International Journal of Antimicrobial Agents 67 (2026) 107937

Contents lists available at ScienceDirect

International Journal of Antimicrobial Agents

journal homepage: www.elsevier.com/locate/ijantimicag

Review

Evaluating rapid diagnostics for severe infections and sepsis: A systematic review and meta-analysis on behalf of the Hellenic Microbiological Society and the Hellenic Society of Chemotherapy

Evdoxia Kyriazopoulou[a,b], Konstantinos Patras[c,d], Simeon Metallidis[b,e], Konstantina Gartzonika[c,d], Karolina Akinosoglou[b,f], Maria Orfanidou[d,g], Ilias Karaiskos[b,h], Georgia Vrioni[d,i], Evangelos J. Giamarellos-Bourboulis[b,j,*], Athanasios Tsakris[d,i]

[a] Second Department of Critical Care Medicine, National and Kapodistrian University of Athens, Medical School, Athens, Greece
[b] Hellenic Society of Chemotherapy, Athens, Greece
[c] Department of Microbiology, Medical School, Ioannina University Hospital, Ioannina, Greece
[d] Hellenic Microbiological Society, Athens, Greece
[e] First Department of Internal Medicine, Aristotle University of Thessaloniki, Thessaloniki, Greece
[f] Department of Internal Medicine and Infectious Diseases, University General Hospital of Patras, Patras, Greece
[g] Department of Microbiology, General Hospital G. Gennimatas, Athens, Greece
[h] First Department of Internal Medicine and Infectious Diseases, Hygeia Hospital, Athens, Greece
[i] Department of Microbiology, National and Kapodistrian University of Athens, Medical School, Athens, Greece
[j] Fourth Department of Internal Medicine, National and Kapodistrian University of Athens, Medical School, Athens, Greece

ARTICLE INFO

Article history:
Received 17 February 2026
Accepted 14 July 2026

Editor: Stefania Stefani

Keywords:
Molecular diagnostics
Multiplex polymerase chain reaction
Pathogen identification
Antimicrobial treatment
Bloodstream infection
Sepsis

ABSTRACT

Objective: To assess the diagnostic accuracy and clinical benefit of molecular methods for pathogen identification and antimicrobial susceptibility through a systematic review and meta-analysis.
Methods: The protocol was prospectively registered with PROSPERO (CRD420251044218). A comprehensive search of MEDLINE (via PubMed), the Cochrane Library, and ClinicalTrials.gov was conducted to identify randomized controlled trials, non-controlled trials, and observational studies. The review investigated rapid diagnostic tests performed on blood samples for pathogen identification and susceptibility testing. The primary outcomes were Diagnostic Test Accuracy (DTA) for microbiological evaluation and time to pathogen identification, as well as to optimal/adjusted/appropriate antimicrobial treatment for clinical assessment. Secondary outcomes included 30-d mortality and total hospitalization cost. A random-effects model was used for quantitative synthesis.
Results: Of 1353 articles retrieved, 49 and 33 records were included in microbiological and clinical analysis. Pooled bivariate sensitivity for pathogen identification ranged from 0.97 to 0.98 (95% CIs approximately 0.94-0.99), with specificity consistently 0.99-1.00 across Gram-negative bacteria, Gram-positive bacteria, and yeasts. The mean time difference for pathogen identification was -21.43 hours (95% CI: -29.15 to -13.72; $P < 0.0001$; $I^2 = 99\%$), and for optimal/adjusted/appropriate treatment -13.78 hours (95% CI: -18.06 to -9.5; $P < 0.0001$; $I^2 = 97\%$). Total hospitalization costs were reduced; 30-d mortality was unaffected (OR 0.97; 95% CI: 0.88 to 1.07; $I^2 = 23\%$).
Conclusions: Evidence indicates that early, actionable diagnostics, particularly those from positive blood cultures, are essential to effective sepsis care.

1. Introduction

Early, appropriate antimicrobial treatment is key in the management of sepsis and/or bloodstream infection (BSI) [1]. Each hour of treatment delay increases mortality substantially [2]. Pathogen identification and antimicrobial susceptibility testing using phenotypic microbiological methods remains the gold standard for the diagnosis of BSIs but is associated with delays in the initiation of optimal antimicrobial treatment. Rapid microbiological methods have been developed based both on molecular and phenotypic technologies and provide faster results than do traditional methods. Current evidence suggests that molecular tests performed on

* Corresponding author. Mailing address: Fourth Department of Internal Medicine, ATTIKON University Hospital, 1 Rimini Street, 124 62 Athens, Greece.
E-mail address: egiamarel@med.uoa.gr (E.J. Giamarellos-Bourboulis).

https://doi.org/10.1016/j.ijantimicag.2026.107937

positive blood cultures (PBCs) may exhibit high diagnostic accuracy, decrease time to effective and optimal therapy, and lower hospital length of stay [3,4]. Early optimization of antimicrobial therapy may decrease the burden of antimicrobial resistance (AMR) [5].

The Hellenic Society of Chemotherapy and the Hellenic Microbiological Society are non-profit Greek scientific organizations sharing the vision to combat AMR. The members of the two societies conducted a systematic review and meta-analysis aiming to answer what the diagnostic performance of rapid microbiological tests is whether their use decreases time to optimal therapy, length of stay, mortality and hospitalization cost, and then provide a joint position paper on the appropriate use of these tests in patients hospitalized with sepsis/BSI/ severe infections.

2. Methods

2.1. Protocol registration

The protocol was prespecified and registered in the international prospective platform of systematic reviews (PROSPERO identifier: CRD420251044218), and the review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement and the PRISMA for Diagnostic Test Accuracy (DTA) studies statement [6,7].

2.2. Eligibility criteria

The primary question was formulated with a Population Intervention Comparator Outcome (PICO) format as following: *P*, adult patients (>18 years), hospitalized with severe infections, bacteraemia and/or sepsis; *I*, rapid diagnostic tests performed in blood samples for diagnostic purposes of pathogen identification and susceptibility testing. As rapid are defined technologies based either on molecular or phenotypic mechanisms that provide faster results compared with traditional microbiology. For microbiological diagnostic accuracy analyses, eligibility was restricted to tests permitting reconstruction of valid 2 × 2 contingency tables against a phenotypic microbiological reference standard. For clinical analyses, both molecular and rapid phenotypic diagnostic technologies were considered eligible; *C*, usual care, standard-of-care (SOC) phenotypic microbiological methods for pathogen identification and susceptibility testing in blood samples; *O*, The primary microbiological estimand was bivariate sensitivity and specificity at the target-group level (Gram-negative rods [GNR], Gram-positive cocci [GPC], yeast). Because the primary microbiological estimand was bivariate sensitivity and specificity, microorganism identification analyses were restricted to studies performed on PBCs that allowed reconstruction of target-group 2 × 2 contingency tables. Whole-blood assays were not pooled with PBC-based identification studies because of differences in specimen type, diagnostic pathway, and reference standard alignment. Similarly, rapid phenotypic susceptibility systems frequently report categorical agreement, essential agreement, or MIC agreement rather than the true positive/true negative [TN]/false positive [FP]/false negative data required for bivariate diagnostic accuracy modelling and, therefore, were also not included in pooled microorganism identification accuracy analyses. For AMR gene detection analysis, eligible studies were those reporting resistance-marker performance conditional on microorganism presence. The main clinical outcome used for eligibility was time (in hours or days) to pathogen identification, and time (in hours or days) to optimal/adjusted/appropriate antimicrobial treatment as this treatment was defined in individual studies. For clinical outcomes, we sought randomized and non-randomized controlled trials and observational (prospective or retrospective) studies published as full text in English. Non-human studies, paediatric studies, editorials, conference abstracts, case reports, and reviews were excluded. Systematic reviews were only used for additional references (snowballing method).

2.3. Information sources, search strategy, and study selection

A systematic literature search was performed on 6 May 2025 and repeated on 15 January 2026 across PubMed, Cochrane, and ClinicalTrials.gov databases. All articles providing a full-text in the English language were included. The detailed search strategy is provided in the Supplementary Material.

Two independent reviewers (EK, KP) screened all articles retrieved through the search. Those deemed eligible by title and abstract were further assessed by a full-text review. Any controversies were resolved by consensus, whenever needed. The following microbiological data were collected: author and publication year, country, study design, number of samples (PBCs), sampling method (e.g. consecutive or random), site (single centre or multicentre), index test, reference test for phenotypic microorganism identification and antimicrobial susceptibility testing testing, population (e.g. inpatient, ICU, emergency department), and outcomes of interest (true positive, TN, false positive, false negative) per individual study. Similarly the following data were collected for the clinical outcomes: author, publication year, country, study design, number of participants, type of intervention (type of test, presence of antimicrobial stewardship [AMS] program, infectious diseases expert consultation), type of infection (type of pathogen identified, characteristics of patients, hospitalization in ward or ICU) and outcomes of interest (length of hospital stay, mortality, time to pathogen identification, time to optimal/adjusted/appropriate treatment, time to de-escalation, total cost of hospitalization, and total antimicrobial consumption).

2.4. Outcomes

The primary microbiological outcomes were diagnostic test accuracy estimates (sensitivity and specificity) for GNR, GPC, and yeasts. These primary outcomes were defined at the target-group level and were estimated using bivariate models restricted to multiple-coverage panels (more than one target organism or resistance determinant belonging to the same predefined group is simultaneously tested within a single assay), which allow paired sensitivity–specificity estimation at the group level. Secondary microbiological outcomes included diagnostic accuracy estimates for AMR gene detection among GNR and GPC targets, as well as PPV and NPV for pathogen identification and AMR gene detection. In contrast to microorganism identification at the target-group level, single-coverage panels were eligible for inclusion in bivariate AMR gene detection analyses because AMR performance is evaluated conditional on microorganism presence, allowing valid definition of cases and non-cases at the gene level. When only one accuracy dimension was estimable (e.g. single-coverage identification panels at the target-group level), univariate agreement estimates were reported as supplementary analyses (positive percent agreement [PPA]).

The co-primary clinical outcome was time to pathogen identification, and time to optimal/adjusted/appropriate antimicrobial treatment. Time was calculated in hours, and if it was reported in days, it was transformed to hours. Optimal/adjusted/appropriate antimicrobial treatment was defined as in individual studies.

Secondary outcomes were 30-d mortality, long-term (60-d and 90-d) mortality, length of hospital stay, total cost of hospitalization, time (in hours) to de-escalation of antimicrobial treatment, and total antimicrobial consumption during hospitalization. If 28-d mortality was used, it was extrapolated as 30-d mortality. Cost

was transformed into Euros (€) if reported otherwise according to the current exchange rate.

2.5. Data synthesis

Diagnostic accuracy analyses were based on reconstructed 2 × 2 contingency tables derived from study-level raw microbiological data. Analyses primarily included data from monomicrobial PBCs or from mixed datasets comprising monomicrobial and polymicrobial cultures, according to how results were reported in the original publications. When individual components of the 2 × 2 table were incompletely reported, missing cells were derived algebraically from the total sample size and the available counts, provided sufficient information was available to ensure internal consistency. When studies reported partial 2 × 2 data but not TN, it was derived by subtracting the reported components from the total sample size, provided the denominator corresponded to the analysed target group.

Diagnostic accuracy was evaluated at a group target level (e.g. total Gram-negative bacteria, total Gram-positive bacteria, yeasts, or grouped antimicrobial-resistance targets); thus, study-level 2 × 2 tables were constructed by aggregating data across all individual targets belonging to that group. Invalid or indeterminate test results (e.g. "no call") were excluded from the primary analyses; when discrepancy analyses were performed by the original investigators, only the pre-discrepancy results were used for meta-analysis.

Sensitivity and specificity for pathogen identification were jointly estimated using a bivariate random-effects logistic regression model, treating sensitivity and specificity as correlated binomial outcomes [8]. Analyses were conducted within a frequentist framework using generalized linear mixed-effects models, with random intercepts for both sensitivity and specificity at the study level [9]. Summary estimates were obtained on the logit scale and back-transformed to the probability scale and are presented as pooled estimates with corresponding 95% CIs. The summary operating point was obtained by inverse-logit transformation of the pooled logit parameters.

The primary microbiological analyses were restricted to studies for which paired sensitivity–specificity estimates could be robustly modelled. For microorganism identification, primary bivariate analyses were restricted to multiple-coverage panels at the target-group level. Panels that did not permit paired sensitivity–specificity estimation at the target-group level (i.e. single-coverage identification panels) were not included in the primary bivariate identification syntheses; these contributed only to univariate sensitivity-only agreement analyses (PPA) and were treated as supplementary. For AMR gene detection, bivariate sensitivity–specificity and PPV–NPV analyses were performed when gene-level 2 × 2 tables were available, including studies using single-coverage panels because AMR detection was evaluated conditional on microorganism presence.

When bivariate models failed to converge because of sparse data, model simplification was applied by reducing the random-effects structure and/or fixing the correlation parameter; if convergence remained unattainable, univariate random-effects models were used as an analytical fallback for the same estimand. Separately, panels that did not permit paired estimation at the target-group level (single-coverage identification panels) were analysed using univariate sensitivity-only models (PPA) as predefined supplementary analyses. All models were fitted using the lme4 package in R, employing the glmer function with a binomial likelihood and adaptive Gaussian quadrature [10].

Between-study heterogeneity was evaluated visually using summary receiver operating characteristic (SROC) plots derived from the fitted bivariate models. The extent of heterogeneity in sensitivity and specificity on the original probability scale was reflected by the size of the predictive region around the summary operating point. SROC curves and associated confidence and prediction regions were generated by inverse transformation of the model-based logit estimates. For analyses based on univariate models, SROC curves were not generated.

For clinical outcomes, a separate synthesis (with or without quantitative analysis) was planned for each outcome. For dichotomous outcomes, we extracted the number of patients in each arm and expressed those as proportions with 95% CIs. Differences between arms were expressed as pooled OR with 95% CI. For continuous outcomes, we recorded the mean or median and measure of variability of the sample (S.D., IQR) in each arm, as reported. If median and IQR were reported, they were converted to mean and S.D. [11]. Experimental effect was measured by mean difference and respective 95% CI.

Meta-analyses for clinical outcomes were performed using the R software v. 4.0.2 after installing the packages "meta," "metaphor," and "dmetar" [12,13]. In all cases, the random-effects model (Der Simonian and Laird) was used to account for both within- and across-trial variation [14]. For each analysis, the corresponding forest plot was produced, while publication bias was assessed with the Egger test via funnel plot asymmetry [15]. Heterogeneity was assessed with the I^2 statistic [16] and cut-offs of $\leq$30, 30-59, 60-75, and $\geq$75 delimitated low, moderate, substantial, and considerable heterogeneity, respectively.

Pre-specified subgroup analysis for the primary microbiological outcomes was conducted for culture composition (e.g. monomicrobial vs. mixed), while sensitivity analyses were performed by excluding studies with a high risk of bias or applicability concerns in any QUADAS-2 domain [17]. For clinical outcomes, the following subgroup analyses were pre-planned to address heterogeneity regarding the clinical primary endpoint: RCT vs. non-RCT; ICU vs. non-ICU; AMS program vs. no-AMS; by type of pathogen (Gram positive- vs. Gram negative-bacteria, multidrug resistant (MDR) vs. non-MDR); by type of rapid diagnosis method used. Subgroup differences were evaluated by the Q-statistic [18].

For all analyses, a *P*-value of $<$0.05 was considered statistically significant.

2.6. Reporting bias assessment

For diagnostic accuracy studies, the risk of bias was assessed using the Quality Assessment of Diagnostic Accuracy Studies (QUADAS)-2 tool [17]. For the purposes of the present review, a modified version of the tool was applied to previously published guidelines [19]. Any controversies were resolved by consensus.

For clinical outcomes, risk of bias assessment for individual studies was performed independently by the two reviewers, using the RoB2 tool (The Cochrane Handbook for Systematic Reviews of Interventions) for randomized trials and the Newcastle Ottawa Scale for observational studies [20].

Assessment of publication bias in diagnostic test accuracy meta-analyses is challenging because sensitivity and specificity are paired outcomes, standard funnel plots are not informative, and conventional Egger regression is not valid in a bivariate framework [21]. Therefore, potential small-study effects were evaluated instead using a regression-based approach specifically developed for multivariate diagnostic accuracy data [22]. We applied the Multivariate Small Study Effect Test (MSSET), a generalized Egger test for bivariate outcomes, via the MVPBT package in R, which jointly assesses asymmetry in sensitivity–specificity estimates under a bivariate random-effects model [23]. Both second-order and third-order generalized regression tests were performed. Evidence of small-study effects was inferred when the corresponding regression test yielded a statistically significant result. These analyses

were conducted separately for Gram-negative, Gram-positive, and yeast identification. Publication bias for clinical outcomes was assessed by Funnel plot asymmetry.

2.7. Certainty of the evidence

Certainty of evidence (CoE) regarding the primary microbiological outcomes was assessed based on the Grading of Recommendations Assessment, Development and Evaluation (GRADE) for diagnostic accuracy studies [23]. CoE regarding the clinical primary outcomes was assessed using the GRADE criteria [24].

3. Results

3.1. Study selection

The search for microbiological outcomes yielded 1353 citations. After applying exclusion criteria, 82 records were included in microbiological (n = 49) and clinical (n = 33) analysis, respectively (Supplementary Fig. 1).

3.2. Study characteristics

A total of 49 studies comprising 10 549 samples were retrieved reporting on the diagnostic test accuracy of rapid molecular diagnostic methods performed in blood samples of adult patients with severe infections, bacteraemia, and/or sepsis [25–73]. Thirty-seven studies reported data on GNR identification, 28 on GPC identification, 20 on yeast identification, 26 on GNR AMR gene detection, and 29 on GPC AMR gene detection. Diagnostic accuracy data were reported at the target-group level (GNR, GPC, yeast, or AMR gene groups), with individual targets aggregated as described in the Methods section. A total of 18 studies included exclusively monomicrobial PBCs, 19 studies reported mixed datasets comprising both monomicrobial and polymicrobial cultures, and 12 studies did not explicitly report blood culture composition. For pathogen identification, only studies permitting paired estimation of sensitivity and specificity at the target-group level contributed to the primary bivariate analyses (GNR = 18, GPC = 18); studies with single-coverage identification panels were included in supplementary PPA (sensitivity-only) analyses (GNR = 19, GPC = 10). In contrast, single-coverage panels contributed to the bivariate analyses of AMR gene detection, where accuracy was conditional on organism presence (GNR AMR = 17 of 26, GPC = 11 of 29). Characteristics of included DTA studies are presented in Supplementary Table 1.

A total of 33 studies were retrieved reporting on the clinical use of rapid/molecular diagnostic methods in blood samples of patients with bacteraemia and/or sepsis (70,72, 74–104; 13 were RCTs and 20 observational studies). Eighteen studies reported on multiplex PCR tests, 5 on MALDI-TOF, and 10 on other, mainly rapid phenotypic methods. Four studies reported on Gram-negative bacteraemia only; seven on Gram-positive bacteraemia only, mainly by *Staphylococcus aureus*, one study reported on candidemia, and one on MDR bacteraemia. Four studies included patients with haematological malignancy and/or febrile neutropenia. In 13 studies, rapid diagnosis was performed within an AMS program. Characteristics of included studies are presented in Supplementary Table 2.

3.3. Risk of bias in included studies

The risk of bias and concerns for applicability for included DTA studies by the QUADAS-2 tool are provided in Supplementary Figs. 2 and 3. For more than half of the studies, there were some concerns regarding the reference standard; high risk of bias was noted for ~25% of studies for patient selection; similarly, for ~25% some concerns were noted for the index test. Quality assessment of observational studies, according to the Newcastle Ottawa Scale (NOS) and of RCTs according to the RoB2 tool, are provided in Supplementary Tables 3 and 4, respectively. The overall quality was high.

3.4. Microbiological outcomes

3.4.1. Primary outcomes

For GNR identification, the summary estimates for sensitivity and specificity were 0.98 (95% CI: 0.95-0.99) and 1.00 (95% CI: 0.99-1.00), respectively (Fig. 1A). No potential outlier was identified based on the SROC plot for GNR identification (Fig. 1B). Evidence of small-study effects was observed for GNR identification (bootstrap MSSET T = 9.05, P = 0.007), suggesting that smaller studies tended to report higher diagnostic accuracy than larger studies. Studies evaluating single-coverage panels, which were analysed separately using univariate sensitivity-only models, yielded a pooled PPA of 0.95 (95% CI: 0.91-0.97) (Supplementary Fig. 4A). While acknowledging the different estimands addressed by these analyses, the univariate estimate was consistent with the sensitivity estimate derived from the bivariate analyses of multiple-coverage panels.

For GPC identification, the summary estimates for sensitivity and specificity were 0.97 (95% CI: 0.94-0.98) and 1.00 (95% CI: 0.99-1.00), respectively (Fig. 2A). No potential outlier was identified based on the SROC plot for GPC identification (Fig. 2B). There was no evidence of small-study effects for GPC identification (bootstrap MSSET T = 5.85, P = 0.06), although the result was borderline and directionally consistent with a possible small-study effect. Studies evaluating single-coverage panels yielded a pooled PPA of 0.97 (95% CI: 0.93-0.97) (Supplementary Fig. 4B) consistent with the sensitivity estimate derived from the bivariate analyses of multiple-coverage panels.

For yeast identification, the summary estimates for sensitivity and specificity were 0.97 (95% CI: 0.94-0.98) and 1.00 (95% CI: 0.99-1.00), respectively (Fig. 3A). No potential outlier was identified based on the SROC plot for GPC identification (Fig. 3B). There was no evidence of small-study effects for GPC identification (bootstrap MSSET T = 4.24, P = 0.08).

Subgroup analysis by culture composition across organisms indicated that pooled sensitivity and specificity estimates were largely consistent between subgroups, with overlapping CIs. No subgroup demonstrated a clinically meaningful deviation from the primary pooled estimates (Supplementary Table 5).

Sensitivity analyses excluding studies with high risk of bias or applicability concerns in any QUADAS-2 domain yielded results that were identical to the primary analyses, indicating robustness of the findings (Supplementary Fig. 5).

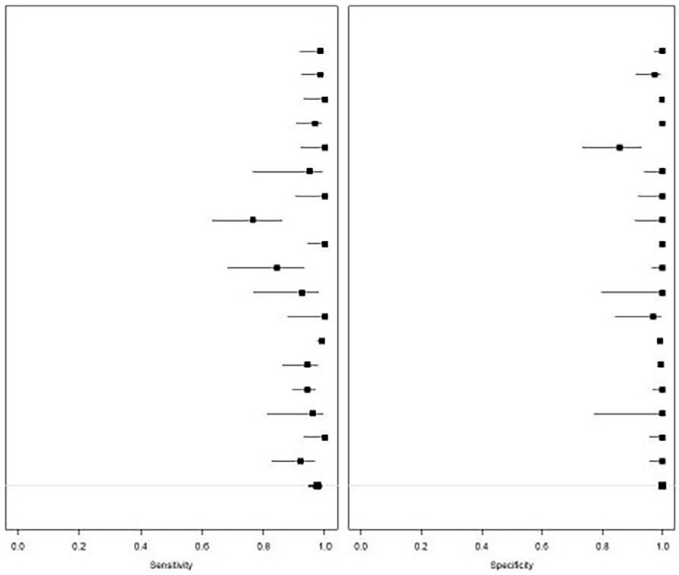
The CoE was assessed separately for each primary microbiological outcome (Supplementary Table 6). The CoE for GNR identification was moderate, downgraded due to unclear risk of bias in the reference standard domain across almost all studies. No serious concerns were identified for indirectness, inconsistency, imprecision, or small-study effects, and sensitivity analyses did not change pooled estimates. Similarly, the CoE for GPC identification was moderate, downgraded due to unclear risk of bias in the reference standard domain across almost all studies. Finally, the CoE for yeast identification was moderate, downgraded again due to unclear risk of bias in the reference standard domain across almost all studies.

3.4.2. Secondary outcomes

GNR-AMR had pooled estimates of sensitivity and specificity of 0.93 (95% CI: 0.87-0.96) and 1.00 (95% CI: 1.00-1.00), respectively

A

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Altun2013	66	0	1	133	0.99 (0.92–1.00)	1.00 (0.97–1.00)
Berinson2021	71	2	1	75	0.99 (0.93–1.00)	0.97 (0.91–0.99)
Burrack-Lange2018	52	7	0	2839	1.00 (0.93–1.00)	1.00 (0.99–1.00)
Camelena2023	88	0	3	2189	0.97 (0.91–0.99)	1.00 (1.00–1.00)
ElSherif2022	45	7	0	42	1.00 (0.92–1.00)	0.86 (0.73–0.93)
Fhoobiall2016	19	0	1	60	0.95 (0.76–0.99)	1.00 (0.94–1.00)
Holma2022	36	0	0	44	1.00 (0.90–1.00)	1.00 (0.92–1.00)
Kang2020	39	0	12	38	0.76 (0.63–0.86)	1.00 (0.91–1.00)
Kim2024	64	0	0	1691	1.00 (0.94–1.00)	1.00 (1.00–1.00)
Oberhettinger2020	27	0	5	104	0.84 (0.68–0.93)	1.00 (0.96–1.00)
Otlu2015	25	0	2	15	0.93 (0.77–0.98)	1.00 (0.80–1.00)
Peri2022	28	1	0	31	1.00 (0.88–1.00)	0.97 (0.84–0.99)
Rhoads2023	331	60	3	7124	0.99 (0.97–1.00)	0.99 (0.99–0.99)
Rule2021	66	5	4	783	0.94 (0.86–0.98)	0.99 (0.99–1.00)
Smith2023	148	0	9	116	0.94 (0.89–0.97)	1.00 (0.97–1.00)
Sparks2021	25	0	1	13	0.96 (0.81–0.99)	1.00 (0.77–1.00)
Verhoeven2018	52	0	0	88	1.00 (0.93–1.00)	1.00 (0.96–1.00)
Verroken2019	58	0	5	86	0.92 (0.83–0.97)	1.00 (0.96–1.00)
Summary estimate (bivariate glmer)					**0.98 (0.95–0.99)**	**1.00 (0.99–1.00)**



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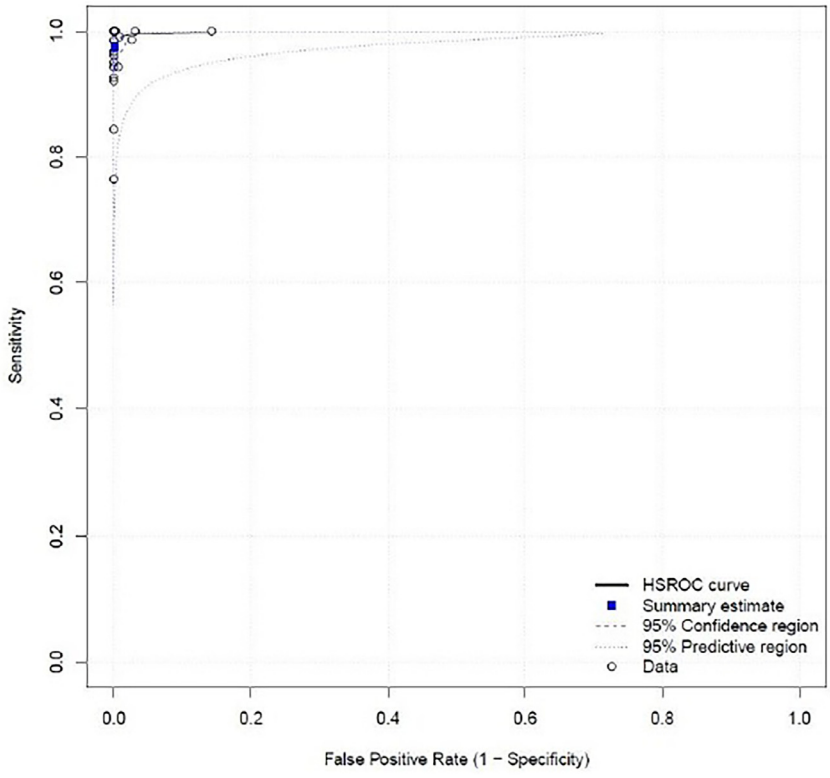


Fig. 1. Summary sensitivity-specificity (A) and summary receiver operating characteristic (SROC) (B) for Gram-negative rods (GNR) identification.

(Supplementary Fig. 6A). There was no between-study heterogeneity for specificity as was evident based on the SROC plot for GNR-AMR gene identification (Supplementary Fig. 6B).

GPC-AMR had pooled estimates of sensitivity and specificity of 0.98 (95% CI 0.96 to 0.99) and 1.00 (95% CI 0.99 to 1.00) respectively (Supplementary Fig. 7A). No potential outlier was identified based on the SROC plot for GPC-AMR gene identification (Supplementary Fig. 7B).

PPV and NPV for GNR identification were 1.00 (95% CI: 0.97-1.00) and 0.99 (95% CI: 0.98-1.00), respectively (Supplementary Fig. 8A). Single coverage panels for GNR identification had 1.00 (95% CI: 0.97-1.00) PPV (Supplementary Fig. 8B).

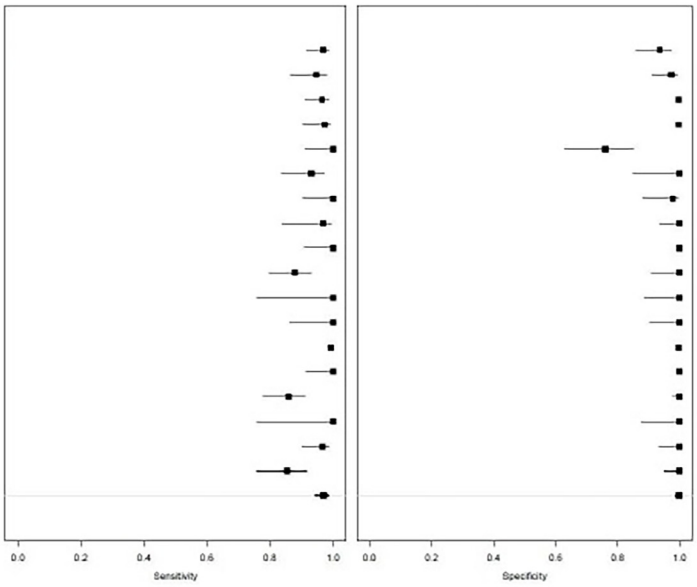
PPV and NPV for GPC identification were 0.99 (95% CI: 0.96-1.00) and 0.99 (95% CI: 0.97-1.00), respectively (Supplementary Fig. 9A). Single coverage panels for GPC identification had 1.00 (95% CI: 0.99-1.00) PPV (Supplementary Fig. 9B).

PPV and NPV for yeast identification were 0.95 (95% CI: 0.80-0.99) and 1.00 (95% CI 1.00-1.00), respectively (Supplementary Fig. 10).

PPV and NPV for GNR-AMR were 1.00 (95% CI: 0.98-1.00) and 0.99 (95% CI: 0.98-1.00), respectively (Supplementary Fig. 11), while PPV and NPV for GPC-AMR were 0.99 (95% CI: 0.96-1.00) and 0.99 (95% CI: 0.99-1.00), respectively (Supplementary Fig. 12).

A

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Altun2013	117	5	4	74	0.97 (0.92–0.99)	0.94 (0.86–0.97)
Berinson2021	68	2	4	75	0.94 (0.87–0.98)	0.97 (0.91–0.99)
Burrack-Lange2018	108	10	4	2362	0.96 (0.91–0.99)	1.00 (0.99–1.00)
Camelena2023	70	6	2	1976	0.97 (0.90–0.99)	1.00 (0.99–1.00)
ElSherif2022	40	13	0	41	1.00 (0.91–1.00)	0.76 (0.63–0.85)
Fhooblall2016	54	0	4	22	0.93 (0.84–0.97)	1.00 (0.85–1.00)
Holma2022	36	1	0	43	1.00 (0.90–1.00)	0.98 (0.88–1.00)
Kang2020	30	0	1	58	0.97 (0.84–0.99)	1.00 (0.94–1.00)
Kim2024	39	0	0	1248	1.00 (0.91–1.00)	1.00 (1.00–1.00)
Oberhettinger2020	86	0	12	38	0.88 (0.80–0.93)	1.00 (0.91–1.00)
Otlu2015	12	0	0	30	1.00 (0.76–1.00)	1.00 (0.89–1.00)
Peri2022	24	0	0	36	1.00 (0.86–1.00)	1.00 (0.90–1.00)
Rhoads2023	653	19	5	4693	0.99 (0.98–1.00)	1.00 (0.99–1.00)
Rule2021	41	0	0	583	1.00 (0.91–1.00)	1.00 (0.99–1.00)
Smith2023	90	0	15	168	0.86 (0.78–0.91)	1.00 (0.98–1.00)
Sparks2021	12	0	0	27	1.00 (0.76–1.00)	1.00 (0.88–1.00)
Verhoeven2018	82	0	3	55	0.96 (0.90–0.99)	1.00 (0.93–1.00)
Verroken2019	64	0	11	74	0.85 (0.76–0.92)	1.00 (0.95–1.00)
Summary estimate (bivariate glmer)					**0.97 (0.94–0.98)**	**1.00 (0.99–1.00)**



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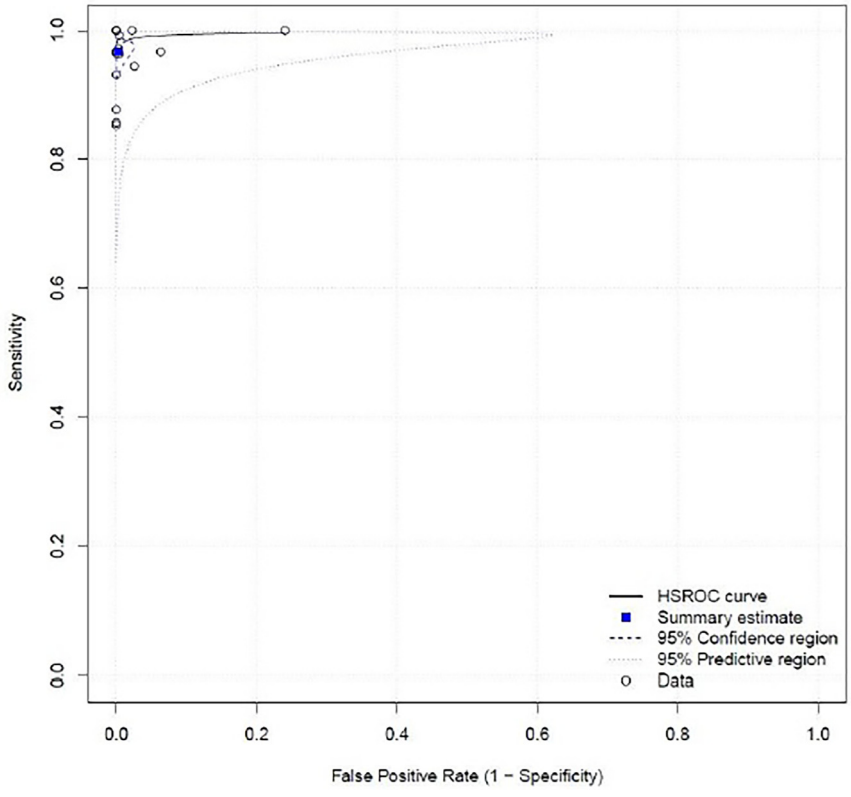


Fig. 2. Summary sensitivity-specificity (A) and summary receiver operating characteristic (SROC) (B) for Gram-positive cocci (GPC) identification.

3.5. Clinical outcomes

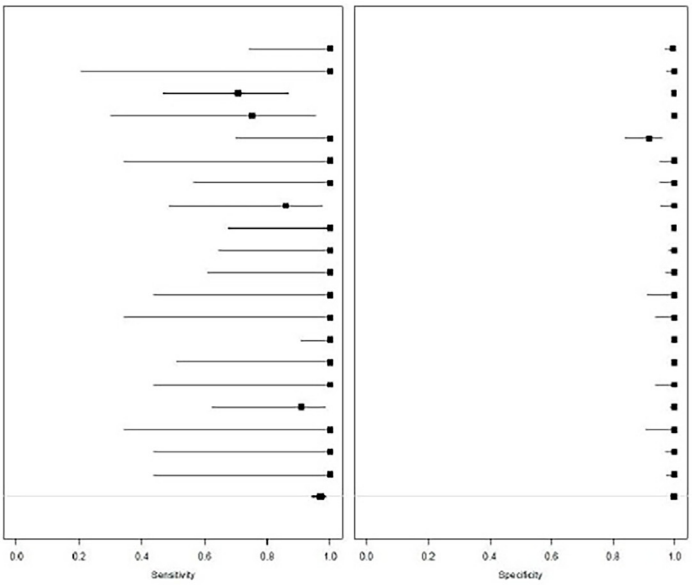
3.5.1. Primary outcome

Data on mean time to pathogen identification were available from 11 studies including 9398 patients. The mean difference was -21.43 hours (95% CI: -29.15 to -13.72; $P < 0.0001$; $I^2 = 99\%$), as shown in Table 1 and Supplementary Fig. 13, without evidence of publication bias (Supplementary Fig. 14). Data on mean time to pathogen optimal/adjusted/appropriate treatment were available from 18 studies including 10 497 patients. The pooled mean difference was -13.78 hours (95% CI: -18.06 to -9.5; $P < 0.0001$; $I^2 = 97\%$), as shown in Table 1 and Supplementary Fig. 15, without evidence of publication bias (Supplementary Fig. 16). Subgroup analysis by type of study (RCT vs. non-RCT), type of rapid diagnosis (multiplex PCR vs. MALDI-TOF vs. other [mainly rapid phenotypic tests]), type of hospitalization (ICU vs. non-ICU vs. mixed), type of pathogen (Gram-negative vs. Gram positive vs. mixed) and presence of AMS program (AMS vs. non-AMS) did not eliminate heterogeneity and the meta-analytical outcome was constant among subgroups for both time to pathogen identification and time to optimal/adjusted/appropriate therapy (Fig. 4, Supplementary Figs. 17-26).

The overall certainty in the meta-analytical result was moderate, due to the inclusion of almost 50% of the evidence coming

A

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Altun2013	11	1	0	188	1.00 (0.74–1.00)	0.99 (0.97–1.00)
Berinson2021	1	0	0	148	1.00 (0.21–1.00)	1.00 (0.97–1.00)
Burrack-Lange2018	12	4	5	1635	0.71 (0.47–0.87)	1.00 (0.99–1.00)
Camelena2023	3	0	1	1060	0.75 (0.30–0.95)	1.00 (1.00–1.00)
ElSherif2022	9	7	0	78	1.00 (0.70–1.00)	0.92 (0.84–0.96)
Fhooblall2016	2	0	0	78	1.00 (0.34–1.00)	1.00 (0.95–1.00)
Holma2022	5	0	0	75	1.00 (0.57–1.00)	1.00 (0.95–1.00)
Kang2020	6	0	1	82	0.86 (0.49–0.97)	1.00 (0.96–1.00)
Kim2024	8	2	0	809	1.00 (0.68–1.00)	1.00 (0.99–1.00)
McCoy2016	7	0	0	193	1.00 (0.65–1.00)	1.00 (0.98–1.00)
Oberhettinger2020	6	0	0	130	1.00 (0.61–1.00)	1.00 (0.97–1.00)
Otlu2015	3	0	0	39	1.00 (0.44–1.00)	1.00 (0.91–1.00)
Peri2022	2	0	0	58	1.00 (0.34–1.00)	1.00 (0.94–1.00)
Rhoads2023	37	3	0	7478	1.00 (0.91–1.00)	1.00 (1.00–1.00)
Rule2021	4	0	0	386	1.00 (0.51–1.00)	1.00 (0.99–1.00)
Saito2018	3	0	0	57	1.00 (0.44–1.00)	1.00 (0.94–1.00)
Smith2023	10	0	1	262	0.91 (0.62–0.98)	1.00 (0.99–1.00)
Sparks2021	2	0	0	37	1.00 (0.34–1.00)	1.00 (0.91–1.00)
Verhoeven2018	3	0	0	137	1.00 (0.44–1.00)	1.00 (0.97–1.00)
Verroken2019	3	0	0	146	1.00 (0.44–1.00)	1.00 (0.97–1.00)
Summary estimate (bivariate glmer)					**0.97 (0.94–0.98)**	**1.00 (0.99–1.00)**



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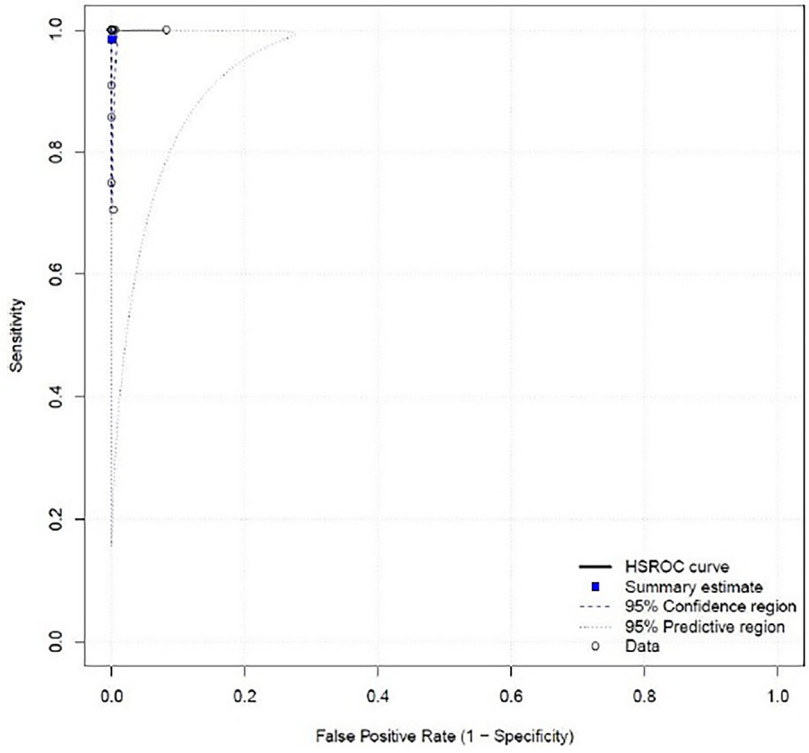

Fig. 3. Summary sensitivity-specificity (A) and summary receiver operating characteristic (SROC) (B) for yeast identification.

Table 1
Summary of pooled estimates for primary and secondary clinical outcomes.

Outcome	Studies (*n*)	Patients (*n*)	Mean difference (95% CI) or OR (95% CI)	*P*-value	I^2 (%)
Primary					
Time to pathogen identification (hours)	11	9398	-21.43 (-29.15 to -13.72)	<0.0001	99
Time to pathogen optimal/adjusted/appropriate treatment (hours)	18	10 497	-13.78 (-18.06 to -9.5)	<0.0001	97
Secondary					
Time to de-escalation (hours)	4	2581	-17.16 (-27.32 to -7.0)	0.0009	99
30-d mortality	28	12244	0.97 (0.88-1.07)	0.0792	23
Length of hospital stay (days)	22	12555	-0.13 (-1.34 to 1.09)	0.8365	92
Length of antimicrobial treatments (days)	3	5906	-0.91 (-2.26 to 0.44)	0.1849	95
Total cost of hospitalization (€)	5	7824	-2510.15 (-4992.90 to -27.39)	0.0475	96

Abbreviations CI: confidence interval, OR: odds ratio.

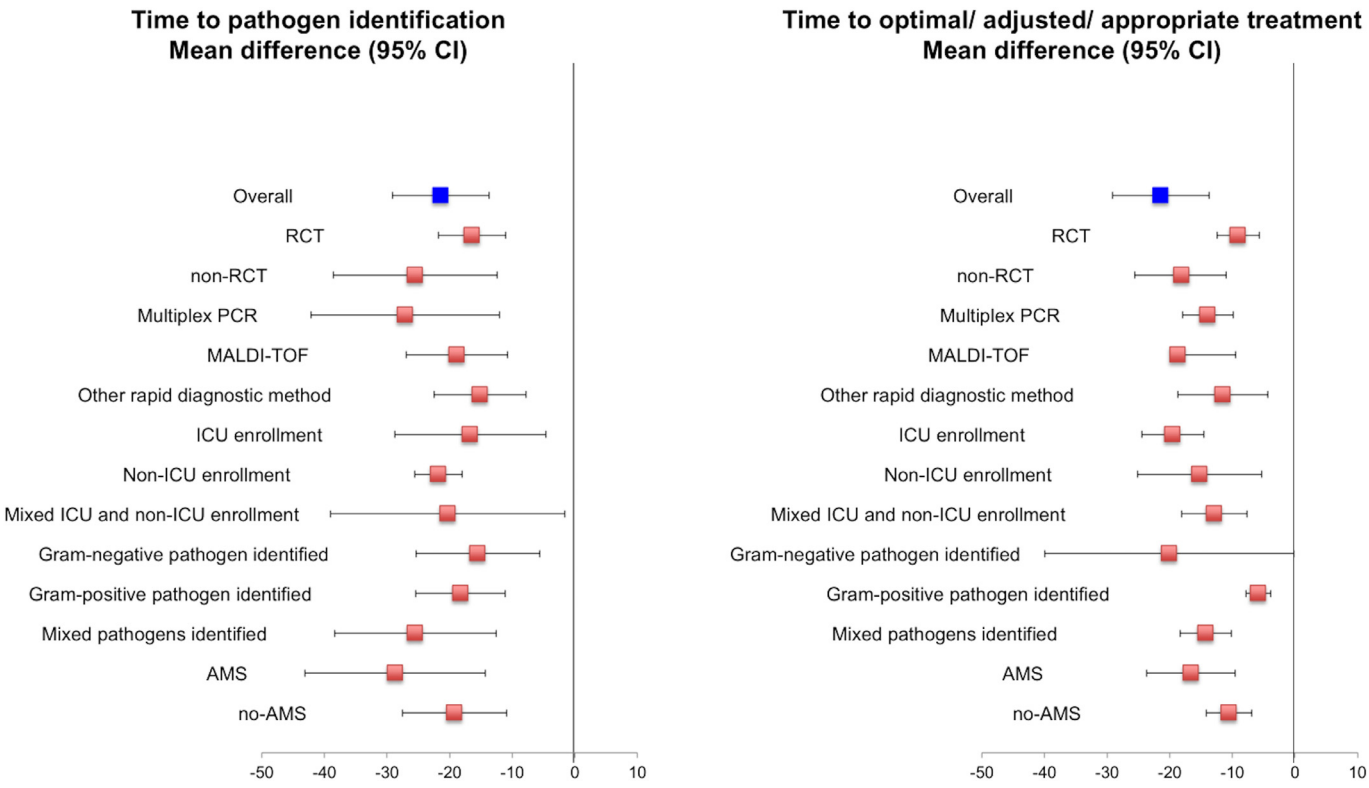

Fig. 4. Summary of subgroup analyses for the effect of rapid/molecular diagnostic tests in bactaeremia/sepsis on (a) the mean time to pathogen identification (in hours) and (b) the mean time to optimal/adjusted/appropriate treatment (in hours). Abbreviations: CI confidence interval; ICU intensive care unit; MALDI-TOF MS Matrix-assisted laser desorption/ionization mass spectrometry; RCT randomized clinical trial.

from observational trials, which lack randomization and high observed heterogeneity (Supplementary Fig. 27).

3.5.2. Secondary outcomes

Among secondary outcomes, rapid/molecular diagnostic methods for pathogen identification reduced time to de-escalation (mean difference -17.16 hours; 95% CI: -27.32 to -7.0; I^2 = 99%; 4 studies including 2581 patients) and total cost of hospitalization (mean difference -2510.15 €; 95% CI: -4992.90 to -27.39; I^2 = 96%; 5 studies including 7824 patients) but did not affect the length of hospital stay (mean difference -0.13 d; 95% CI: -1.34 to 1.09; I^2 = 92%; 22 studies including 12 555 patients) and the length of antimicrobial treatment (mean difference -0.91 d; 95% CI: -2.26 to 0.44; I^2 = 95%; 3 studies including 5906 patients); Table 1, Supplementary Figs. 28-35. Regarding 30-d mortality, rapid/molecular diagnostic methods for pathogen identification showed a trend for lower mortality compared with SOC microbiological methods, but this did not reach statistical significance (OR 0.97; 95% CI: 0.88-1.07; I^2 = 23%; 28 studies including 12 244 patients); Table 1, Supplementary Figs. 36-37. As MALDI-TOF was considered as interventional arm in some studies and SOC procedure in others, a post hoc sensitivity analysis was performed excluding all studies with MALDI-TOF as intervention [78,86,90,92,94], providing similar results for primary and secondary outcomes (Supplementary Table 7).

The overall certainty in the meta-analytical secondary results for length of hospital stay and cost was very low and that for mortality was low, respectively (Supplementary Fig. 38).

3.6. Recommendations based on microbiological outcomes

All following recommendations are summarized in Table 2.

3.6.1. Recommendation 1 –PBC panels

We suggest using PBC panels as an adjunct to SoC identification to provide earlier pathogen identification and resistance gene detection once a blood culture turns positive (Strength of recommendation: Conditional; CoE: Moderate).

Justification

- Pooled sensitivity and specificity are very high (≈96-98% and ≈99-100%).
- CoE is downgraded mainly by study-level risk of bias.
- PBC panels cannot replace SoC, which remains essential for full pathogen spectrum coverage, phenotypic susceptibility testing, and epidemiology vigilance.

3.7. Recommendations based on clinical outcomes

All following recommendations are summarized in Table 2.

3.7.1. Recommendation 2 – time to optimal therapy

We recommend using PBC panels *to decrease the time to optimal or targeted therapy*.

(Strength of recommendation: Conditional; CoE: Moderate)

Justification

- Multiple systematic reviews demonstrate a statistically significant reduction in time to organism identification and optimal therapy initiation compared with SoC.
- Earlier therapy adjustments are associated with improved clinical decision-making and lower rates of prolonged exposure to broad-spectrum antimicrobials.
- The effect is observed across ICU and non-ICU settings, reinforcing generalizability.

Table 2
Summary of microbiological and clinical recommendations on the appropriate use of rapid/molecular diagnostic tests in patients hospitalized with sepsis/bloodstream infections/severe infections.

Recommendation	*Strength*	*Certainty*	*Key Clinical Use*
PBC DTA	Conditional	⊕⊕⊕◯ (Moderate)	Enables rapid pathogen identification and early therapy optimization; must be combined with SoC
Time to optimal therapy	Conditional	⊕⊕⊕◯ (Moderate)	Significantly shortens time to diagnosis and appropriate therapy
Mortality impact	Conditional (neutral effect)	⊕⊕◯◯ (Low)	No mortality change; supports safe early therapeutic decisions
Length of stay	Conditional	⊕◯◯◯ (Very Low)	Evidence suggests reduction under AMS guidance

AMS, antimicrobial stewardship; PBC, positive blood culture; SoC, standard of care.

3.7.2. Recommendation 3 – mortality impact

We suggest informing clinicians that PBC panel–guided management *does not significantly change mortality compared with SOC but is safe* and supports earlier optimization of therapy.

(Strength of recommendation: Conditional; CoE: Low)

Justification

- Pooled outcome data from meta-analyses consistently show no statistically significant difference in 30-d all-cause mortality between rapid diagnostic and SoC groups.
- No increase in adverse clinical outcomes or inappropriate therapy rates, supporting the clinical safety of panel-guided interventions.
- These results suggest that PBC panels primarily exert their effect through improvement of care processes, including accelerated pathogen identification and antimicrobial optimization, which indirectly contribute to better resource utilization rather than direct mortality

3.7.3. Recommendation 4 – length of stay

We suggest that PBC panel use *may contribute to a shorter hospital length of stay* as part of AMS-guided management.

(Strength of recommendation: Conditional; CoE: Very Low)

Justification

- Observational studies report a trend toward reduced length of stay, particularly when PBC results are integrated into structured AMS interventions.
- Heterogeneity of study designs and patient populations limits certainty, but available evidence supports a favourable direction of effect.

4. Discussion

In this systematic review and meta-analysis, we showed that rapid molecular pathogen identification methods have a very high diagnostic performance with pooled bivariate sensitivity for pathogen identification ranging from 0.97 to 0.98 (95% CIs approximately 0.94-0.99) and specificity consistently 0.99-1.00 across GNR, GPC, and yeasts, reduces time to pathogen identification, optimal antimicrobial treatment and de-escalation, as well as total hospitalization costs compared with SOC microbiological techniques without a risk for higher short-term mortality.

Appropriate patient management as well as informed clinical decision-making with a shorter turnaround time is not possible without high DTA of rapid molecular testing, which serves as the technical foundation for potential improvements in clinical outcomes [105]. Our pooled estimates for the primary outcomes closely mirror those reported by Wang et al., [3] with similarly high sensitivity and near-perfect specificity across GNR, GPC, and yeast targets, supporting the robustness and reproducibility of these findings. Furthermore, our subgroup analysis indicated that diagnostic accuracy for pathogen identification did not differ between studies restricted to monomicrobial blood cultures and those reporting mixed datasets including polymicrobial cultures. This finding suggests that the performance of rapid molecular panels at the target-group level is robust to culture complexity. The absence of effect modification likely reflects the group-level estimand used in this analysis and supports the applicability of pooled accuracy estimates to real-world clinical settings. Rapid diagnostic molecular tests for pathogen identification rely on either culture-based or culture-independent methods [106]. A recent large meta-analysis concluded that culture-independent panels currently lack the sensitivity to be recommended as a replacement to blood cultures for detecting BSI pathogens [107]. Compared with our primary outcomes, inclusion of culture-independent rapid molecular tests would have required a different estimand (no clear denominator for TNs at target level; cannot construct valid 2×2 tables comparable to culture-dependent panels).

In the secondary analyses of AMR gene detection, pooled sensitivity was lower for GNR than for GPC (0.93 vs. 0.98). A plausible biological explanation for this difference is the substantially greater diversity and complexity of resistance mechanisms in GNR. In contrast to GPC pathogens, where resistance to key antimicrobial classes is often driven by a limited number of well-characterized genetic determinants (e.g. mecA in staphylococci or vanA/vanB in enterococci), GNR resistance frequently arises from a combination of gene-encoded mechanisms and gene-independent processes, including porin loss, efflux pump overexpression, and regulatory mutations affecting chromosomal genes [108]. As a result, molecular assays that detect predefined resistance genes may function as an incomplete proxy for phenotypic susceptibility, especially in GNR pathogens, leading to lower apparent sensitivity when compared with phenotypic reference standards. This structural limitation of gene-based AMR detection is consistent with the recent diagnostic accuracy meta-analysis by Wang et al., [3] and likely contributes to the observed difference between GNR and GPC AMR detection performance. Combining molecular tests with SOC provides additional confidence to clinicians for making an informed decision on optimal antimicrobial treatment in light of AMR. We do caution though that clinicians should interpret PPV/NPV in regard to their unit's current prevalence or the individual patient's pretest probability [105].

Time to appropriate antimicrobial treatment is key when facing severe infections; this is of particular importance in the current era of increasing AMR. In a recent study of non-neutropenic patients with hospital-onset enterococcal BSIs, delayed therapy of $\geq$48 hours was associated with a threefold increase in 30-d mortality even after adjustment for severity of illness and comorbidity. Vancomycin resistance was the only independent predictor of delayed therapy underscoring the potential role of rapid diagnostic testing for early identification of vancomycin-resistant enterococci [109]. Similarly, targeted use of novel β-lactams/β-lactamase inhibitors within 48 hours of infection onset has been associated with favourable outcomes in Gram-negative MDR infections [110,111]. Reducing the time to response (i.e. the time from the collection of microbiological specimens to the identification of the causative pathogen) and thus the time to antimicrobial treatment by means of rapid diagnostic tests is expected to improve the outcome of patients with severe infections, even those caused by MDR

organisms but also limit unnecessary antimicrobial overconsumption.

Anton-Vazquez et al. have performed a meta-analysis on the clinical impact of rapid antimicrobial susceptibility testing vs. SOC microbiology for Cochrane in 2021 including 6 RCTs [112]. The authors did not succeed in demonstrating that the theoretical benefits of rapid susceptibility testing have improved mortality, time to discharge, or time to appropriate antibiotics. Since then, new studies have been conducted as rapid/molecular tests are becoming widely used. Observational studies could not be excluded from this analysis as the impact of rapid susceptibility testing on AMS programs is usually evaluated in before-after studies. In our analysis, time to pathogen identification and time to appropriate/adjusted/optimal treatment was reduced compared with SOC in contrast to previously published evidence and a trend towards lower mortality was also observed. High heterogeneity in the meta-analytical result has to be acknowledged, but the enormous mean difference of the effect measure should also be seriously taken into consideration.

Individual studies report that most of the clinical benefit in targeted treatment and AMS is observed in patients with documented BSI and PBCs. Similarly, in individual studies, most of the clinical benefit is observed when rapid molecular diagnostics are applied in the context of an established AMS program with infectious diseases consultation; this was not validated by our subgroup meta-analytic outcome as benefit was observed even in the absence of an adjunct AMS program [77,113,114]. Recently, the American Society for Microbiology issued practice guidelines for the diagnosis of BSI using rapid tests and strongly recommended their use combined with active communication to decrease the time to targeted therapy and length of stay but also mortality, although the strength of the evidence for the impact on mortality was low [115].

Our study has some limitations. In the majority of records screened, the main objective was DTA of rapid/molecular methods in pathogen identification; clinical outcomes reflecting the impact of these tests in everyday practice are underreported, mainly in observational pre-post quasi-experimental studies but not RCTs, introducing possible bias and heterogeneity. Another limitation is the high heterogeneity observed in most analyses, particularly for time metrics and cost; this may be attributed to different levels of AMR, differences in health care settings (ward/ICU, low/high-income countries), and differences in outcome definitions and reporting intervals in individual studies. As an example, the definition of optimal/adjusted/appropriate treatment varied in individual studies as well as the starting point (from hospital admission or blood draw or Gram staining etc.) and stopping point (until preliminary results or complete pathogen reporting and complete antibiogram).

Our extensive literature search also revealed current research gaps on the use of molecular tests. Only a very limited number of studies reported on total antimicrobial consumption, and there was considerable heterogeneity in the expression of effect measures (length of treatment or defined daily doses). Data are lacking on long-term mortality, patient-reported outcomes, quality of life, and/or functional recovery, and data are sparse on other important outcomes reflecting antimicrobial overconsumption, such as development of antimicrobial resistance (two studies: 72,78), *Clostridioides difficile* infection (five studies: 77,78,85,93,102) and antimicrobial drug toxicity (one study: 78). All these outcomes and gaps should be a priority in future clinical trials and investigations and are of paramount importance in countries with high levels of AMR like Greece. Moreover, it should be acknowledged that certain low- and middle-income countries (LMICs) may have limited access to such tests underlying existing barriers between uptake and implementation. Even in high-income settings and due to the considerable costs associated with these tests, it is crucial for laboratories and health care services to prioritize diagnostic stewardship when incorporating these panels into the laboratory [116].

While molecular diagnostics alone are not a panacea, their integration into care pathways, especially when supported by timely clinical communication has clear value in managing BSIs and sepsis. Clinicians, microbiologists, and policymakers should prioritize the deployment of rapid tests in PBC workflows, the development of clear AMS workflows that actively communicate results to decision-makers, and the evaluation of cost-effectiveness and resistance impact in multicenter RCTs. Diagnostic stewardship frameworks that ensure test results are interpreted and acted on promptly should be adopted.

Our findings reinforce the growing consensus that early actionable diagnostics, particularly from PBCs, are essential to sepsis care. When combined with artificial intelligence models, these tools can integrate molecular, clinical, and physiological data to enhance prediction accuracy, guide personalized treatment, and improve patient outcomes. The true clinical benefit hinges on thoughtful implementation, especially through active result communication and integration into broader stewardship frameworks. Future research must focus not only on refining test accuracy but also on evaluating long-term outcomes, resistance trajectories, and strategies to ensure their effective real-world use.

Funding: This study was funded by an unrestricted educational grant by BioMérieux SA.

Declaration of competing interests: KA reports honoraria for lectures from Gilead, Pfizer, MSD, GSK/ViiV Hellas, and support for attending meetings and/or travel from Gilead, Pfizer, MSD, Norma Hellas, Sobi as well as participation on a Data Safety Monitoring Board or Advisory Board for Gilead. GV reports honoraria for lectures from Gilead, and support for attending meetings and/or travel from Autobio Diagnostics Co., Ltd. IK reports honoraria for lectures from Pfizer, Menarini. AT reports honoraria for lectures from Pfizer, Abbott. EJGB reports grants from Abbott Products Operations, bioMérieux Inc, Gentian, MSD, PHC Europe BV, Swedish Orphan Biovitrum AB (publ) (Sobi) paid to the Hellenic Institute for the Study of Sepsis and from InCyte, MSD, Novartis, Sanofi, UCB paid to the National and Kapodistrian University of Athens; consulting fees from Sobi paid to the National and Kapodistrian University of Athens; honoraria for lectures from Abbott Products Operations AG, bioMérieux Inc and ThermoFisher Brahms GmbH, paid to the National and Kapodistrian University of Athens. The other authors declare no conflicts of interest. Given their roles as editors for IJAA, EG-B and AT had no involvement in the peer-review of this article and had no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to another journal editor.

Ethical approval: Not required

Author contributions: EK performed a literature search, data analysis, and drafted the manuscript. KP performed a literature search and data analysis.S M, KG, KA, IK, MO, and GV critically revised the manuscript. EJGB and AT supervised the project, acquired funding, and critically revised the manuscript.

Sequence information: Not applicable

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ijantimicag.2026.107937.

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