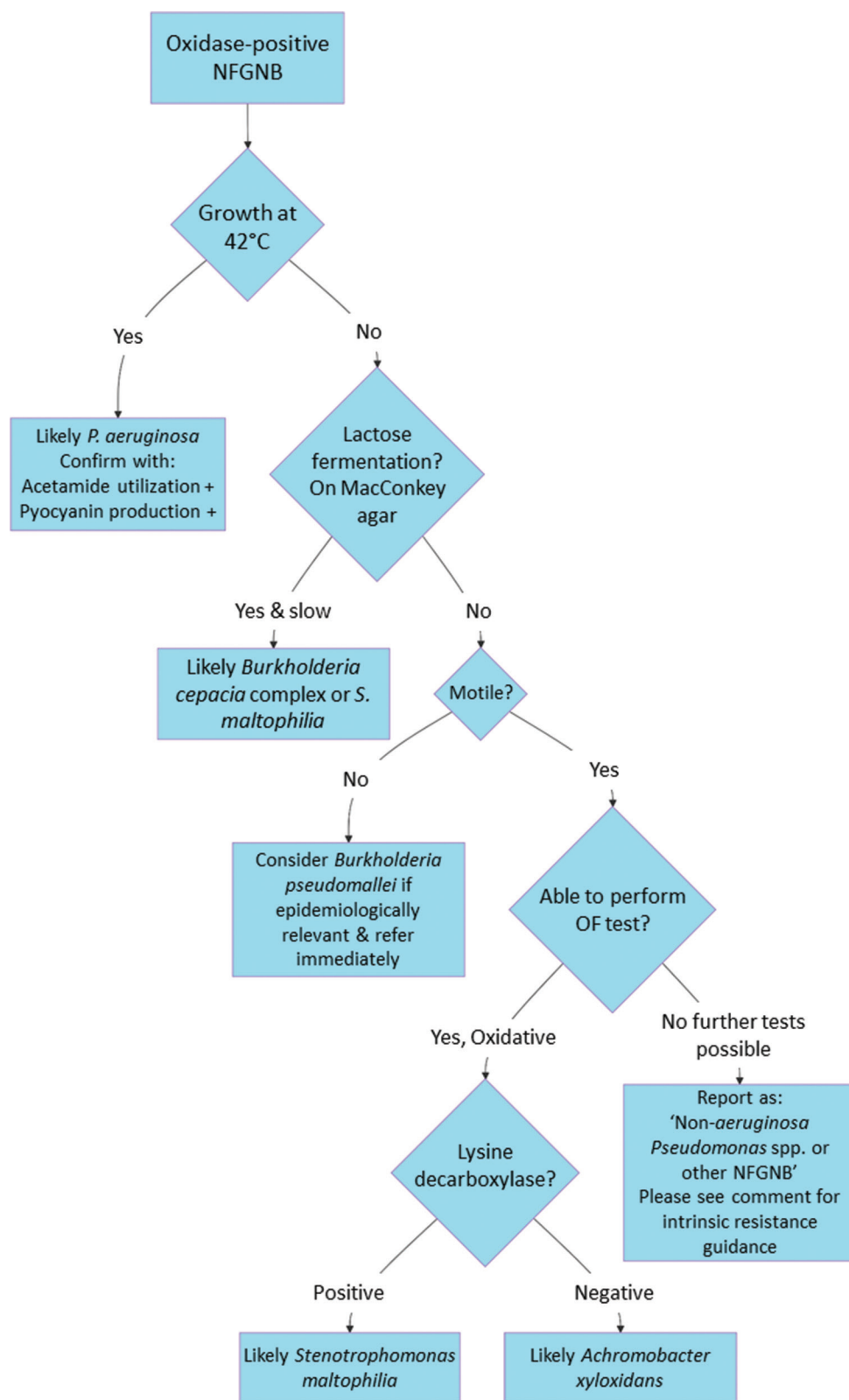


Figure 2. Diagnostic algorithm for non-fermenting Gram-negative bacilli incorporating key low-cost differentiation tests and parameters
Abbreviation: NFGNB: Non-fermenting Gram-negative bacilli.

Second, direct disk diffusion testing is a reliable and expedient method for the detection of antibiotic non-susceptibility, particularly in bloodstream infections caused by resistant organisms.^{14,15} This method facilitates rapid on-plate antimicrobial susceptibility results directly from positive blood cultures, enabling timely therapeutic decisions and optimized patient management. In view of its cost-effectiveness, this method can be more efficiently adopted in low-resource clinical microbiology laboratories, offering a valuable tool for managing antimicrobial resistance and improving outcomes in bloodstream infections.^{14,15}

Third, investment in human capacity is paramount. Sustained and practical training programs for laboratory technologists, focused on the interpretation of basic biochemical tests for NFGNB, are essential. This could be achieved through partnerships with national reference laboratories, international societies, and online training modules.

Fourth, regional collaboration and networking should be strengthened. Establishing a network where low-resource settings can refer difficult-to-identify or critical isolates, e.g., from sterile sites or outbreaks, to a central, better-equipped reference laboratory for confirmation is a highly cost-effective model. This builds capacity and ensures patient care is not compromised for the most complex cases.

Fifth, the scientific community must advocate for and develop affordable diagnostic solutions. This includes the development and validation of low-cost, latex agglutination tests for common pseudomonads, or the creation of open-source, offline database solutions for basic MALDI-TOF systems that could be donated or acquired at lower cost.

Finally, reporting practices must be standardized. If an isolate cannot be differentiated beyond a broad group level, the report should clearly state this uncertainty, for instance, “*Pseudomonas* spp. not *aeruginosa*” or “non-fermenting, oxidase-positive Gram-negative rods unidentified to genus and/or species level,” with an accompanying comment on the potential for variable antimicrobial resistance patterns, prompting closer consultation between the microbiologist and the clinician.

8. Conclusion

The habitual misidentification of pseudomonads in low-resource microbiology laboratories is a critical blind spot in the global healthcare system and individual patient safety. It is a practice that fosters antimicrobial misuse, obscures outbreak sources, and ultimately leads to preventable treatment failures. By adopting a pragmatic approach that

leverages existing tools through improved training and structured algorithms, strengthening referral networks, and advocating for the development of appropriate technologies, significant progress can be made. Moving beyond the default identification of “*Pseudomonas* species” is not merely a taxonomic exercise; it is a fundamental obligation of diagnostic microbiology to ensure precise therapy and effective infection control regardless of resource constraints.

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References

1. Lewandowska W, Mahillon J, Drewnowska JM, Swiecicka I. Insight into the phylogeny and antibiotic resistance of *Pseudomonas* spp. Originating from soil of the Białowieża National Park in Northeastern Poland. *Front Microbiol.* 2025;16:1454510. doi: 10.3389/fmicb.2025.1454510
2. Bahrami M, Bostanghadiri N, Goudarzi M, Khodaei N, Hashemi A. Antibiotic resistance and virulence factors in clinical isolates of *Stenotrophomonas maltophilia* from hospitalized patients in Tehran, Iran. *Int J Microbiol.* 2024;2024(1):8224242. doi: 10.1155/2024/8224242
3. Sfeir MM. *Burkholderia cepacia* complex infections: More complex than the bacterium name suggest. *J Infect.* 2018;77(3):166-170. doi: 10.1016/j.jinf.2018.07.006
4. Shaw T, Assig K, Tellapragada C, et al. Environmental factors

- associated with soil prevalence of the melioidosis pathogen *Burkholderia pseudomallei*: A longitudinal seasonal study from South West India. *Front Microbiol.* 2022;13:902996.
doi: 10.3389/fmicb.2022.902996
5. Klimkaitė L, Drevinskaitė R, Krinickis K, Sužiedėlienė E, Armalytė J. *Stenotrophomonas maltophilia* of clinical origin display higher temperature tolerance comparing with environmental isolates. *Virulence.* 2025;16(1):2498669.
doi: 10.1080/21505594.2025.2498669
 6. Qin S, Xiao W, Zhou C, *et al.* *Pseudomonas aeruginosa*: Pathogenesis, virulence factors, antibiotic resistance, interaction with host, technology advances and emerging therapeutics. *Signal Transduct Target Ther.* 2022;7(1):199.
doi: 10.1038/s41392-022-01056-1
 7. Farhan SM, Raafat M, Abourehab MA, *et al.* Effect of imipenem and amikacin combination against multi-drug resistant *Pseudomonas aeruginosa*. *Antibiotics (Basel).* 2021;10(11):1429.
doi: 10.3390/antibiotics10111429
 8. Gibb J, Wong DW. Antimicrobial treatment strategies for *Stenotrophomonas maltophilia*: A focus on novel therapies. *Antibiotics (Basel).* 2021;10(10):1226.
doi: 10.3390/antibiotics10101226
 9. Petkar HM, Caseres-Chiuco I, Al-Shaddad A, *et al.* Outbreak of *Pseudomonas aeruginosa* on a neonatal intensive care unit: Lessons from a Qatari setting. *J Infect Prev.* 2024;25(4):103-109.
doi: 10.1177/17571774241236248
 10. Bilgin H, Altınkanat Gelmez G, Bayrakdar F, *et al.* An outbreak investigation of *Burkholderia cepacia* infections related with contaminated chlorhexidine mouthwash solution in a tertiary care center in Turkey. *Antimicrob Resist Infect Control.* 2021;10(1):143.
doi: 10.1186/s13756-021-01004-8
 11. Yung CF, Maiwald M, Loo LH, *et al.* *Elizabethkingia anophelis* and association with Tap Water and handwashing, Singapore. *Emerg Infect Dis.* 2018;24(9):1730-1733.
doi: 10.3201/eid2409.171843
 12. Gupta A. Diagnostic stewardship in LMICs: The way forward. *J Antimicrob Steward Pract Infect Dis.* 2024;2(2):5-8.
doi: 10.62541/jaspi036
 13. Whistler T, Sangwichian O, Jorakate P, *et al.* Identification of Gram negative non-fermentative bacteria: How hard can it be? *PLoS Negl Trop Dis.* 2019;13(9):e0007729.
doi: 10.1371/journal.pntd.0007729
 14. Cercenado E. Rapid techniques for therapeutic optimization. Diagnostic stewardship. *Rev Esp Quimioter.* 2022;35(Suppl 3):80-83.
doi: 10.37201/req/s03.17.2022
 15. Zakhour J, Haddad SE, Kerbage A, *et al.* Diagnostic stewardship in infectious diseases: A continuum of antimicrobial stewardship in the fight against antimicrobial resistance. *Int J Antimicrob Agents.* 2023;62(1):106816.
doi: 10.1016/j.ijantimicag.2023.106816
 16. Virieux-Petit M, Ferreira J, Masnou A, *et al.* Assessing the role of environment in *Pseudomonas aeruginosa* healthcare-associated bloodstream infections: A one-year prospective survey. *J Hosp Infect.* 2025;156:26-33.
doi: 10.1016/j.jhin.2024.11.009
 17. Julia L, Vilankar K, Kang H, Brown DE, Mathers A, Barnes LE. Environmental reservoirs of nosocomial infection: Imputation methods for linking clinical and environmental microbiological data to understand infection transmission. *AMIA Annu Symp Proc.* 2017;2017:1120-1129.
 18. Pham TM, Büchler AC, Voor In 't Holt AF, *et al.* Routes of transmission of VIM-positive *Pseudomonas aeruginosa* in the adult intensive care unit-analysis of 9 years of surveillance at a university hospital using a mathematical model. *Antimicrob Resist Infect Control.* 2022;11(1):55.
doi: 10.1186/s13756-022-01095-x
 19. Shempela DM, Mudenda S, Kasanga M, *et al.* A situation analysis of the capacity of laboratories in faith-based hospitals in Zambia to conduct surveillance of antimicrobial resistance: Opportunities to improve diagnostic stewardship. *Microorganisms.* 2024;12(8):1697.
doi: 10.3390/microorganisms12081697