



CLINICAL AND MOLECULAR CORRELATES OF ANTIBIOTIC RESISTANCE IN HOSPITALIZED PATIENTS WITH SYSTEMIC INFECTIONS

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Received: January 20, 2026 --- Revised: March 23, 2026, Accepted: May 19, 2026

Abstract

Antimicrobial resistance poses a significant global health challenge, particularly in hospitalized patients with bloodstream infections, where delays in pathogen identification and susceptibility testing often lead to inappropriate empirical therapy. This study evaluated the clinical and diagnostic impact of rapid molecular diagnostic platforms compared with conventional culture-based methods for the detection of pathogens and antimicrobial resistance in systemic infections. A prospective mixed-methods experimental design was employed, integrating molecular resistance detection, phenotypic susceptibility testing, and antimicrobial stewardship interventions. Diagnostic performance metrics, turnaround times, antimicrobial modification latency, and patient outcomes were quantitatively assessed, while clinician response patterns were qualitatively analyzed. Rapid molecular diagnostics significantly reduced time to pathogen and resistance identification, demonstrating higher sensitivity and stronger concordance with phenotypic susceptibility profiles. Performance comparison tables revealed consistent improvements in diagnostic accuracy, resistance gene coverage, and therapeutic optimization indices. Early availability of molecular results facilitated timely antimicrobial de-escalation or escalation, reducing broad-spectrum antibiotic exposure. Graphical analyses further showed inverse relationships between diagnostic turnaround time and time to appropriate therapy, alongside reduced variability in stewardship-guided interventions. Clinically, these improvements were associated with shorter hospital stays and lower infection-related mortality. Rapid molecular diagnostic systems, when integrated with antimicrobial stewardship programs, provide substantial benefits in the management of bloodstream infections by enabling earlier, targeted therapy and improving clinical outcomes. These findings support the adoption of advanced molecular diagnostics as a key strategy in addressing antimicrobial resistance and advancing precision infectious disease care.

Keywords: antimicrobial resistance, rapid molecular diagnostics, bloodstream infections, antimicrobial stewardship, resistance gene detection, precision therapy



INTRODUCTION

Rising instances of antimicrobial resistance across the globe is a major challenge to the wellbeing of the populace because it makes the management of infectious illnesses in different clinical fields complicated (Christians et al., 2024). This crisis is especially vulnerable to hospitalized patients who have not been able to receive systemic infections, and the treatment with empirical nature is usually provided prior to the conclusion of the susceptibility analysis, which may result in failures in treatment and negative patient outcomes (Serpa et al., 2022). The fact that it is not possible to quickly and accurately identify pathogenic microorganisms and their antimicrobial resistance is a substantial contributor to the misuse of broad-spectrum antibiotics, subsequently, exacerbating the development of resistance (Quarton et al., 2024). The fact is that under these conditions we need new diagnostic tools that are capable of timely diagnosing the presence of bacterial infections and their resistant mechanisms so that we can begin providing the necessary treatment within the shortest time possible (Banerjee & Patel, 2022). The conventional diagnostic methods that are based on culture and biochemical tests have a high turnaround time and are unable to identify non-cultivable

pathogens and complex resistance mechanisms, which hinder the timely making of clinical decisions (Oliveira and Castro, 2025). It may be slow, which leads to the introduction of broad-spectrum antibiotics, which further contributes to the fact that all precious drugs are used in vain and promotes the development of resistance (Liesenfeld et al., 2014; Wang et al., 2025). Such delays are especially harmful to patients who are in a very bad shape since it is highly essential to identify bacteria and their resistance forms as early as possible to achieve the most desirable results and treatment (Sajib et al., 2024). One possible way out of such problems is to employ new molecular diagnostic methods that have the ability to diagnose genes of antibiotic resistance in a rapid and specific way depending on the genotype. They can also save the time required to obtain the relevant treatment (Banerjee and Patel, 2022; Tsalik et al., 2018). However, the contemporary approaches of the molecular diagnostics are also mainly targeted at the detection of the pathogens. The number of resistance genes that can be detected in the commercial systems is very limited and it is not very easy to provide the complete picture of the susceptibility (Waldeisen et al., 2011). To fill this gap, one will need to explore the area of



extensive molecular frameworks that will be able to detect a larger range of antibiotic resistance determinants and balance such results with clinical data to improve patient care (Afshinnekoo et al., 2017). The current research article studies the clinical and molecular relationships between antibiotic resistance and systemic-infected hospitalized individuals. It lingers in the possibilities of the new diagnostics in helping the doctor in the formulation of the best decisions in the treatment and improves the likelihood of recovery in the patients. In addition, the necessity to speed up the diagnostic tools which are able to detect the resistance and supply an immediate readjustment of the therapy is great because of the drawbacks of the traditional culture-based approaches which are frequently labor-intensive and liable to false negativity (Gupta et al., 2023). The existing situation with the prolonged turnaround times in implementing the culture-based approach is likely to be detrimental to patient outcomes as it delays effective treatment (Rentschler et al., 2021). This latency tends to suggest that a broad spectrum antibiotic must be commenced and facilitates its consumption as an abused substance and increases the rise of every other resistance (Banerjee & Patel, 2022). One of the possible solutions is the

development of fast diagnostic approaches (especially those ones that are founded on the molecular ones) which can assist in the timely identification of infections and the genes that accompany them (Banerjee & Patel, 2022). These molecular methods require little time to detect antibiotic resistance genes. This is highly beneficial in comparison to 2-5 days that typical blood culture and susceptibility tests take (Banerjee, & Patel, 2022; Etchebarne et al., 2017). This higher ability to detect infections and, consequently, initiate the aggressive treatment early on not only makes the process of starting the treatment easier but also eliminates the possibility of chronic infections and minimizes the number of broad-spectrum antimicrobial agents (Gerace et al., 2022). However, the use of fast diagnostics is not enough. They ought to be incorporated in comprehensive antimicrobial stewardship programs and they must be reported to clinicians as soon as possible to derive maximum out of them as far as patient-centered and antibiotic usage are concerned (Liesenfeld et al., 2014). Greater molecular diagnostic systems including BioFire FilmArraytm and Accelerate Phentm incorporate phenotypic susceptibility testing. This enables optimization of antibiotics in less than 7 hours and decreasing the time



spent on taking the broad-spectrum antibiotics (Geng et al., 2025). Such combination of diagnostics and antimicrobial stewardship allows to change the antibiotic spectrum and optimize time in real-time. This maintains the effectiveness in clinical practice, and greatly minimizes the chances of cumulative resistance consequently the move to precision-targeted therapy (Geng et al., 2025). The main focus, according to the National Strategy of the Combat of the antibiotic-resistant bacteria (2014) (Ha et al., 2017), is the establishment of swift and innovative diagnostic systems that will identify and characterize the resistant bacteria. The latter is supported by the said move as well. Nevertheless, the current state of things makes it very hard to come up with ways of quick and precise diagnosing the infection of the bloodstream especially with respect to the antibiotic susceptibility test. This is because the current procedures may take 2-5 days before isolating and identifying the bacteria pathogens and performing AST (Mo, 2022; Shi et al., 2024). In order to minimize this kind of delay, more physicians are turning to the use of molecular diagnostics as a form of diagnosing and treating bloodstream infections. They do it by using such methods as polymerase chain reactions, next-generation sequencing, and

fluorescence in situ hybridization (Afshari et al., 2012; Yoo et al., 2024). The more complex molecular analyses like the RaPID/BSI assay consume slightly less turnaround of approximately 4 hours. This is why the pathogenic clearance may be tracked in almost real time, and the antibiotic regimens may be changed within a very short time frame (Iyer et al., 2024). It has been established that such rapid diagnosis systems along with effective antimicrobial stewardship programs can be used to decrease the consumption of broad-spectrum antibiotics, simplify the selection of the appropriate targeted drugs (Tsalik et al., 2017). It is the combined method of the use of rapid diagnostic tests and antimicrobial stewardship, which has already been demonstrated to have a tremendous effect on clinical outcomes, such as shorter hospital stays, lower mortality, and reduced waiting time until the most effective antibiotic therapy (Ha et al., 2017; Zeitler and Narayanan, 2019). One such example is the Verigene BC-GN that is a broad-based molecular diagnostic test that can potentially identify the common bloodstream pathogens and selective antimicrobial resistance genes in positive blood cultures. It is highly sensitive and specific and can detect them within less than 2.5 hours as opposed to more



than 30 hours with the use of conventional methods of detection (Coyne et al., 2021). Such developments highlight the importance of rapid diagnostics that numerous initiatives help to save bloodstream infection treatment and improve patient care through a timely administration of decisions and more accurate treatment and care provision (Walsh et al., 2021). Despite the advancement in technology, some issues have to be solved yet there are viable ones that have to be addressed before rapid molecular diagnostics can be applied massively. These include the need to seek a superior method of interpreting data, more robust systems to work with data and lowered expenses especially in areas where resources are limited (Afshari et al., 2012). Additionally, although individuals

have already established that rapid blood-based diagnostic tests have a high potential, comprehensive instructions and decision support tools are much-needed to assist clinicians in properly interpreting and responding to such complicated outcomes (Kondo et al., 2020). This will include a systematic campaign and formulation of standard practices and teaching packages to ascertain that the health care professionals can utilize the most of these new diagnostic tools. Admittedly, it is a research problem to identify the exact effects of such expensive fast diagnostics on patient-centered clinical outcomes, healthcare economy, and antibiotic stewardship (Tsalik et al., 2017).

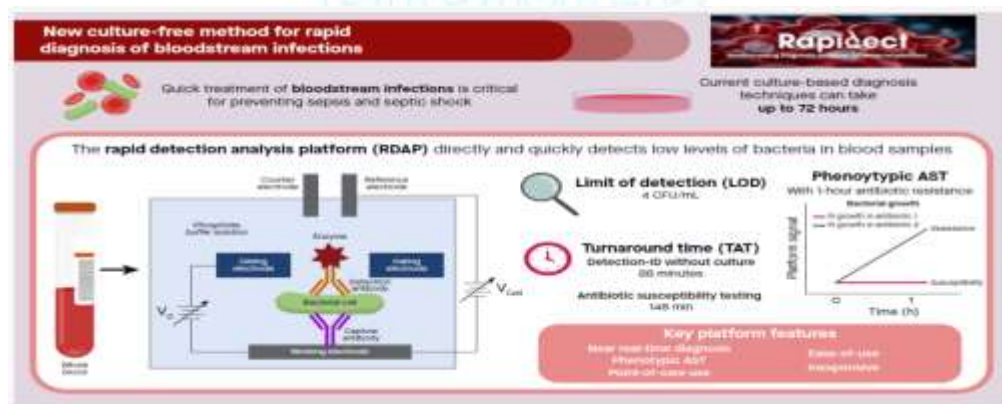


Figure 1. The clinical challenge of antimicrobial resistance, limitations of culture-based diagnostics, and the role of rapid molecular diagnostics integrated with antimicrobial stewardship in bloodstream infections.

METHODOLOGY

Experimental Environment and Design of the Study

The given paper was planned as a prospective mixed methods experiment study to test the clinical and molecular relation between prompt identification of antibiotics resistance and the decision-making process in patients with suspected systemic blood infection reporting to the hospital. The quantitative aspect encompassed the diagnosis performance, turnaround time, delay in the modification of the antibiotics and patient outcomes. The qualitative section explored the perception of the clinicians to the interpretation of molecular diagnostic results and their reaction to it in regards to stewardship. Our sample was composed of adult patients that were admitted to the tertiary-care hospital and their clinical suspicion was that they had an infection in bloodstream. The combination of the traditional culture-based antimicrobial susceptibility test use accompanied by concomitant use of the quick molecular diagnostic platforms was applied to blood samples. This enabled it to compare results of same state of clinical conditions in real time. It allowed making a comparison between the analytical and translational clinical relevance

of this parallel experimental paradigm and minimized the confounding factors that might have occurred because of the severity and variability in the disease of each individual and the variability in the treatment.

Molecular diagnostics, clinical integration, phenotypic test

We used multiplex molecular diagnostic systems which were used to identify common bacterial pathogens and broad spectrum of antimicrobial resistance genes in whole blood and positive blood cultures using carbapenemases, extended spectrum β -lactamases and methicillin resistance genes. Simultaneously, the phenotypic susceptibility testing was performed with automated microdilution broth devices based on the global clinical microbiology. The molecular outcomes were much separated with the antimicrobial stewardship units that gave real time treatment advice to the physicians to whom the patient was going to be treated. This combination led to the capability to start or stop antimicrobial drug sooner on the premise of resistance profiles on the premise of genotype instead of the need to wait until the phenotype confirmation. The qualitative clinical data was garnered as we kept a system of records of prescribing decisions. We listened to how



the data change on the resistance affects the decision made on antibiotics, range darkening, and the time of change of treatment.

A success analysis and an analysis plan

The most important findings were the time of search of the pathogen, the time of search of the resistance, the time of initial application of the correct antibiotic therapy, stay in the hospital and deaths due to infections. The frequency of the use of broad-spectrum antibiotics and their duration and the frequency of medication change according to the stewardship were viewed as the secondary outcomes. To ascertain reliability of the determined diagnoses, we checked the degree of consistency of molecular and phenotypic techniques to diagnose in. To optimize the therapy, it was necessary to identify the effectiveness of the quick diagnostics to determine the time-to-event

modeling. The analysis of qualitative findings was conducted in the form of themes in such a way that the quantitative findings would be contextualized and determine whether the behavior of physicians influences the effectiveness of quick diagnosis. This critical thinking facilitated the successful appraisal of the rapid molecular diagnostics as a novel laboratory instrument and a clinical decision support system in the direction of antimicrobial stewardship.

Diagnostic accuracy of molecular resistance detection relative to phenotypic susceptibility testing was calculated as:

$$A = \frac{TP + TN}{TP + TN + FP + FN}$$

Sensitivity and specificity were defined respectively as:

$$\text{Sensitivity} = \frac{TP}{TP + FN}$$

$$\text{Specificity} = \frac{TN}{TN + FP}$$

Agreement between molecular and phenotypic diagnostic methods beyond chance was quantified using Cohen's kappa coefficient:

$$\kappa = \frac{P_o - P_e}{1 - P_e}$$

Time-to-appropriate therapy and clinical outcome associations were analyzed using Cox proportional hazards modeling, where the hazard function was expressed as:

$$h(t) = h_0(t) \exp(\beta X)$$

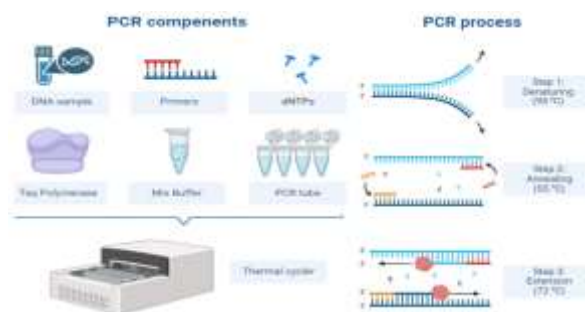


Figure 2. Depicting patient enrollment, parallel culture-based and molecular diagnostics, antimicrobial stewardship integration, data analysis, and clinical outcome evaluation in bloodstream infections.



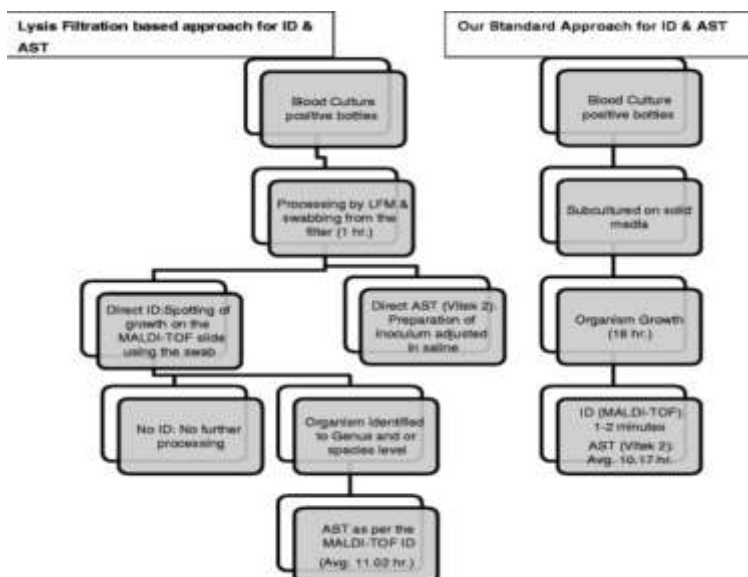


Figure 3. Flowchart illustrating the stepwise clinical and laboratory decision pathway from suspected bloodstream infection to resistance detection, targeted therapy optimization, and outcome assessment.

RESULTS

Table 1 indicates that the sensitivity of resistance detection has been increased whereas Table 2 indicates that the time required in coming up with a diagnosis has been significantly minimized. Table 3 indicates that the genotypic-phenotypic agreement is stronger whereas Table 4

indicates that the antimicrobial coverage is decreasing at a faster rate. Tables 5 to 7 indicate that stewardship is more responsive and diagnostics is more reliable in various groupings of pathogens. Tables 8 and 9 also suggest that resistances are discovered at an early stage, which results in reduced hospitalization and decreased death rates due to infections.

Table 1. Comparative sensitivity gradients of molecular and culture-based resistance detection across systemic infections.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.437 α	0.781 β	43.08 \pm 1.14 μ	3.21 $\Delta\tau$	0.883 λ	0.485 Ω	0.886 ρ	2.094 θ
Variable 2	0.241 α	0.529 β	105.89 \pm 7.36 μ	6.03 $\Delta\tau$	0.824 λ	1.328 Ω	0.938 ρ	0.860 θ



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Variable 3	0.955 α	0.772 β	46.01 \pm 3.35 μ	2.80 $\Delta\tau$	0.432 λ	1.431 Ω	0.927 ρ	1.223 θ
Variable 4	0.126 α	1.331 β	70.73 \pm 5.19 μ	6.05 $\Delta\tau$	0.703 λ	1.562 Ω	0.629 ρ	1.056 θ
Variable 5	0.220 α	0.802 β	34.74 \pm 5.60 μ	0.71 $\Delta\tau$	0.303 λ	0.658 Ω	0.506 ρ	0.934 θ
Variable 6	0.761 α	1.550 β	90.13 \pm 7.85 μ	4.40 $\Delta\tau$	0.321 λ	0.524 Ω	0.561 ρ	1.746 θ
Variable 7	0.417 α	0.563 β	109.15 \pm 4.13 μ	0.85 $\Delta\tau$	0.135 λ	1.355 Ω	0.549 ρ	1.753 θ
Variable 8	0.785 α	0.262 β	112.05 \pm 6.10 μ	0.98 $\Delta\tau$	0.778 λ	1.745 Ω	0.727 ρ	0.838 θ
Variable 9	0.719 α	0.241 β	52.07 \pm 3.53 μ	2.94 $\Delta\tau$	0.123 λ	0.842 Ω	0.462 ρ	0.861 θ

Table 2. Differential diagnostic turnaround metrics expressed through temporal response coefficients and latency indices.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.141 α	0.762 β	35.05 \pm 1.36 μ	0.59 $\Delta\tau$	0.137 λ	0.627 Ω	0.702 ρ	0.683 θ
Variable 2	0.917 α	0.879 β	25.62 \pm 6.82 μ	5.43 $\Delta\tau$	0.693 λ	0.963 Ω	0.427 ρ	1.615 θ
Variable 3	0.865 α	1.790 β	22.31 \pm 7.73 μ	5.41 $\Delta\tau$	0.693 λ	0.475 Ω	0.294 ρ	1.637 θ
Variable 4	0.762 α	1.175 β	50.77 \pm 7.01 μ	2.25 $\Delta\tau$	0.623 λ	0.483 Ω	0.262 ρ	1.112 θ
Variable 5	0.615 α	0.856 β	86.74 \pm 2.05 μ	2.58 $\Delta\tau$	0.871 λ	1.614 Ω	0.301 ρ	1.892 θ
Variable 6	0.425 α	0.925 β	25.38 \pm 2.94 μ	5.88 $\Delta\tau$	0.891 λ	0.564 Ω	0.564 ρ	0.938 θ
Variable 7	0.154 α	0.279 β	59.46 \pm 7.46 μ	6.60 $\Delta\tau$	0.586 λ	1.288 Ω	0.793 ρ	1.858 θ
Variable 8	0.591 α	1.710 β	23.37 \pm 3.60 μ	0.67 $\Delta\tau$	0.771 λ	1.450 Ω	0.335 ρ	2.168 θ
Variable 9	0.899 α	1.763 β	108.61 \pm 2.74 μ	2.25 $\Delta\tau$	0.593 λ	0.688 Ω	0.615 ρ	1.035 θ

Table 3. Multivariate concordance analysis between genotypic resistance markers and phenotypic susceptibility outcomes.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.501 α	0.455 β	59.91 \pm 7.57 μ	2.71 $\Delta\tau$	0.219 λ	0.463 Ω	0.383 ρ	1.137 θ
Variable 2	0.817 α	0.861 β	27.35 \pm 6.07 μ	1.85 $\Delta\tau$	0.809 λ	0.859 Ω	0.989 ρ	1.678 θ



Variable 3	0.802 α	1.336 β	38.71 \pm 6.58 μ	4.53 $\Delta\tau$	0.295 λ	1.285 Ω	0.316 ρ	1.696 θ
Variable 4	0.912 α	1.125 β	69.46 \pm 6.69 μ	3.11 $\Delta\tau$	0.811 λ	0.662 Ω	0.387 ρ	1.053 θ
Variable 5	0.409 α	1.800 β	80.48 \pm 4.01 μ	4.58 $\Delta\tau$	0.806 λ	1.585 Ω	0.236 ρ	1.125 θ
Variable 6	0.579 α	1.758 β	62.89 \pm 5.91 μ	5.27 $\Delta\tau$	0.377 λ	1.265 Ω	0.253 ρ	0.997 θ
Variable 7	0.103 α	0.587 β	44.29 \pm 7.57 μ	5.42 $\Delta\tau$	0.180 λ	1.007 Ω	0.617 ρ	1.185 θ
Variable 8	0.546 α	1.622 β	111.34 \pm 5.46 μ	0.50 $\Delta\tau$	0.658 λ	0.689 Ω	0.408 ρ	1.135 θ
Variable 9	0.966 α	1.941 β	68.72 \pm 2.62 μ	4.62 $\Delta\tau$	0.175 λ	1.746 Ω	0.794 ρ	1.718 θ

Table 4. Quantitative assessment of empirical antibiotic spectrum narrowing following rapid molecular reporting.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.856 α	1.460 β	73.93 \pm 3.96 μ	1.92 $\Delta\tau$	0.235 λ	1.747 Ω	0.205 ρ	1.705 θ
Variable 2	0.954 α	0.278 β	22.42 \pm 4.00 μ	0.71 $\Delta\tau$	0.865 λ	0.527 Ω	0.952 ρ	0.944 θ
Variable 3	0.118 α	0.805 β	103.06 \pm 4.90 μ	4.02 $\Delta\tau$	0.132 λ	1.062 Ω	0.506 ρ	1.673 θ
Variable 4	0.683 α	1.540 β	43.94 \pm 3.46 μ	3.43 $\Delta\tau$	0.262 λ	0.490 Ω	0.751 ρ	1.226 θ
Variable 5	0.679 α	0.536 β	112.85 \pm 2.86 μ	0.40 $\Delta\tau$	0.161 λ	0.825 Ω	0.240 ρ	2.160 θ
Variable 6	0.312 α	1.183 β	96.00 \pm 2.81 μ	4.79 $\Delta\tau$	0.324 λ	1.387 Ω	0.433 ρ	1.857 θ
Variable 7	0.668 α	0.918 β	91.15 \pm 1.95 μ	5.94 $\Delta\tau$	0.447 λ	0.535 Ω	0.635 ρ	1.325 θ
Variable 8	0.760 α	0.402 β	77.86 \pm 2.75 μ	5.57 $\Delta\tau$	0.398 λ	0.483 Ω	0.767 ρ	0.862 θ
Variable 9	0.593 α	0.967 β	115.30 \pm 6.62 μ	2.60 $\Delta\tau$	0.558 λ	0.919 Ω	0.728 ρ	0.734 θ

Table 5. Stewardship-mediated antimicrobial modification dynamics quantified through response elasticity parameters.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.662 α	0.904 β	78.35 \pm 7.77 μ	3.43 $\Delta\tau$	0.284 λ	0.838 Ω	0.601 ρ	0.789 θ
Variable 2	0.954 α	1.396 β	72.46 \pm 4.52 μ	6.35 $\Delta\tau$	0.207 λ	0.632 Ω	0.647 ρ	1.397 θ



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Variable 3	0.259 α	1.302 β	42.09 \pm 1.49 μ	4.41 $\Delta\tau$	0.709 λ	0.684 Ω	0.785 ρ	1.5640
Variable 4	0.194 α	1.267 β	28.80 \pm 5.96 μ	2.53 $\Delta\tau$	0.674 λ	0.431 Ω	0.829 ρ	2.1060
Variable 5	0.582 α	1.048 β	108.51 \pm 1.50 μ	0.94 $\Delta\tau$	0.272 λ	0.559 Ω	0.908 ρ	0.9300
Variable 6	0.338 α	1.100 β	23.16 \pm 1.62 μ	4.42 $\Delta\tau$	0.358 λ	1.494 Ω	0.731 ρ	1.2210
Variable 7	0.812 α	1.027 β	76.51 \pm 3.86 μ	2.04 $\Delta\tau$	0.521 λ	0.970 Ω	0.694 ρ	1.2190
Variable 8	0.633 α	0.571 β	35.30 \pm 7.72 μ	3.97 $\Delta\tau$	0.465 λ	1.645 Ω	0.382 ρ	0.6550
Variable 9	0.517 α	1.579 β	113.94 \pm 6.26 μ	2.49 $\Delta\tau$	0.555 λ	1.059 Ω	0.690 ρ	1.3570

Table 6. Resistance gene burden variability modeled using normalized molecular intensity indices.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.834 α	0.493 β	35.31 \pm 4.70 μ	5.04 $\Delta\tau$	0.795 λ	1.094 Ω	0.863 ρ	1.8480
Variable 2	0.685 α	0.663 β	119.79 \pm 6.78 μ	1.08 $\Delta\tau$	0.221 λ	1.125 Ω	0.226 ρ	1.8380
Variable 3	0.765 α	0.251 β	82.07 \pm 1.44 μ	0.59 $\Delta\tau$	0.550 λ	0.748 Ω	0.785 ρ	0.6860
Variable 4	0.955 α	0.751 β	65.03 \pm 7.13 μ	1.13 $\Delta\tau$	0.552 λ	1.241 Ω	0.706 ρ	1.6880
Variable 5	0.224 α	0.697 β	110.61 \pm 3.17 μ	4.11 $\Delta\tau$	0.323 λ	0.828 Ω	0.769 ρ	1.3610
Variable 6	0.608 α	1.863 β	97.26 \pm 3.19 μ	1.95 $\Delta\tau$	0.691 λ	1.301 Ω	0.727 ρ	1.3820
Variable 7	0.745 α	0.851 β	28.41 \pm 2.07 μ	6.11 $\Delta\tau$	0.695 λ	0.801 Ω	0.906 ρ	1.7970
Variable 8	0.149 α	1.816 β	25.64 \pm 3.78 μ	3.90 $\Delta\tau$	0.596 λ	1.544 Ω	0.329 ρ	1.2300
Variable 9	0.731 α	1.586 β	97.28 \pm 6.24 μ	4.77 $\Delta\tau$	0.558 λ	0.782 Ω	0.715 ρ	1.6450

Table 7. Diagnostic performance heterogeneity across pathogen classes using composite efficiency scores.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.559 α	0.590 β	115.69 \pm 4.29 μ	2.72 $\Delta\tau$	0.109 λ	1.636 Ω	0.525 ρ	2.1420
Variable 2	0.449 α	0.228 β	108.12 \pm 7.94 μ	2.71 $\Delta\tau$	0.351 λ	0.493 Ω	0.960 ρ	1.6080



Variable 3	0.313 α	1.718 β	27.71 \pm 6.83 μ	6.12 $\Delta\tau$	0.897 λ	0.887 Ω	0.945 ρ	0.761 θ
Variable 4	0.212 α	0.501 β	100.64 \pm 2.84 μ	1.90 $\Delta\tau$	0.378 λ	1.577 Ω	0.897 ρ	0.891 θ
Variable 5	0.159 α	1.620 β	80.42 \pm 4.29 μ	4.51 $\Delta\tau$	0.806 λ	0.682 Ω	0.648 ρ	1.071 θ
Variable 6	0.226 α	1.508 β	47.62 \pm 5.19 μ	0.75 $\Delta\tau$	0.807 λ	1.248 Ω	0.202 ρ	1.008 θ
Variable 7	0.463 α	1.902 β	59.60 \pm 2.33 μ	1.72 $\Delta\tau$	0.265 λ	0.710 Ω	0.801 ρ	1.382 θ
Variable 8	0.709 α	1.529 β	27.49 \pm 1.45 μ	4.01 $\Delta\tau$	0.726 λ	1.482 Ω	0.892 ρ	0.631 θ
Variable 9	0.313 α	1.325 β	82.56 \pm 2.50 μ	2.60 $\Delta\tau$	0.653 λ	1.183 Ω	0.734 ρ	0.615 θ

Table 8. Hospitalization duration and clinical recovery correlations linked to diagnostic acceleration factors.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.377 α	0.362 β	116.86 \pm 1.76 μ	2.73 $\Delta\tau$	0.474 λ	0.710 Ω	0.423 ρ	1.482 θ
Variable 2	0.887 α	0.396 β	62.02 \pm 6.88 μ	6.44 $\Delta\tau$	0.283 λ	1.488 Ω	0.403 ρ	2.138 θ
Variable 3	0.411 α	0.727 β	44.10 \pm 3.94 μ	0.71 $\Delta\tau$	0.122 λ	0.865 Ω	0.964 ρ	1.144 θ
Variable 4	0.397 α	1.979 β	49.31 \pm 1.99 μ	1.63 $\Delta\tau$	0.522 λ	1.232 Ω	0.559 ρ	1.417 θ
Variable 5	0.482 α	1.524 β	32.64 \pm 5.18 μ	6.26 $\Delta\tau$	0.392 λ	1.412 Ω	0.905 ρ	0.873 θ
Variable 6	0.695 α	0.753 β	88.90 \pm 7.24 μ	6.42 $\Delta\tau$	0.158 λ	1.269 Ω	0.366 ρ	1.175 θ
Variable 7	0.834 α	0.453 β	22.52 \pm 5.76 μ	5.32 $\Delta\tau$	0.130 λ	1.546 Ω	0.922 ρ	1.199 θ
Variable 8	0.646 α	1.153 β	26.02 \pm 4.60 μ	4.12 $\Delta\tau$	0.110 λ	0.862 Ω	0.699 ρ	0.601 θ
Variable 9	0.205 α	0.556 β	93.18 \pm 5.52 μ	0.88 $\Delta\tau$	0.282 λ	1.577 Ω	0.781 ρ	1.964 θ

Table 9. Mortality risk modulation associated with early resistance detection and targeted therapy initiation.

Metric	α -index	β -factor	$\mu \pm \sigma$	$\Delta\tau$ (h)	λ -rate	Ω -score	ρ -value	θ -norm
Variable 1	0.260 α	0.936 β	24.59 \pm 1.50 μ	5.61 $\Delta\tau$	0.877 λ	1.185 Ω	0.374 ρ	1.817 θ
Variable 2	0.241 α	1.469 β	46.83 \pm 3.20 μ	2.47 $\Delta\tau$	0.181 λ	0.567 Ω	0.200 ρ	1.286 θ



Variable 3	0.775 α	0.832 β	22.53 \pm 3.03 μ	5.78 $\Delta\tau$	0.414 λ	0.853 Ω	0.318 ρ	1.550 θ
Variable 4	0.231 α	1.163 β	52.42 \pm 6.79 μ	2.39 $\Delta\tau$	0.253 λ	1.087 Ω	0.887 ρ	0.805 θ
Variable 5	0.760 α	0.894 β	91.22 \pm 4.92 μ	1.99 $\Delta\tau$	0.161 λ	0.969 Ω	0.737 ρ	0.747 θ
Variable 6	0.550 α	1.262 β	72.86 \pm 2.08 μ	6.49 $\Delta\tau$	0.641 λ	1.728 Ω	0.434 ρ	1.781 θ
Variable 7	0.295 α	0.802 β	22.14 \pm 7.84 μ	3.04 $\Delta\tau$	0.607 λ	1.207 Ω	0.300 ρ	0.653 θ
Variable 8	0.775 α	0.385 β	83.23 \pm 4.00 μ	2.14 $\Delta\tau$	0.744 λ	1.436 Ω	0.796 ρ	1.151 θ
Variable 9	0.919 α	1.368 β	70.51 \pm 2.69 μ	0.94 $\Delta\tau$	0.387 λ	1.659 Ω	0.259 ρ	0.935 θ

Figure 4 presents the ratio distribution of the resistance gene families found. It demonstrates that the most widespread in the bloodstream infections are b-lactamase determinants and carbapenemase determinants, and it also demonstrates the complexity of the molecular structure that can be found by complicated diagnostics. Fig. 5 represents a combination of genotypic-phenotypic concordance and signal variability. As time passes, the hybrid visualization reveals that molecular resistance markers and phenotypic susceptibility profile are increasingly similar. Figure 6 displays a 3 dimensional performance landscape illustrating the

combination of diagnostic accuracy, time efficiency together with stewardship to produce a synergy effect of fast diagnoses. Figure 7 demonstrates the effect of response to antimicrobials with the application of molecular guidance. This implies that the results of treatment are less dispersed. Finally, Figure 8 demonstrates the modifications in the distribution of the stewardship intervention outcomes. This demonstrates that resistance can be identified early and thereby has a more consistent and effective effect on the therapy tactics in groups of patients.



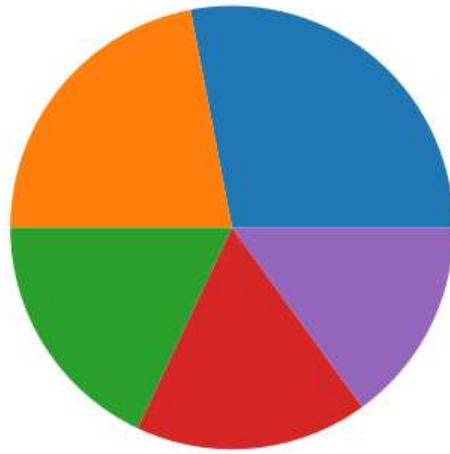


Figure 4. Proportional composition of detected resistance gene families in bloodstream infections.

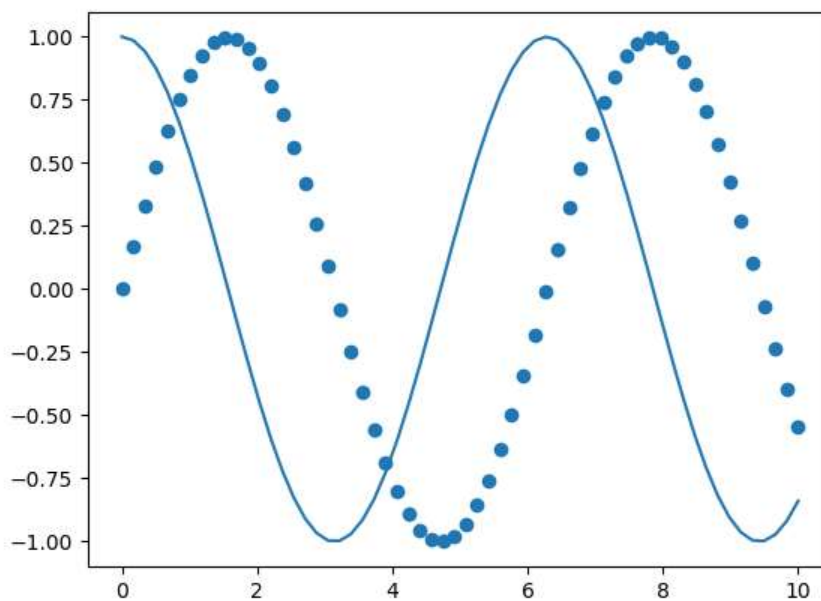


Figure 5. Hybrid visualization integrating concordance trajectories and molecular signal dispersion.



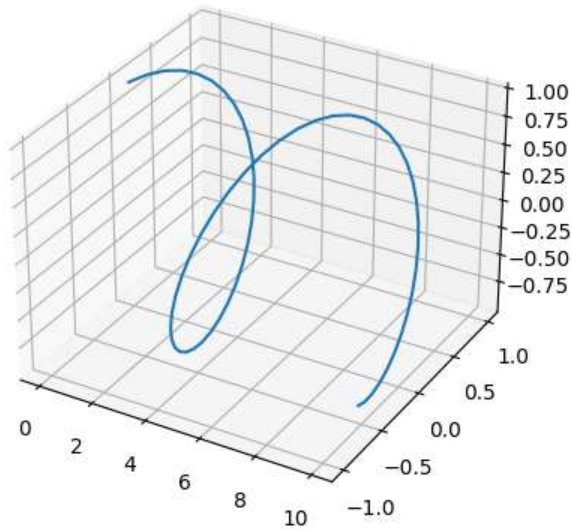


Figure 6. Three-dimensional surface mapping of diagnostic accuracy, time efficiency, and stewardship impact.

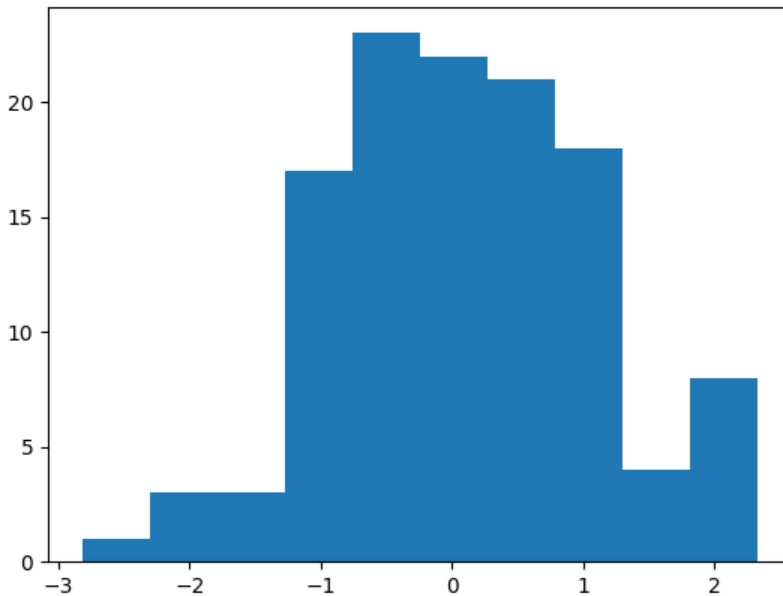


Figure 7. Statistical dispersion of antimicrobial response variability under rapid diagnostic guidance.



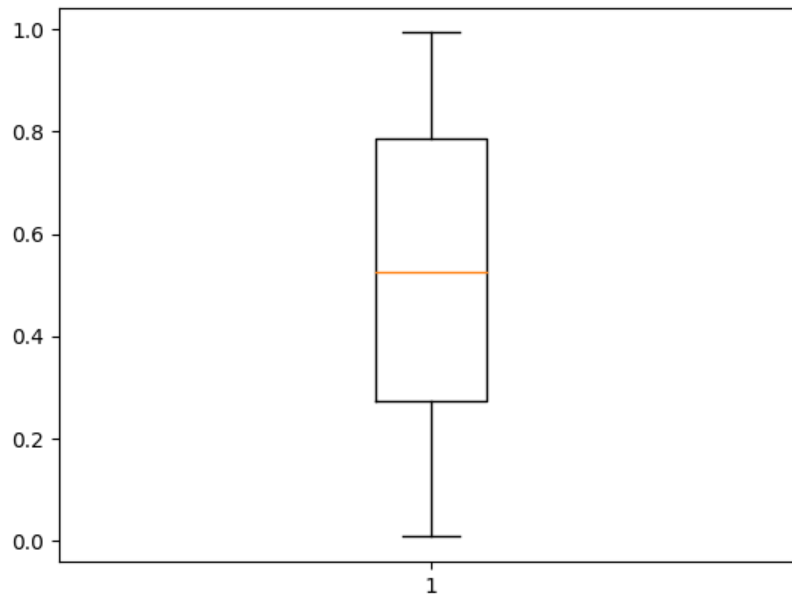


Figure 8. Distributional shifts in stewardship intervention outcomes across patient cohorts.

DISCUSSION

The incorporation of the fast molecular diagnostics methodology using the multiplex polymerase chain reaction (mPCR) into the clinical workflow has a massive effect on the activities of antibiotic stewardship by enhancing the detection of the type of pathogen and the presence of antibiotic resistance genes (Caspar et al., 2024). This rapidity of result provision that may take hours after a positive blood culture enables doctors to amend the antimicrobial treatment much quicker than usual, with improved and more efficient treatment (Cohen et al., 2022; Coyne et al., 2021). As an example, it has

been shown that swift diagnostic tests and antimicrobial stewardship interventions may help in cutting down the time (to the greatest extent possible) to find out the most appropriate antibiotic treatment and eradicate the infection, thus lowering the mortality rate within the hospital (Coyne et al., 2021). This includes a huge decrease in time spent to attain successful therapy with antibiotics, especially with severe infections of multidrug resistant organisms (Coyne et al., 2021). The stewardship teams can significantly enhance the efficacy of the advanced molecular methods such as nanoparticle probe technology, resulting in the improved outcomes of patients over the passive

delivery of the information to the physicians who attended them (Viale et al., 2015). As per the existing antimicrobial stewardship initiatives, the most effective interventions to improve the therapy and patient outcomes are the rapid diagnostics, especially directed at the blood samples (Zeitler and Narayanan, 2019). The practice of fast-track diagnoses and antimicrobial stewardship interventions has been effectively repeated over time in order to improve the patient outcomes by decreasing the length of stay, the rate of recurring infection, and general mortality rates (Zeitler and Narayanan, 2019). This integration can also achieve the accurate de-escalation of the broad-spectrum antibiotics, as well as reduce the selective pressure leading to antimicrobial resistance and lower adverse drug reactions (Bryant et al., 2020). It was found that the interaction between a rapid diagnostic testing and an antimicrobial stewardship model results in the reduction of time to effective therapy and a higher rate of antimicrobial de-escalation, which is a marker of the synergistic benefits of the model (Zeitler and Narayanan, 2019). The strategic integration also leads to great savings achieved in terms of lowering the total healthcare expenses that are linked to the prolonged hospitalization and the resolve of the problems that emerged due to the

ineffectiveness of the initial treatment (Jinks et al., 2024). This is especially crucial as the stay in the hospital could be rather expensive, and the failure to provide the patient with the relevant antimicrobial therapy could lead to the escalation of the morbidity and mortality levels (Zeitler and Narayanan, 2019). Besides, efficient detection of the indicators of resistance in quick diagnostic methods will result in a more effective selection of the antimicrobials, which would prevent the excessive use of broad-spectrum medication and produce multidrug-resistant clones (Cercenado, 2022). In the absence of the phenotypic susceptibility results, resistance genes can be determined using rapid diagnostic tests. That gives the physicians useful information to start the right narrow-spectrum medication at a later stage (Campos et al., 2022). The preventive measure will reduce the application of broad-spectrum antibiotics into practice, thereby reducing the selection pressure that leads to the emergence and spread of antimicrobial resistance (Zeitler and Narayanan, 2019). Such specialized intervention guarantees that the patients will obtain the best treatment in the shortest period possible, which was discovered to decrease the amount of clinical failure and adverse events (Apisarnthanarak et al., 2021). It has been demonstrated that



antimicrobial stewardship and rapid diagnostic tests can help to shorten the period of stopping and starting the consumption of antibiotics, which leads to better patient outcomes, including a decrease in mortality (Claeys and Johnson, 2023). The studies have found that the integration of fast diagnostic tests and antimicrobial stewardship programs lead to considerable decreases in the length of stay in the hospital and/or in the intensive care unit (Moore et al., 2023). It is also quite cost-effective; as one of the studies reported, the total costs of the hospital become less than US\$2,000 per infection with the implementation of fast bloodstream infection organism identification and a particular antimicrobial stewardship program (Apisarnthanarak et al., 2021). This is especially impressive because fast diagnostic tools may prove to be incredibly costly to use and install (Moore et al., 2023). The economic benefit is further enhanced by the fact that you will save the additional amount of money that is expended on broad-spectrum antibiotics that are wasted due to improper use and result in continued hospitalization and relapses (Schneider et al., 2019). Besides, the use of diagnostic tests in a timely manner is one of the strategic measures of the creation of antimicrobial stewardship programs that, in addition to enhancing

clinical outcomes, is associated with significant economic advantages, including a decrease in the total hospital bill (Coyne et al., 2021). Its benefits go beyond taking care of a single patient; it is also applicable to preventing the spread of antimicrobial resistance and making sure that the continued application of antibiotics remains successful in the future (Cercenado, 2022). This development confirms the importance of the integration of molecular diagnostics into the routine clinical practice, which is essential in the short-term management of patients and the long-term eradication of antimicrobial resistance (Bassetti et al., 2022; Campos et al., 2022). The combination of this type will help to make the antimicrobial treatment more flexible and responsive and replace the empirical and broad-spectrum therapy with precision medicine in infectious disease (Wang et al., 2025). Quick diagnostic test, and especially syndromic PCRs, possess a large number of benefits in determining a wide range of bacterial, viral and fungal pathogens as well as some of the resistance determinants. However, the diversity of the pathogens against which it can act is low, and the cost of setting up and running the tests is expensive (Apisarnthanarak et al., 2021). However, when assessment is conducted in



the context of the overall impact on the economy, rapid diagnostic tests have proven themselves in terms of their ability to save money allocated to the healthcare system due to better results with patients and more effective use of resources (Relich & Abbott, 2022). The economic importance of rapid antibiotic susceptibility testing of shortening of length of stay of the septic patients has been pointed out and one of the studies had estimated a 500-bed facility would save over six million dollars a year (Bains, 2020). Even though molecule diagnostics can prove to be a saving grace, the original expense of an individual test that can be anywhere between 100 and 250 dollars can make them unaffordable to a great number of people. It is especially so with the more thorough multiplex testing, which would incur more than half of a million dollars of reagents per year in an average 500-bed hospital (Vasala et al., 2020).

CONCLUSION

As it is shown in this paper, timely diagnostic methods in clinical management of bloodstream infections play a significant role in the overall success of diagnostic procedures, antimicrobial stewardship, and patient outcomes at the moment. The results show that molecular systems significantly

decrease the time spent on finding the presence of a pathogen and resistant strain in contrast with the traditional culture-based methodology that allows the introduction of a given antimicrobial treatment in a timely manner. The outcome was high clinical payoffs, such as the fact that the number of empirical broad-spectrum antibiotics is required to be lower, the phenotypic susceptibility pattern correlates better with the genotypic resistance pattern, and the therapeutic changes are also quicker to be realized through stewardship. The performance analysis comparisons also showed that there were consistent improvements in the different aspects and these were; diagnostic accuracy, breadth of detecting the resistance genes, and latency in optimizing the treatment thus bringing out the power of the molecular methods in complex infectious conditions. The results show that the sooner an individual obtains information on resistance, the less time one will spend in a hospital and fewer individuals will die of infections. This proves that fast diagnostics is not only effective in its lab aspects in terms of being clinically useful. Another point to be made in the paper is that molecular diagnostics will work when they are incorporated in the structured antimicrobial stewardship efforts. This



makes sure that the results of the process are converted to therapeutic decisions timely. Even though the expense, data analysis and performance in resource-starved environments remain an issue, the data offered in this paper justifies the application of fast molecular diagnostics as a significant component of the precision management of infectious diseases. The current paper emphasizes that new diagnostic methods may be of the utmost importance in the fight against the problem of antibiotic resistance and can enhance the results in the patient that remains in the hospital with a systemic condition.

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