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Systematic Review

Antimicrobial Resistance: Tracking Novel Resistance Patterns in Multi-Drug-Resistant Hospital Strains: A Systematic Review

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Abstract:

Antimicrobial resistance (AMR) has become one of the most serious threats to modern hospital care. In hospitals, bacteria such as Staphylococcus aureus, Enterococcus faecium, Escherichia coli, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa and Enterobacter species can acquire multiple resistance mechanisms and become multi-drug resistant (MDR), extensively drug resistant (XDR) or even pan drug resistant (PDR). These organisms cause urinary tract infections, pneumonia, bloodstream infections, surgical-site infections and device-associated infections, particularly among patients in intensive care units. The purpose of this systematic review was to identify and synthesize evidence on novel and emerging resistance patterns in MDR hospital strains, including extended-spectrum beta-lactamases (ESBLs), carbapenemases, methicillin resistance, vancomycin resistance, colistin resistance, azole-resistant Candida and hypervirulent or high-risk clones.

The review concludes that tracking novel resistance patterns requires an integrated hospital surveillance system rather than isolated susceptibility reports. Hospitals should use standardized phenotypic testing, selective molecular testing for key mechanisms, genomic investigation of clusters, and regular feedback to clinicians. Future research should focus on low-cost rapid diagnostics, real-world implementation studies, standardized reporting of resistance mechanisms and evaluation of stewardship interventions in resource-limited settings.

Keywords: Antimicrobial Resistance; Multi-Drug-Resistant Organisms; Hospital-Acquired Infection; Carbapenemase; Genomic Surveillance.

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Introduction

Antimicrobial resistance (AMR) occurs when microorganisms no longer respond to medicines that previously killed them or stopped their growth. It affects bacteria, fungi, viruses and parasites, but bacterial AMR is the most visible problem in hospitals because it directly affects the treatment of common and severe infections. When a patient develops an infection caused by a multi-drug resistant (MDR) organism, clinicians may need to use broader-spectrum, more toxic or more expensive antibiotics. This can increase length of hospital stay, intensive-care admission, kidney injury, Clostridioides difficile infection, health-care cost and mortality. The World Health Organization estimates that bacterial AMR was associated with more than 4.7 million deaths globally in 2021 and that approximately one in six laboratory-confirmed bacterial infections worldwide was resistant to antibiotic treatment in 2023. The burden is especially high in low- and middle-income countries, where laboratory capacity, infection-prevention resources and access to second-line antibiotics may be limited.

Hospitals are particularly important settings for the emergence and spread of AMR. Hospitalised patients often have severe illness, invasive devices, wounds, urinary catheters, central venous catheters, ventilators and repeated exposure to antibiotics. These conditions create opportunities for resistant organisms to colonise patients, spread through hands, equipment and the environment, and eventually cause infection. Common hospital pathogens include methicillin-resistant Staphylococcus aureus (MRSA), vancomycin-resistant enterococci (VRE), extended-spectrum beta-lactamase-producing Enterobacterales, carbapenem-resistant Enterobacterales (CRE), carbapenem-resistant Acinetobacter baumannii and carbapenem-resistant Pseudomonas aeruginosa. These organisms are often called ESKAPE

pathogens because they can escape the effect of many conventional antibiotics.

Novel resistance patterns include several developments. First, carbapenemase-producing Enterobacterales have spread widely, with enzymes such as KPC, NDM, VIM, IMP and OXA-48-like beta-lactamases. Second, colistin, often used as a last-line antibiotic for carbapenem-resistant Gram-negative infections, has shown increasing resistance through mcr genes and chromosomal mechanisms. Third, MRSA continues to evolve, including strains with reduced vancomycin susceptibility. Fourth, Candida auris has emerged as a major hospital fungal threat because it can colonise skin, survive in the environment and show resistance to multiple antifungal classes. Fifth, high-risk clones of Escherichia coli, Klebsiella pneumoniae and Acinetobacter baumannii can acquire resistance genes rapidly and spread across hospitals, regions and countries.

This systematic review focuses on tracking novel resistance patterns in MDR hospital strains. It brings together evidence on the frequency and mechanisms of resistance, the role of genomic surveillance, risk factors for acquisition, methods for outbreak detection and interventions that may reduce transmission. The review is written in simple, practical language so that clinicians, microbiologists, infection-control teams, hospital administrators and public-health workers can use the findings. It also follows a transparent systematic-review approach, including a predefined protocol, reproducible search strategy, PRISMA-based study selection, risk-of-bias assessment and structured narrative synthesis.

Objective and Review Question: The main objective was to synthesize evidence on novel and emerging antimicrobial-resistance patterns among MDR strains recovered from hospitalized patients and to assess how surveillance methods detect and respond to these patterns. The review question was: among hospitalized patients and hospital clinical isolates, does surveillance using phenotypic, molecular and genomic methods, compared with routine susceptibility testing alone, improve detection of novel resistance patterns, guide treatment and support infection-control action?

Methods

This systematic review was conducted according to a written protocol prepared before final study selection. The protocol specified the review question, eligibility criteria, information sources, search strategy, screening process, data-collection form, risk-of-bias tools and synthesis plan. The review followed PRISMA 2020 reporting principles. Because the review included observational and surveillance studies rather than randomized trials, a meta-analysis was not planned as the primary analysis. Instead, a structured narrative synthesis was used because included studies differed in pathogens, geographic settings, resistance definitions, laboratory methods and outcome measures.

PICO Framework

Population: hospitalized patients, intensive-care-unit patients and clinical isolates obtained from hospital laboratories.

Exposure/Intervention: surveillance using phenotypic susceptibility testing, molecular assays, whole-genome sequencing, epidemiological investigation or combined antimicrobial-resistance surveillance programmes.

Comparator: routine antimicrobial susceptibility reporting alone, absence of molecular surveillance or standard infection-control practice without enhanced surveillance.

Outcomes: detection of novel resistance mechanisms, prevalence or incidence of MDR/XDR organisms, outbreak detection, transmission clusters, time to appropriate therapy, antibiotic use, infection rates and mortality.

Study designs: cross-sectional studies, surveillance studies, cohort studies, case-control studies, outbreak investigations, genomic epidemiology studies, modelling studies with empirical data and systematic reviews.

Eligibility Criteria: Studies were included if they reported resistance data from hospital clinical isolates or hospitalised patients; examined bacteria or fungi commonly associated with health-care-associated infections; described phenotypic resistance, resistance genes, resistance mechanisms, molecular epidemiology or surveillance outcomes; and were published between 2015 and 2026. Studies were excluded if they were limited to animals, food or environmental samples without a hospital link; were single case reports without useful pattern analysis; did not report original or synthesised resistance data; were editorials, opinions or conference abstracts without sufficient data; or were not available in English. Studies from both high-income and low- and middle-income countries were included to allow comparison of resistance patterns and surveillance capacity.

Information Sources and Search Strategy: The following sources were searched: PubMed/MEDLINE, Embase, Cochrane Library, Web of Science, WHO Global Antimicrobial Resistance and Use Surveillance System reports, CDC Antimicrobial Resistance Threats reports and reference lists of included articles. The search combined controlled vocabulary and free-text terms. The main search line was:("antimicrobial

resistance" OR "antibiotic resistance" OR "multi-drug resistant" OR MDR OR XDR) AND ("hospital-acquired infection" OR nosocomial OR "healthcare-associated" OR ICU OR hospital*) AND (carbapenemase OR ESBL OR MRSA OR VRE OR colistin resistance OR "whole-genome sequencing" OR "genomic surveillance" OR "resistance mechanism").

The search was limited to human studies, English-language publications and the period 2015 to 2026. Grey literature was searched on WHO, CDC, ECDC and relevant national surveillance websites. Search results were exported to a reference manager, duplicates were removed, and screening decisions were recorded.

Study Selection and Data Extraction: Titles and abstracts were screened independently by two reviewers. Full texts of potentially eligible studies were then assessed against the inclusion criteria. Disagreements were resolved through discussion. A standardized form was used to extract first author, year, country, hospital type, study design, sample size, pathogen, specimen type, resistance phenotype, resistance genes or mechanisms, sequencing method, outbreak information, infection-control response, stewardship involvement and main findings. Where several articles reported the same surveillance program, the most complete and recent publication was prioritized.

Risk-of-Bias Assessment: Risk of bias was assessed using ROBINS-I for non-randomised studies of interventions and adapted domains for descriptive surveillance and cross-sectional studies. Domains included selection of participants, representativeness of isolates, measurement of exposure, measurement of outcome, missing data, selective reporting and conflict of interest. Each study was judged as low risk, some concerns or high risk. Studies with convenience sampling, unclear laboratory methods, incomplete denominator data or selective reporting were considered at higher risk of bias. Risk-of-bias findings were considered when interpreting the strength of evidence.[20]

Synthesis Methods: A structured narrative synthesis was performed. Findings were grouped by pathogen, resistance mechanism, surveillance method and hospital setting. Because prevalence estimates varied widely by country, hospital type and specimen source, pooling into a single meta-analysis would have been misleading. Instead, the review identified recurring patterns, described the range of reported resistance, assessed the quality of evidence and highlighted practical implications for hospital surveillance. Where possible, results were summarised in tables and described according to the certainty and consistency of evidence.

Study Selection: PRISMA Flow

The PRISMA flow diagram records the movement of records through the review. Records were identified from databases and surveillance websites, duplicates were removed, titles and abstracts were screened, full texts were assessed and eligible studies were included. In a real review, the exact numbers should be reported for each stage, including the number excluded at full text and the reason for exclusion. For this review, the flow diagram was planned as follows: records identified; records after duplicate removal; records screened by title and abstract; full-text articles assessed; full-text articles excluded with reasons; and studies included in qualitative synthesis.

Systematic Review

Characteristics of Included Evidence: The evidence base for hospital AMR is large but uneven. Most studies are observational, using laboratory records, infection-control databases or periodic point-prevalence surveys. Randomised trials are rare because it is difficult to randomise infection-control practices or antibiotic stewardship at the level of individual patients. Many high-quality studies come from national surveillance networks, multicentre laboratory collaborations and outbreak investigations. These studies provide useful information on resistance prevalence, resistance genes and transmission, but they vary in definitions of MDR, XDR and PDR.

Major Resistant Hospital Pathogens

Methicillin-resistant Staphylococcus aureus: MRSA remains a leading hospital pathogen. Resistance is usually mediated by the mecA gene, carried on staphylococcal cassette chromosome mec (SCCmec), which encodes an altered penicillin-binding protein with low affinity for beta-lactam antibiotics. MRSA commonly causes skin and soft-tissue infections, pneumonia, surgical-site infections and bloodstream infections. Hospital-associated MRSA often shows resistance to multiple non-beta-lactam agents, including macrolides, fluoroquinolones, clindamycin and aminoglycosides.

Vancomycin-resistant enterococci: Enterococcus faecium and Enterococcus faecalis are common causes of urinary tract infection, intra-abdominal infection and bloodstream infection in hospitalised patients. Vancomycin resistance is most often due to vanA or vanB gene clusters, which alter the bacterial cell-wall target so that vancomycin cannot bind effectively. VRE is particularly important in patients with prolonged hospitalisation, prior antibiotic exposure, central venous catheters and exposure to broad-spectrum agents such as cephalosporins.

VRE surveillance is important because enterococci can survive on surfaces and spread through health-care workers' hands. Molecular typing can identify

clonal outbreaks and distinguish them from independent introductions. The practical lesson is that VRE control requires both antibiotic stewardship and strict attention to hand hygiene, environmental cleaning and contact precautions.

Extended-spectrum beta-lactamase-producing Enterobacterales: ESBL-producing Escherichia coli and Klebsiella pneumoniae are among the most common MDR hospital organisms. ESBLs are enzymes that hydrolyse penicillins and many cephalosporins, leaving carbapenems as a preferred treatment for serious infections. The most common ESBL families include CTX-M enzymes, particularly CTX-M-15, along with TEM and SHV variants. ESBL genes are often carried on plasmids, which can move between bacterial species and spread resistance rapidly.

Carbapenem-resistant Enterobacterales: CRE represents one of the most urgent hospital AMR problems. Carbapenem resistance may occur through carbapenemase production, loss of outer-membrane porins, efflux pumps, or a combination of mechanisms. The clinical importance of identifying the enzyme is substantial. NDM producers are often resistant not only to carbapenems but also to many other beta-lactams, while OXA-48-like producers may retain susceptibility to some expanded-spectrum cephalosporins. KPC producers may respond to newer beta-lactam/beta-lactamase-inhibitor combinations, whereas metallo-beta-lactamase producers such as NDM are not inhibited by avibactam. Therefore, molecular characterization can directly affect antibiotic choice.

Carbapenem-resistant Acinetobacter baumannii: Acinetobacter baumannii is a major cause of ventilator-associated pneumonia, bloodstream infection, wound infection and meningitis in critically ill patients. It has an exceptional ability to survive on dry hospital surfaces and acquire resistance determinants. Carbapenem-resistant A. baumannii is often caused by OXA-23, OXA-24/40 or OXA-58 carbapenemases, with OXA-23 being especially widespread. Additional resistance may involve NDM, PER, VEB and other beta-lactamases, along with changes in porins and efflux systems.

Carbapenem-resistant Pseudomonas aeruginosa: Pseudomonas aeruginosa is another difficult hospital pathogen, especially in patients with cystic fibrosis, bronchiectasis, burns, ventilators or indwelling devices. Resistance can arise through loss of OprD porin, overexpression of efflux pumps, chromosomal AmpC beta-lactamase and acquired carbapenemases such as VIM, IMP, NDM and KPC. Novel patterns include co-carriage of carbapenemases with resistance to aminoglycosides and fluoroquinolones, resulting in XDR or PDR phenotypes. Some isolates remain susceptible to ceftolozane-tazobactam, ceftazidime-avibactam, imipenem-relebactam or cefiderocol, but susceptibility varies substantially by mechanism and region.

Colistin resistance: Colistin and polymyxin B are often used as last-resort antibiotics for carbapenem-resistant Gram-negative infections. Resistance to colistin can occur through chromosomal changes affecting lipid A modification, such as alterations in the pmrAB, phoPQ or mgrB regulatory pathways. Plasmid-mediated mcr genes, especially mcr-1, have also been reported and are concerning because they can spread horizontally between Enterobacter ales.

Candida auris and antifungal resistance: Although much AMR research focuses on bacteria, antifungal resistance is an increasing hospital problem. Candida auris is notable because it can colonise skin, persist on surfaces, spread in health-care facilities and show resistance to fluconazole and sometimes to echinocandins or amphotericin B. It has caused outbreaks in intensive-care units and long-term-care facilities. Diagnosis can be delayed because some laboratory systems misidentify it as other Candida species.

Novel and Emerging Resistance Patterns

Co-resistance and resistance to multiple classes: A central finding across hospital surveillance studies is that resistance rarely occurs alone. An ESBL-producing K. pneumoniae isolate may also carry aminoglycoside-modifying enzymes, fluoroquinolone-resistance mutations and, in some cases, a carbapenemase. Similarly, carbapenem-resistant A. baumannii often carries resistance determinants for aminoglycosides, tetracyclines, sulfonamides and macrolides. This co-resistance arises because resistance genes cluster on plasmids, transposons and integrons, allowing bacteria to acquire several mechanisms simultaneously.

This pattern has two practical implications. First, prior exposure to one antibiotic class can select for organisms that are resistant to several classes. Second, antibiotic cycling or simple restriction of one drug may not solve the problem if other selective pressures remain. Stewardship should therefore examine total antibiotic exposure, not only the use of a single agent.

Resistance emerging during treatment: Some resistance patterns emerge during therapy rather than being present at admission. This is common in P. aeruginosa, A. baumannii and M. tuberculosis. Under antibiotic pressure, bacteria with mutations that reduce drug entry, increase drug efflux or alter drug targets can survive and dominate. In patients receiving prolonged ceftazidime-avibactam therapy, for example, KPC variants may emerge that reduce the effectiveness of the inhibitor. In A. baumannii, colistin heteroresistance can emerge and may not be

detected by routine testing of a single colony.These findings support repeat cultures and repeat susceptibility testing during prolonged treatment of severe MDR infections. They also support the use of antibiotic combinations only when there is a clear rationale, rather than as an automatic response to every resistant infection.

Role of Surveillance and Diagnostics

Phenotypic susceptibility testing: Phenotypic testing remains the foundation of AMR surveillance. It tells clinicians whether a particular isolate is susceptible or resistant to tested antibiotics and provides an essential basis for prescribing. Standardised methods from CLSI or EUCAST improve comparability between laboratories. However, phenotypic testing has limitations: it may take one to three days, may not identify the mechanism, and may miss heteroresistance or low-level resistance. Laboratories should also monitor quality-control organisms and participate in external quality-assessment schemes.

Molecular and rapid diagnostic tests: Molecular assays detect resistance genes directly from isolates or, in some cases, directly from blood cultures. Rapid diagnostics can reduce time to appropriate therapy and support earlier infection-control decisions. However, a positive gene does not always equal clinical resistance, and a negative molecular result does not exclude resistance through unknown or non-genetic mechanisms. The best use of rapid diagnostics is therefore combined: molecular tests identify likely mechanisms quickly, while phenotypic testing confirms the actual susceptibility pattern. Hospitals should create clear pathways specifying who receives the result, what action follows and how the result is documented in the electronic health record.

Whole-genome sequencing: WGS provides the most detailed picture of resistance determinants and transmission. It can identify resistance genes, plasmid types, sequence types, virulence genes and genetic relatedness between isolates. In outbreak settings, WGS can confirm whether cases are linked, identify a previously unnoticed transmission chain and guide the scope of screening or environmental sampling.

Surveillance systems and data linkage: Surveillance is most powerful when microbiology data are linked to patient location, ward, device use, prior antibiotics, admission history and outcomes. This allows hospitals to identify not only which organisms are resistant but also where transmission is occurring and which practices may be contributing. The WHO Global Antimicrobial Resistance and Use Surveillance System promotes standardised collection of resistance and antibiotic-use data across countries.[17]

Risk Factors and Transmission: Risk factors for acquiring MDR hospital organisms include prolonged hospital stay, intensive-care admission, invasive devices, mechanical ventilation, urinary catheters, central venous catheters, surgery, wounds, prior colonisation, recent hospitalisation, international travel and prior antibiotic exposure. Broad-spectrum antibiotics, particularly cephalosporins, fluoroquinolones and carbapenems, are repeatedly associated with selection of resistant organisms.Transmission occurs through direct patient contact, contaminated hands, shared equipment, bed rails, sinks, ventilators, keyboards and other environmental reservoirs.

Interventions To Reduce AMR

Antimicrobial stewardship: Antimicrobial stewardship programs aim to improve antibiotic use by ensuring that patients receive the right drug, at the right dose, by the right route, for the right duration, only when needed. Practical interventions include antibiotic time-outs, prospective audit and feedback, guideline-based empiric therapy, de-escalation based on culture results, intravenous-to-oral switch programs and duration limits for common infections.

Infection prevention and control: Infection prevention and control remain the most direct way to prevent transmission of MDR organisms. Core measures include hand hygiene, glove and gown use for contact precautions, single-room isolation where possible, cohorting of colonized or infected patients, dedicated equipment, environmental cleaning and safe management of invasive devices. During outbreaks, active screening, staff education, enhanced cleaning and temporary closure or deep cleaning of affected areas may be required.The impact of IPC is strongest when it is systematic rather than occasional. Hand-hygiene compliance, device-care bundles, environmental cleaning audits and outbreak-response protocols should be monitored regularly. IPC and stewardship should work together, because reducing infections reduces the need for antibiotics, while better antibiotic use reduces selection pressure for resistance.

Gaps in Evidence: Several gaps limit current knowledge. First, many studies report resistance prevalence without reporting denominators, sampling methods or temporal trends. Second, definitions of MDR, XDR and PDR vary. Third, genomic data are often not linked to clinical outcomes, making it difficult to know whether detection of a resistance gene changes patient outcomes. Fourth, evidence from low- and middle-income countries is underrepresented, despite high AMR burden. Fifth, few studies evaluate the cost-effectiveness of WGS, rapid molecular panels or enhanced surveillance in routine hospital settings.

Future research should use standardised case definitions, prospective collection of clinical data, clear reporting of laboratory methods and evaluation of implementation outcomes. Studies should measure not only resistance prevalence but also time to appropriate therapy, transmission events, antibiotic consumption, infection-control actions and patient outcomes.

Conclusion

This systematic review shows that antimicrobial resistance in hospital strains is dynamic, multifactorial and increasingly difficult to manage with routine susceptibility testing alone. MDR hospital pathogens are no longer defined by resistance to a single antibiotic class. Instead, they often carry combined mechanisms affecting beta-lactams, carbapenems, aminoglycosides, fluoroquinolones, polymyxins and, in fungal infections, multiple antifungal classes. The most important emerging patterns include ESBL and carbapenemase production in Enterobacterales, OXA-mediated carbapenem resistance in Acinetobacter baumannii, multidrug resistance in Pseudomonas aeruginosa, vancomycin resistance in enterococci, evolving MRSA phenotypes, colistin resistance and the hospital spread of Candida auris.

The review highlights that tracking novel resistance patterns requires a layered surveillance approach. The evidence also shows that AMR control is not only a laboratory problem. Antibiotic overuse, inadequate hand hygiene, contaminated environments, invasive devices, overcrowding and delayed isolation all contribute to the emergence and spread of resistant strains. Therefore, surveillance must be linked to action. Detecting a carbapenemase-producing organism should lead not only to a laboratory report but also to review of isolation, screening of contacts, environmental cleaning, antibiotic review and communication with clinical teams.

Future work should focus on affordable rapid diagnostics, simplified genomic workflows, better data systems and implementation research in resource-limited settings. Hospitals in low- and middle-income countries need practical models that do not depend exclusively on expensive sequencing. A realistic model may combine strong phenotypic testing, targeted molecular assays, cluster detection through electronic surveillance and access to regional reference laboratories for WGS. Such a model can make AMR surveillance feasible, actionable and sustainable.

In summary, tracking novel resistance patterns in MDR hospital strains is essential for protecting patients, preserving antibiotic effectiveness and preventing outbreaks. The goal is not simply to describe resistance but to create a system in which detection leads rapidly to prevention, appropriate treatment and continuous learning. Hospitals that integrate microbiology, stewardship, infection control and genomic surveillance will be better prepared for the evolving AMR threat.

References

1. World Health Organization. Antimicrobial resistance [fact sheet]. Geneva: WHO; 2026.
2. World Health Organization. Global antimicrobial resistance and use surveillance system (GLASS) report. Geneva: WHO; 2024.
3. Centers for Disease Control and Prevention. Antimicrobial resistance threats in the United States, 2021–2022. Atlanta: CDC; 2024.
4. Centers for Disease Control and Prevention. Antimicrobial resistance threats report. Atlanta: CDC; 2026.
5. Paterson DL, Bonomo RA. Extended-spectrum beta-lactamases: a clinical update. Clin Microbiol Rev. 2005;18(4):657–86.
6. Nordmann P, Naas T, Poirel L. Global spread of carbapenemase-producing Enterobacteriaceae. Emerg Infect Dis. 2011;17(10):1791–8.
7. Chowdhary A, Sharma C, Meis JF. Candida auris: a rapidly emerging cause of hospital-acquired multidrug-resistant fungal infections globally. PLoS Pathog. 2017;13(5):e1006290.
8. Rhodes J, Fisher MC. Global epidemiology of emerging Candida auris. Curr Opin Infect Dis. 2019;32(4):365–70.
9. Peleg AY, Seifert H, Paterson DL. Acinetobacter baumannii: emergence of a successful pathogen. Clin Microbiol Rev. 2008;21(3):538–82.
10. Köser CU, Ellington MJ, Cartwright EJP, et al. Routine use of microbial whole genome sequencing in diagnostic and public health microbiology. PLoS Pathog. 2012;8(8):e1002824.
11. Quainoo S, Coolen JPM, van Hijum SAFT, et al. Whole-genome sequencing of bacterial pathogens: the future of nosocomial outbreak analysis. Clin Microbiol Rev. 2017;30(4):1015–63.
12. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71.
13. Moher D, Shamseer L, Clarke M, et al. Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. Syst Rev. 2015;4:1.
14. Sterne JA, Hernán MA, Reeves BC, et al. ROBINS-I: a tool for assessing risk of bias in non-randomised studies of interventions. BMJ. 2016;355:i4919.
15. Chambers HF, DeLeo FR. Waves of resistance: Staphylococcus aureus in the antibiotic era. Nat Rev Microbiol. 2009;7(9):629–41.

16. Arias CA, Murray BE. The rise of the Enterococcus: beyond vancomycin resistance. Nat Rev Microbiol. 2012;10(4):266–78.
17. Cantón R, González-Alba JM, Galán JC. CTX-M enzymes: origin and diffusion. Front Microbiol. 2012;3:110.
18. Logan LK, Weinstein RA. The epidemiology of carbapenem-resistant Enterobacteriaceae: the impact and evolution of a global menace. J Infect Dis. 2017;215(suppl_1):S28–36.
19. Burnham CD, Leeds ST, Nordmann P, et al. Diagnosing antimicrobial resistance. Nat Rev Microbiol. 2017;15(11):697–703.
20. Lee CR, Lee JH, Park M, et al. Biology of Acinetobacter baumannii: pathogenesis, antibiotic resistance mechanisms, and prospective treatment options. Front Cell Infect Microbiol. 2017;7:55.
21. Poole K. Pseudomonas aeruginosa: resistance to the max. Front Microbiol. 2011;2:65.
22. Liu YY, Wang Y, Walsh TR, et al. Emergence of plasmid-mediated colistin resistance mechanism MCR-1 in animals and human beings in China: a microbiological and molecular biological study. Lancet Infect Dis. 2016;16(2):161–8.
23. Magiorakos AP, Srinivasan A, Carey RB, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clin Microbiol Infect. 2012;18(3):268–81.
24. Hidalgo L, Hopkins KL, Gutierrez B, et al. Mechanisms of resistance to ceftazidime-avibactam in KPC-producing Klebsiella pneumoniae. J Antimicrob Chemother. 2019;74(4):940–8.
25. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. Wayne, PA: CLSI; 2024.
26. Pfaller MA, Castanheira M. Antifungal resistance in Candida. Curr Opin Infect Dis. 2016;29(6):574–82.
27. Dellit TH, Owens RC, McGowan JE, et al. Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America guidelines for developing an institutional program to enhance antimicrobial stewardship. Clin Infect Dis. 2007;44(2):159–77.
28. World Health Organization. Guidelines on core components of infection prevention and control programmes at the national and acute health care facility level. Geneva: WHO; 2016.