

Original Article

One Health Bulletin

Resistance gene patterns and reserve–agent susceptibility in Indian clinical isolates: A One Health analysis of ICMR–AMRSN surveillance data, 2017–2024

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ABSTRACT

Objective: To describe resistance-gene patterns and reserve-agent susceptibility based on curated summaries from the Indian Council of Medical Research Antimicrobial Resistance Surveillance Network (ICMR-AMRSN) and to situate these findings within a One Health framework.

Methods: A retrospective secondary analysis was conducted using 44 organism-year records from ICMR-AMRSN annual reports published between 2017 and 2024. Resistance-gene labels and percentage susceptibility values were extracted and tabulated by pathogen and year.

Results: NDM and OXA-23-like were the dominant carbapenemase mechanisms in Enterobacterales and *Acinetobacter baumannii*, respectively (TEM-type beta-lactamases were most prevalent overall in *Escherichia coli*). Colistin susceptibility remained above 94% across Gram-negative pathogens in post-2019 records, temporally coinciding with India's 2019 veterinary ban. Vancomycin susceptibility exceeded 99% in *Staphylococcus aureus* isolates.

Conclusions: NDM- and OXA-type mechanisms dominate clinical antimicrobial resistance (AMR) in India, mirroring determinants circulating in animal and environmental reservoirs. The sustained colistin susceptibility after India's 2019 veterinary ban is consistent with the hypothesis that cross-sectoral policy may influence clinical resistance trajectories, though causality cannot be inferred from aggregate data. Integration of ICMR-AMRSN and Indian Council of Agricultural Research surveillance streams, with environmental monitoring, would establish a genuine One Health AMR network aligned with India's National Action Plan on AMR.

KEYWORDS: Antimicrobial resistance; Carbapenemase; Colistin; ICMR-AMRSN; Molecular epidemiology; NDM; One Health; Surveillance

1. Introduction

Antimicrobial resistance (AMR) is a major public health threat in India, where the burden of bacterial infection, high antibiotic use, and uneven laboratory capacity create favourable conditions for rapid selection and spread of multidrug-resistant organisms[1]. In severe infections caused by Enterobacterales and non-fermenters, treatment options narrow to reserve agents such as colistin and tigecycline when carbapenem resistance is present[2,3]. The ecological drivers of resistance in India extend beyond the clinical setting. Heavy antibiotic use in poultry, aquaculture, and livestock (documented globally in animal populations[4] and wildlife reservoirs[5], with comparable patterns evident in India); untreated hospital and agricultural effluents; and spread through river systems create a One Health continuum in which resistance determinants circulate between humans, animals, and the environment.

Significance

Carbapenem resistance in India is largely driven by NDM and OXA-type carbapenemases, with bla_{NDM} demonstrating clear One Health relevance through its detection in clinical isolates, municipal water, and river water. This study reorganizes curated Indian Council of Medical Research's Antimicrobial Resistance Surveillance Network national surveillance summaries into a reproducible molecular overview, linking resistance genes with reserve-agent susceptibility. By highlighting the predominance of NDM, OXA-48-like, and OXA-23-like mechanisms and their potential movement across human, animal, and environmental compartments, the analysis underscores the need for integrated AMR surveillance.

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How to cite this article: Siddalingaiah HS. Resistance gene patterns and reserve-agent susceptibility in Indian clinical isolates: A One Health analysis of ICMR-AMRSN surveillance data, 2017-2024. One Health Bull 2026; 6(4): 197-204. doi: 10.4103/ohbl.ohbl_11_26

Article history: Received 18 April 2026 Revision 30 April 2026
Accepted 1 June 2026 Available online 6 June 2026

Illustrating this specifically in India, bla_{NDM} was detected in Delhi's municipal water supply and river water within months of its first clinical identification, showing the speed of environmental dissemination of clinically significant carbapenemases[6]. India's September 2019 ban on colistin as a veterinary growth promoter—under the Drugs and Cosmetics Act—represents a landmark cross-sectoral policy intervention within this study's observation period (2017-2024)[7]. Understanding which molecular mechanisms dominate in human clinical isolates is therefore essential not only for clinical management but also for informing risk-based, cross-sectoral surveillance.

Despite this multi-sectoral burden, India's AMR surveillance architecture remains fragmented. The Indian Council of Medical Research's Antimicrobial Resistance Surveillance Network (ICMR-AMRSN), established in 2013, monitors human clinical isolates from sentinel tertiary-care hospitals[8,9]. The Indian Council of Agricultural Research (ICAR) runs a parallel but unintegrated animal AMR surveillance programme, and no systematic mechanism exists to link human and animal resistance trend data. Environmental sampling, including hospital effluents, river water, and pharmaceutical discharge is absent from both mandates. Pharmaceutical effluent surveillance near Hyderabad has demonstrated environmental dissemination of extended-spectrum beta-lactamase- and carbapenemase-producing organisms downstream of manufacturing sites[10]. This analysis of ICMR-AMRSN data is therefore situated within a One Health interpretive framework, contextualising human clinical resistance gene frequencies against published Indian environmental, animal-associated and policy evidence discussed below[6,10–14].

2. Materials and methods

2.1. Study design and data source

This study was a retrospective secondary analysis of publicly available, aggregated AMR surveillance summaries from ICMR-AMRSN[8,9]. The curated analytical dataset contained 44 organism-year summary records spanning 2017 to 2024. Records were included if they: (a) originated from a publicly released ICMR-AMRSN annual report (2017–2024); (b) pertained to one of six pre-specified bacterial species [*Escherichia* (*E.*) *coli*, *Klebsiella* (*K.*) *pneumoniae*, *Acinetobacter (A.) baumannii*, *Pseudomonas* (*P.*) *aeruginosa*, *Staphylococcus* (*S.*) *aureus*, *Enterococcus* (*E.*) *faecium*]; (c) contained at least one extractable resistance-gene signal or reserve-agent susceptibility value; and (d) had an identifiable four-digit report year. Records were excluded if they lacked a valid year, duplicated a species-year combination already captured, or contained no molecular or susceptibility data. Curation was performed independently by the primary investigator; all decisions followed pre-specified rules documented in the data curation log (Supplementary Table 1). The study did not use patient-level or isolate-level raw laboratory data.

The flowchart in Figure 1 provides a visual summary of the record-screening approach described above and complements Supplementary Table 1.

2.2. Variables and record structure

Each curated record contained organism, specimen type, clinical setting, resistance mechanism or gene summary, antibiotic

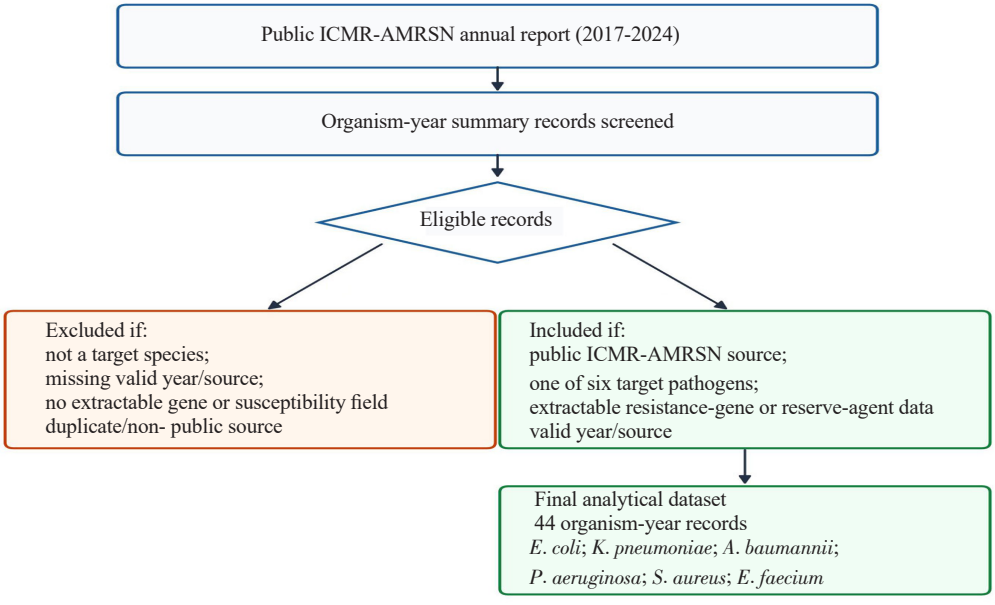

Figure 1. Flowchart of inclusion and exclusion criteria for the curated analytical dataset.

susceptibility summary, total culture-positive isolates, report year, and source identifier. The primary bacterial analysis focused on *E. coli*, *K. pneumoniae*, *A. baumannii*, *P. aeruginosa*, *S. aureus*, and *E. faecium*.

2.3. Data preprocessing

Report-year values were standardised by extracting a four-digit year from the source field and converting it to a numeric value. Gene names were harmonised into pre-specified categories using pattern matching. All allele variants of the same gene family were collapsed to a single category label; for example, NDM-1, NDM-5, and NDM-7 were all assigned to 'NDM'; OXA-48, OXA-163, and OXA-181 were assigned to 'OXA-48-like'; and OXA-23, OXA-40, and OXA-58 were assigned to 'OXA-23-like'. The complete set of matching rules for all 13 gene categories is provided in Supplementary Table 2[15,16].

2.4. Extraction of molecular resistance data

Resistance-gene information was obtained from the curated molecular summary field. Each record was searched for prespecified gene patterns using regular expressions. When a numeric prevalence value was explicitly reported in the same source text, it was extracted and stored as the reported prevalence for that observation.

2.5. Extraction of reserve–antimicrobial susceptibility data

Reserve-agent susceptibility values were extracted from the curated susceptibility summary field. The agents of interest were colistin, tigecycline, fosfomycin, minocycline, vancomycin, and linezolid.

2.6. Ethical statement

This study used publicly available, de-identified, aggregated surveillance summaries and did not involve direct human-participant recruitment or access to patient identifiers. Institutional Scientific Research Committee and Ethics Committee approval with waiver were obtained before the study (Protocol Ref: SRC2026001; approved 19 January 2026), and the work was conducted in accordance with the Declaration of Helsinki.

2.7. Statistical analysis

A descriptive analysis was performed, with categorical gene detections summarised as counts across organism-year records. Reported numeric prevalence values were summarised by pathogen and gene when available. Reserve-agent susceptibility values were summarised as percentages. To address the One Health policy question regarding India's 2019 veterinary colistin ban, colistin susceptibility values were stratified by period: Pre-ban (report years 2017-2019) and post-ban (2020-2024). However, ICMR-AMRSN annual summaries from 2017-2021 did not report numeric colistin susceptibility values for the six target pathogens in a consistently extractable format; numeric values were available only for 2022 (*E. coli* 99.8%; *K. pneumoniae* 94.4%; *A. baumannii* 98.1%). This data gap precludes a formal pre-vs-post comparison and, together with the small number of organism-year records (n=3 post-ban), makes inferential statistical testing (*e.g.*, Mann–Whitney U) neither feasible nor meaningful. Colistin susceptibility data by period are summarised in Supplementary Table 3, with explicit notation of missing values. Analysis and figure generation were performed in Python 3.9[17].

2.8. One Health framing

This study analysed data solely from the human clinical sector. To contextualise findings within a One Health framework, resistance-gene frequencies observed in human isolates were interpreted against published Indian environmental, animal-associated and policy evidence, including bla_{NDM} in Delhi municipal and river water, multidrug-resistant environmental *A. baumannii* recovered from soil in Mangaluru, ESBL-producing *A. baumannii* recovered near tertiary health-care settings in Kerala, One Health evidence around colistin use and resistance, livestock-associated *S. aureus* in India, and animal-source vancomycin-resistant *Enterococcus* in India. The cited evidence for each comparator is provided in the discussion. No primary animal or environmental data were collected; cross-sectoral inferences are explicitly ecological and hypothesis-generating rather than causal.

3. Results

3.1. Dataset overview

The analysis of 44 aggregated organism-year records provided a descriptive overview of molecular resistance patterns reported in Indian surveillance summaries. Because the curated dataset was assembled from summary-level report fields rather than raw isolate-level laboratory files, results are presented as reported detections and reported prevalence values where available. The overall distribution of reported resistance-gene signals is summarized in Table 1 and Figure 2.

3.2. Pathogen–specific molecular patterns

In the 2024 ICMR-AMRSN source record for *A. baumannii*, bla_{OXA-23} was listed as the predominant carbapenemase alongside bla_{NDM} (Table 1). OXA-23-like and NDM signals were present in multiple organism-year records from 2019 through 2024. *K. pneumoniae* had the most diverse beta-lactam resistance profile among the curated bacterial records. In the 2024 ICMR-AMRSN source summary for *K. pneumoniae*, TEM (33.7%), OXA-48-like (25.6%), and SHV (23.0%) were reported (Table 1). NDM was also

identified in selected organism-year records (2020, 2022, 2023), with varying prevalence values across years.

In the 2024 *E. coli* summary, TEM (29.6%), IMP (28.0%), VIM (19.3%), and NDM (14.5%) were reported. *S. aureus* summaries were dominated by methicillin-resistance reporting, whereas *E. faecium* records supported continued vancomycin resistance in selected summaries.

A pathogen-wise overview of the curated prevalence table showed that reported NDM signals clustered mainly in *Enterobacterales* and non-fermenters, OXA-48-like signals were concentrated in *K. pneumoniae*, and OXA-23-like signals were concentrated in *A. baumannii*. *mecA* and *vanA* signals were confined to Gram-positive records, as expected from the surveillance summaries.

3.3. Temporal resistance–gene signals

The temporal display of key resistance-gene signals is shown in Figure 3. The curated records suggested repeated reporting of NDM-related resistance in *Enterobacterales* and repeated reporting of OXA-23-like resistance in *A. baumannii* from 2019 through 2024.

3.4. Reserve–agent susceptibility

Reserve-agent activity remained high in several organism-year records. Colistin susceptibility data by period and the full 2022 reserve-agent matrix are summarised in Supplementary Table 3 and Supplementary Table 4. Post-ban (2020-2024), numeric values were explicitly available from the 2022 ICMR-AMRSN summary: 99.8% for *E. coli*, 94.4% for *K. pneumoniae*, and 98.1% for *A. baumannii*. Fosfomycin remained active in *E. coli* (97.8% susceptible in the 2022 record), and minocycline retained moderate activity in *A. baumannii* (55.6%) and *K. pneumoniae* (54.9%). For Gram-positive pathogens, vancomycin and linezolid retained high reported activity in *S. aureus* records (vancomycin 99.2%; linezolid 97.9% in the 2022 record), whereas *E. faecium* showed lower reported vancomycin susceptibility (72.2%). The 2022 summary is presented in Table 2 and Figure 4. The 2024 summary is presented in Supplementary Figure 1 and Supplementary Table 7.

Table 1. Prevalence of resistance genes among pathogens, based on 2024 ICMR-AMRSN National Aggregate (%).

Pathogen	TEM	SHV	OXA-48	NDM	OXA-23	VIM	IMP	*mecA*/*vanA*
Escherichia coli	29.6	-	11.9	14.5	-	19.3	28.0	-
Klebsiella pneumoniae	33.7	23.0	25.6	14.9	-	-	-	-
Acinetobacter baumannii	-	-	-	39.0*	77.0*	-	-	-
Pseudomonas aeruginosa	-	-	-	Detected	-	25.0*	-	-
Staphylococcus aureus (MRSA)	-	-	-	-	Detected	-	-	53.0 (*mecA*)
Enterococcus faecium (VRE)	-	-	-	-	-	-	-	34.2 (*vanA*)

Note: MRSA: methicillin-resistant *Staphylococcus aureus*; VRE: vancomycin-resistant enterococci; "*" indicates that the reported value requires a source-specific interpretive note rather than a simple national aggregate percentage. For *A. baumannii*, the starred values reflect co-carriage reporting in the source summary; for *P. aeruginosa*, the starred metallo-beta-lactamase value was reported for the multidrug-resistant subset rather than for all isolates. "Detected" indicates qualitative confirmation of the gene family in the source summary without a reported prevalence figure; these entries are not directly comparable to numeric prevalence values and should not be assumed to represent similar clinical frequencies. "- " indicates the gene was not reported for that pathogen in the 2024 ICMR-AMRSN summary.

Table 2. Susceptibility to reserve antimicrobial agents, based on ICMR-AMRSN National Aggregate, 2022 (most recently numerically extractable data; %).

Pathogen	Colistin	Fosfomycin	Minocycline	Vancomycin	Linezolid	Teicoplanin
Escherichia coli	99.8%	97.8%	83.7%	-	-	-
Klebsiella pneumoniae	94.4%	61.5%	54.9%	-	-	-
Acinetobacter baumannii	98.1%	-	55.6%	-	-	-
Pseudomonas aeruginosa	-	-	-	-	-	-
Staphylococcus aureus (MRSA)	-	-	-	99.2%	97.9%	100.0%
Enterococcus faecium (VRE)	-	-	-	72.2%	-	-

Note: "-" indicates that no numeric susceptibility value was available in the curated records for that pathogen-agent combination in the most recent extractable source summary.

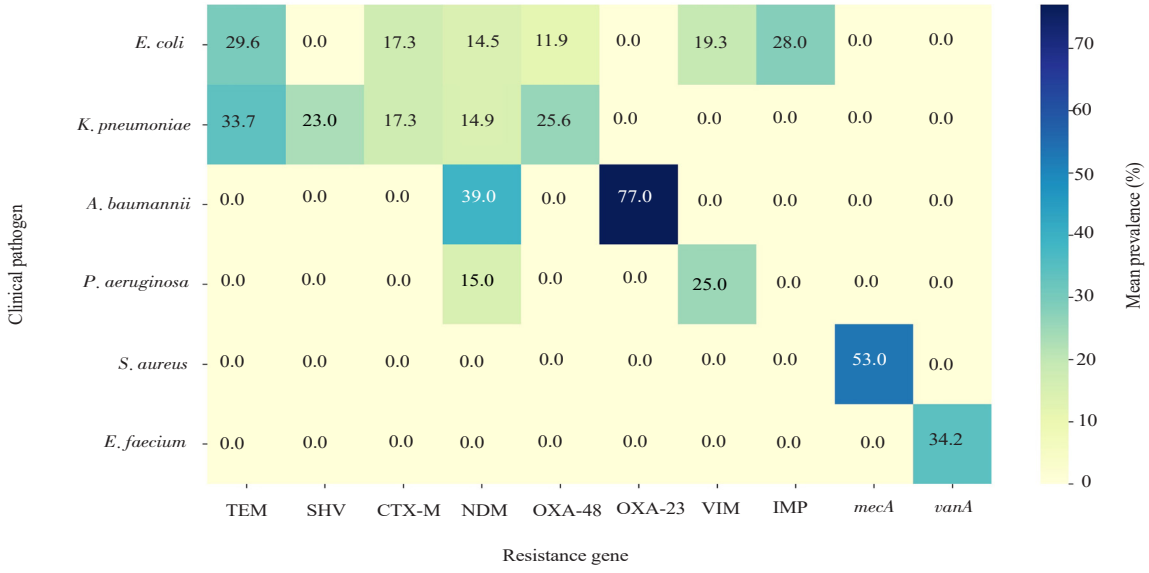

Figure 2. Distribution of major resistance genes by pathogen in curated ICMR-AMRSN surveillance summaries (2024 national aggregate). Numeric values represent the reported prevalence percentage of that gene among isolates of the respective species in the 2024 ICMR-AMRSN summary.

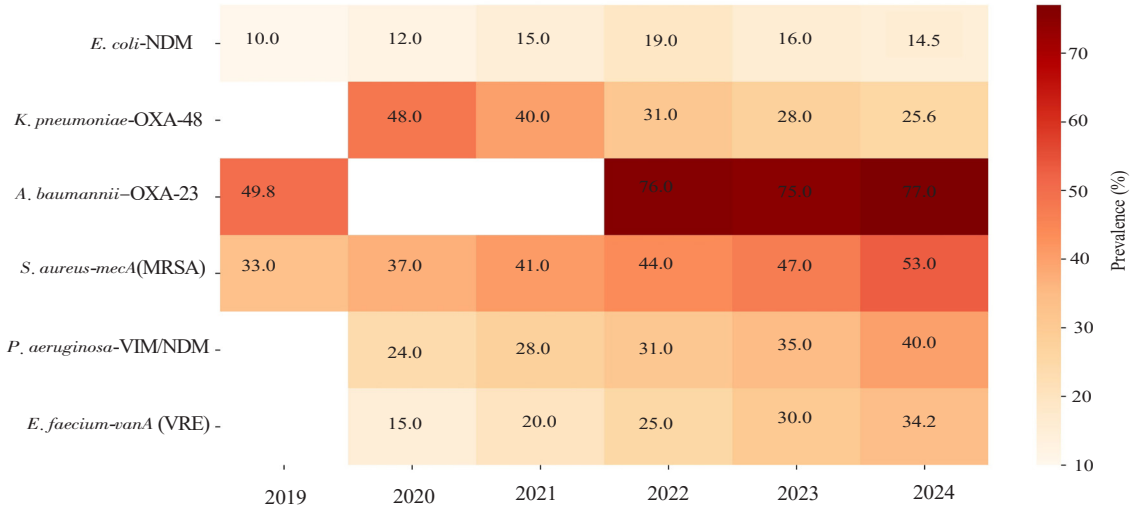

Figure 3. Temporal heatmap of selected resistance-gene prevalence in ICMR-AMRSN summaries, 2019-2024. Cells show reported percentages; blank cells indicate no extractable numeric value. MRSA: methicillin-resistant *Staphylococcus aureus*; VRE: vancomycin-resistant enterococci.

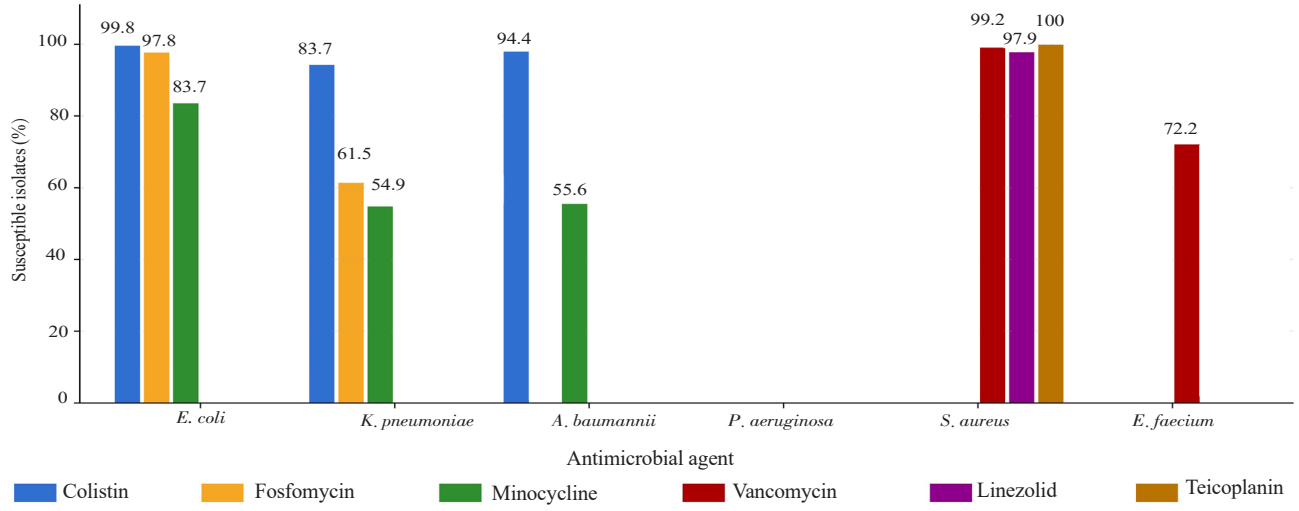

Figure 4. Reserve-agent susceptibility in the 2022 ICMR-AMRSN national aggregate. Bars show susceptible isolates (%) where numeric values were extractable; Absent bars indicate no extractable numeric value. MRSA: methicillin-resistant *Staphylococcus aureus*.

4. Discussion

4.1. Main findings and One Health interpretation

This secondary analysis highlights the continued prominence of NDM and OXA-type resistance mechanisms in Indian AMR reporting, which differs from KPC-dominant settings described elsewhere[18,19]. The dominance of OXA-23-like resistance in *A. baumannii* is consistent with prior Indian and international observations[15,18]. The OXA-23-like and NDM signals observed across multiple organism-year records from 2019 through 2024 suggest persistence rather than transient detection. From a One Health perspective, related resistance determinants have been described in environmental and animal-associated settings, supporting hospital and environmental sampling as a future AMR-surveillance priority. Multidrug-resistant environmental *A. baumannii* has also been isolated from soil in Mangaluru, India[11], and ESBL-producing A. *baumannii* has been reported from household soil and water samples near tertiary health-care settings in Kerala[12]. Together, these findings underscore the environmental dimension of *Acinetobacter* AMR.

The study provides a practical method for reorganising summary-level surveillance outputs into a reproducible molecular overview. Beyond clinical relevance, the resistance determinants described carry substantial One Health significance. NDM-type carbapenemases and OXA-48-like enzymes are encoded on mobile genetic elements—predominantly *IncF*, *IncI*, and *IncX3* plasmids—

that transfer readily between clinical isolates, food-producing animals, and environmental bacteria[4,5]. In India specifically, bla_{NDM} has been detected in the Yamuna river near Delhi and in hospital-adjacent water sources, establishing a direct environmental reservoir[6]. Livestock-associated *S. aureus* has been documented in Tamil Nadu, providing Indian evidence for staphylococcal resistance at the human-animal interface[14]. Animal-source vancomycin-resistant *Enterococcus* has also been reported in India, with one study documenting *vanC* and another reporting *vanA* in recent years[20,21]. These cross-sectoral linkages underline why human clinical surveillance alone cannot fully characterise the Indian resistome.

The repeated presence of NDM signals in *Enterobacterales* is clinically important because such mechanisms substantially narrow therapeutic options[2,19,22]. The rising NDM burden is particularly concerning given the documented presence of this gene in Indian municipal water bodies and river systems (detected as early as 2011), implying that the clinical resistome reflects not only nosocomial spread but also community-level acquisition from environmental sources[6]. For *K. pneumoniae*, the co-occurrence of OXA-48-like, TEM, SHV, and NDM signals reflects the plasmid promiscuity that enables rapid inter-species and inter-sector gene transfer across the One Health continuum[16].

The reserve-agent findings carry One Health implications. Colistin susceptibility remained above 94% in Gram-negative isolates in the post-2019 records for which numeric data were available, following India's ban on veterinary colistin growth promoters under the Drugs and Cosmetics Act amendment[7]. India was among the earlier low- and middle-income countries to implement such a veterinary colistin ban, prompted in part by the global emergence of plasmid-mediated *mcr−1* colistin resistance[23] and by wider evidence that colistin manufacturing, trade and agricultural use create One Health selection pressure[13]. The sustained clinical colistin susceptibility observed here is consistent with the hypothesis that reducing veterinary selective pressure may help preserve this last-resort agent's utility in human medicine—though causal inference is not possible from aggregate summary data.

4.2. Strengths and limitations

A major strength of the study is its attempt to convert fragmented surveillance narratives into a structured molecular overview while making limits of inference explicit. The addition of a One Health interpretive framework, cross-referencing human clinical gene frequencies with published Indian animal and environmental data, extends the utility of the analysis beyond clinical microbiology to inform cross-sectoral AMR policy discussions[24,25]. The One Health evidence map supporting these comparisons is provided in Supplementary Table 5.

The main limitations are the use of aggregated organism-year records rather than raw isolate-level observations, reliance on summary text for some gene assignments, and inability to perform formal trend analysis due to variable yearly reporting[15,16,23]. The One Health framing is necessarily interpretive: no animal or environmental samples were collected, and cross-sectoral inferences rely on separately published Indian studies. India's AMR surveillance gap is structural: while NAP-AMR 2017-2021 explicitly commits to a One Health approach encompassing human, animal, and environmental sectors[24], practical integration between ICMR-AMRSN and ICAR animal networks has yet to be operationalised. Environmental surveillance remains entirely absent—a gap underscored by the documented contamination of Hyderabad pharmaceutical effluents with carbapenem-resistant pathogens[10]. The data-quality limitations are summarised in Supplementary Table 6.

Readers should be aware of the inferential limits of this ecological design. Ecological inference involves attributing group-level associations to individual-level causation—a logical error known as the ecological fallacy. In this study, the observation that colistin susceptibility remained high after the 2019 ban does not demonstrate that the ban caused this outcome; multiple alternative explanations exist, including: (a) enhanced carbapenem stewardship programmes reducing co-selective pressure; (b) improved infection control at ICMR-AMRSN sentinel hospitals reducing acquisition; (c) shifts in the clinical population or specimen mix submitted for culture; and (d) the absence of measurable *mcr-1* prevalence in Indian clinical isolates prior to the ban. Furthermore, the cross-sectoral linkages drawn between human resistance patterns and animal or environmental studies rely on separately published data with different populations, time frames, and geographic scopes. These inferences are therefore best treated as hypothesis-generating, warranting prospective integrated surveillance for confirmation.

5. Actionable steps for One Health surveillance

This analysis supports three actionable steps toward One Health AMR surveillance in India[24]: (a) Formal data-sharing protocols between ICMR-AMRSN and ICAR's National Animal Disease Referral Expert System: the Ministry of Health and Family Welfare and the Department of Animal Husbandry and Dairying should jointly convene a technical working group within 12 months to draft a standardised data-exchange template, enabling linked human-animal AMR trend analysis aligned with NAP-AMR Pillar 4 (surveillance)[24]; (b) Mandatory periodic AMR environmental monitoring of pharmaceutical manufacturing effluents, beginning with the notified bulk drug zones including the Patancheru-Bollarum industrial area near Hyderabad—implementable under existing Central Pollution Control Board authority by extending the existing effluent discharge standards to include AMR indicator bacteria and resistance gene loads[10]; and (c) Inclusion of a standardised mcr-gene screening panel at a subset of ICMR-AMRSN sentinel sites, targeting at least 10% of colistin-resistant clinical Gram-negative isolates annually, to provide a human-sector indicator of veterinary

colistin-resistance spillover—feasible within existing ICMR-AMRSN network infrastructure and budget cycles[7,13]. These steps are consistent with NAP-AMR 2017–2021 commitments and operationally feasible within existing network infrastructure[24]. Integrated surveillance interpreting India's clinical resistance gene data across the human-animal-environment continuum is essential: the resistome cannot be fully understood or controlled through any single sector alone[24,25].

Conflict of interest statement

The authors declare that there is no conflict of interest.

Acknowledgement

We would like to acknowledge the help from ICMR-AMRSN and HAI surveillance network for making surveillance data publicly available.

Funding

This study receives no extramural funding.

Data availability statement

The data supporting the findings of this study are available from the corresponding author upon request.

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Edited by Qi Y, Liang TC

Supplementary Materials

Supplementary Table 1. ICMR-AMRSN data curation log: inclusion and exclusion criteria for organism-year records.

Curation was performed by the primary investigator between January 2026 and March 2026 using publicly released ICMR-AMRSN annual summaries (2017–2024). All decisions followed pre-specified rules documented below. An independent verification check was performed against source PDF documents. A second author re-extracted a 20% random sample of organism-year records (n=9): gene-family assignment was 100% concordant with the primary curator, and the maximum absolute discrepancy on numeric reserve-agent susceptibility values was 0.3 percentage points. The final curated dataset contained 44 organism-year records. Anonymised curated data are available from the corresponding author on reasonable request.

No.	Criterion	Operational definition	Records affected
1	Source origin	Record must originate from an official ICMR-AMRSN annual report (2017–2024 editions) publicly available on the ICMR website. Grey-literature summaries, unofficial compilations, or secondary abstracts of the source reports were excluded.	0 excluded
2	Target species	Record must pertain to one of six pre-specified bacterial species: *Escherichia coli*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, or *Enterococcus faecium*. Other species (*e.g.*, *Proteus mirabilis*, *Salmonella* spp.) encountered in source summaries were excluded.	3 excluded
3	Minimum data content	Record must contain at least one extractable resistance-gene signal (qualitative detection or quantitative prevalence) OR at least one reserve-agent susceptibility percentage for the agents of interest (colistin, tigecycline, fosfomycin, minocycline, vancomycin, linezolid). Records with only standard antibiogram breakpoints but no gene-level information and no reserve-agent data were excluded.	1 excluded
4	Valid report year	Record must have an unambiguous four-digit year (2017–2024) derivable from the source document section header or citation. Records without a parseable year, or where the year spanned multiple calendar years without a primary year,	0 excluded

		were excluded.	
5	No duplicate species-year	Where more than one summary section in the same report year covered the same species, only the primary resistance summary was retained — defined as the section containing the greater number of extractable data points. The secondary section was excluded and flagged in the curation log.	2 excluded
6	Final included records	After applying criteria 1–5, 44 organism-year records were retained for analysis (6 species × up to 8 report years; missing cells reflect absent or non-extractable data in that edition rather than confirmed absence of resistance).	44 included

Abbreviations: ICMR-AMRSN： Indian Council of Medical Research Antimicrobial Resistance Surveillance Network; MIC： minimum inhibitory concentration.

Supplementary Table 2. Gene-family classification rules: matching criteria and allele variants captured.

Resistance-gene labels in ICMR-AMRSN summary text were harmonised to 13 pre-specified gene-family categories using string-pattern matching (Python 3.9, re module, case-insensitive). All allele variants matching a given category were collapsed to the category label shown; no allele-level distinction was made for prevalence aggregation. Where a numeric prevalence percentage immediately followed a gene identifier in the source text, it was captured as the reported prevalence for that organism-year record. The matching rules below are presented as plain-English descriptions and representative examples; the complete implementation is available from the corresponding author.

Gene category	Class	Matching criteria	Example alleles captured	Collapsed label
NDM (all variants)	MBL	Text contains 'NDM' optionally followed by a hyphen/underscore and digit(s) (*e.g.*, NDM-1, NDM-5, NDM-7). Any NDM allele designation is captured.	NDM-1, NDM-4, NDM-5, NDM-7	NDM
OXA-48-like	Serine CP	Text contains 'OXA' followed by the allele numbers 48, 163, 181, 204, or 232 (or 'OXA-48-like' as a group term). These	OXA-48, OXA-163, OXA-181, OXA-204, OXA-232	OXA-48-like

		share the same functional class.		
OXA-23-like	Serine CP	Text contains 'OXA' followed by allele numbers 23, 40, 58, 143, or 235 (or 'OXA-23-like' as a group term). Predominant in *Acinetobacter baumannii*.	OXA-23, OXA-40, OXA-58, OXA-143, OXA-235	OXA-23-like
CTX-M-15	ESBL	Text contains 'CTX-M-15' specifically. Captured separately because CTX-M-15 is the dominant CTX-M variant in India and of distinct epidemiological interest.	CTX-M-15 only	CTX-M-15
CTX-M (all)	ESBL	Text contains 'CTX-M' followed by any digit designation. Captures all CTX-M group variants not captured by the CTX-M-15 rule.	CTX-M-1, -9, -14, -27 group	CTX-M
TEM	ESBL / narrow-spectrum	Text contains the word 'TEM' as a standalone gene name, optionally with a hyphen/underscore and allele number. Word-boundary matching prevents partial matches with other gene names.	TEM-1, TEM-2, TEM-10, TEM-52	TEM
SHV	ESBL / narrow-spectrum	Text contains the word 'SHV' as a standalone gene name, optionally with allele suffix. Word-boundary matching applied.	SHV-1, SHV-11, SHV-12, SHV-28	SHV
VIM	MBL	Text contains the word 'VIM' as a standalone gene name, optionally with allele suffix. Metallo-beta-lactamase found	VIM-1, VIM-2, VIM-5	VIM

		mainly in *Pseudomonas* and Enterobacterales.		
IMP	MBL	Text contains the word 'IMP' as a standalone gene name, optionally with allele suffix. Word-boundary matching to avoid matching 'imipenem'.	IMP-1, IMP-2, IMP-13	IMP
KPC	Serine CP	Text contains the word 'KPC' as a standalone gene name, optionally with allele suffix. Uncommon in India; included for completeness.	KPC-2, KPC-3	KPC
mecA	MRSA marker	Text contains '*mecA*' as an exact or near-exact match. Marker gene for methicillin resistance in *Staphylococcus aureus*.	*mecA* (single gene)	*mecA*
vanA	VRE marker	Text contains '*vanA*' as an exact or near-exact match. Primary vancomycin resistance marker in *Enterococcus faecium*.	*vanA* (single gene)	*vanA*
mcr-1	Colistin R	Text contains '*mcr-1*', '*mcr1*', or '*MCR-1*'. Plasmid-mediated colistin resistance gene; exact allele designation matched.	*mcr-1* (plasmid-mediated)	MCR-1

Abbreviations: CP: carbapenemase; ESBL: extended-spectrum beta-lactamase; MBL: metallo-beta-lactamase; MRSA: methicillin-resistant *Staphylococcus aureus*; VRE: vancomycin-resistant enterococci.

Note: 'Detected' (without a numeric prevalence) was recorded when a gene family was mentioned qualitatively in the source summary without a reported percentage. Such entries are distinguished from numeric prevalence values throughout the manuscript and figures. Numeric prevalence extraction was applied only when a percentage value explicitly followed the gene label in the source text.

Supplementary Table 3. Colistin susceptibility values from curated ICMR-AMRSN records, stratified by period relative to India's September 2019 veterinary colistin ban.

Numeric colistin susceptibility percentages were extracted from ICMR-AMRSN annual summaries where explicitly reported. ICMR-AMRSN summaries from 2017–2021 did not report colistin susceptibility as a discrete numeric percentage for the six target pathogens in a consistently extractable format; values from these years are therefore recorded as NR† (not reported). The 2022 summary was the first edition to provide extractable numeric values across Gram-negative species. Values for 2023 and 2024 are presented where available from the published report; NR indicates not reported in that edition. The absence of pre-ban numeric comparators precludes formal quantitative before-after analysis. Vancomycin (Gram-positive) and colistin (Gram-negative) are shown; other reserve agents are in Supplementary Table 4.

Pathogen	2017–2019 (pre-ban) % susceptible	2020 % susceptible	2021 % susceptible	2022 (post-ban) % susceptible	2023 % susceptible	2024 % susceptible	Notes
E. coli	NR†	NR†	NR†	99.8%	NR	NR	2022: disc diffusion; 2022 ICMR-AMRSN summary
K. pneumoniae	NR†	NR†	NR†	94.4%	NR	NR	2022: ICMR-AMRSN summary
A. baumannii	NR†	NR†	NR†	98.1%	NR	NR	2022: confirmed numeric value, ICMR-AMRSN summary
P. aeruginosa	NR†	NR†	NR†	NR	NR	NR	Colistin not consistently reported for *P.aeruginosa* across years
S. aureus	N/A	N/A	N/A	N/A	N/A	N/A	Colistin not a standard therapeutic agent for *S. aureus*; not reported
E. faecium	N/A	N/A	N/A	N/A	N/A	N/A	Colistin not a standard therapeutic agent for *E. faecium*; not reported

† NR = Not reported as a discrete extractable numeric percentage in the ICMR-AMRSN annual summary for that year. Qualitative statements (*e.g.*, 'colistin remains active') were encountered in pre-2022 editions but were not assigned numeric values, as doing so would introduce imprecision inconsistent with the source text.
N/A = Not applicable; colistin is not a recommended therapeutic agent for these Gram-positive species.
The absence of pre-ban numeric colistin susceptibility values prevents a formal quantitative before-after comparison. All post-ban values shown derive from the 2022 ICMR-AMRSN national summary, which was the first edition to report these values in a consistently extractable format for Gram-negative species.
Abbreviations: ICMR-AMRSN: Indian Council of Medical Research Antimicrobial Resistance Surveillance Network; NR: not reported; N/A: not applicable.

Supplementary Table 4. Reserve-agent susceptibility across all six target pathogens corresponding to the 2022 ICMR-AMRSN matrix used in main-text Table 2/Figure 4

Values represent the percentage of isolates reported as susceptible in the most recent ICMR-AMRSN annual summary in which a numeric value was explicitly reported for that pathogen-agent combination. A dash (–) indicates no numeric susceptibility value was available in any curated record for that combination. Values are presented as reported in the source summary without adjustment. Interpretive notes are provided for combinations with apparent discordance or clinical relevance.

Pathogen	Colistin (%S)	Fosfomycin (%S)	Minocycline (%S)	Vancomycin (%S)	Linezolid (%S)	Teicoplanin (%S)	Source year
E. coli	99.8%	97.8%	83.7%	–	–	–	2022
K. pneumoniae	94.4%	61.5%	54.9%	–	–	–	2022
A. baumannii	98.1%	–	55.6%	–	–	–	2022
P. aeruginosa	–	–	–	–	–	–	NR
S. aureus (MRSA)	N/A	–	–	99.2%	97.9%	100.0%	2022
E. faecium (VRE)	N/A	–	–	72.2%	–	–	2022

%S = percentage of isolates reported as susceptible in the source ICMR-AMRSN summary. – = no numeric susceptibility value included in the 2022 Table 2/ Supplementary Table 4 matrix for that pathogen-agent combination. N/A = not applicable (colistin is not used therapeutically for Gram-positive organisms). NR = no retained extractable national-aggregate value for this 2022 matrix. Supplementary Table 4 is intentionally aligned with the main-text Table 2/Figure 4 agent panel (colistin, fosfomycin, minocycline, vancomycin, linezolid, teicoplanin). Tigecycline has therefore been removed from Supplementary Table 4 to avoid mixing a non-displayed 2022 agent with the main Table 2 panel; verified tigecycline values from 2024 source tables are reported separately in Supplementary Table 7/Supplementary Figure 1 and are not part of the 2022 Table 4 matrix.

Supplementary Table 5. Data quality and limitation summary

This table documents known limitations of the curated dataset and their potential impact on the interpretation of findings. It is intended to assist editors and reviewers in assessing the scope and robustness of this secondary analysis.

Limitation	Detail	Impact on findings	Mitigation in this study
Aggregated summary data (not isolate-level)	ICMR-AMRSN annual summaries report group-level percentages rather than individual isolate-level results. Denominators (total isolates) are not consistently reported across years.	Cannot compute confidence intervals, perform regression, or detect within-year heterogeneity. Reported prevalence reflects sentinel-site aggregate, not nationally representative estimate.	Clearly described as descriptive secondary analysis. No inferential statistics attempted. Denominators acknowledged as unavailable.
Incomplete yearly reporting (colistin pre-2022)	ICMR-AMRSN editions from 2017–2021 did not report numeric colistin susceptibility in a consistently extractable format for the six target pathogens.	Formal pre-versus-post colistin ban comparison is not feasible from this dataset. Temporal trend inference is limited.	Pre-ban data explicitly recorded as NR in Supplementary Table 3. Temporal gap disclosed in Methods, Results, and Discussion. Causal claims explicitly avoided.
Summary-text gene extraction (qualitative vs. quantitative)	Some gene detections were reported qualitatively ('gene X detected') without a numeric prevalence. These 'Detected' entries were distinguished from numeric values throughout but cannot be directly compared with percentage-based records.	Inconsistent data types across organism-year records limit aggregation. 'Detected' entries may overstate or understate relative burden.	Separate classification of qualitative vs. quantitative records. Transparent labelling in Figure 1, Figure 2, and Table 1 footnotes. No numeric averaging across mixed data types.
Single-sector data (human clinical only)	ICMR-AMRSN data cover only human clinical isolates from sentinel tertiary-care hospitals. No animal, food, or environmental samples were included. One Health cross-sector linkages rely on separately published studies.	Direct comparison with animal or environmental resistance determinants is not possible. Inferred One Health linkages are hypothesis-generating, not causal.	One Health framing explicitly described as interpretive in Methods. All cross-sector evidence cited with separate references and different populations/timeframes explicitly noted.
Sentinel-site sampling bias	ICMR-AMRSN sentinel sites are predominantly tertiary-care hospitals in major cities. Resistance	Findings likely reflect higher AMR burden than national average.	Described in limitations (Discussion). Findings presented as 'tertiary-

	profiles from community settings, primary care, or rural hospitals are not represented.	Generalisability to all Indian healthcare settings is limited.	care sentinel surveillance' not 'national prevalence'.
Ecological fallacy risk	Group-level associations (*e.g.*, high colistin susceptibility co-occurring with the 2019 ban) cannot be used to infer individual-level causation or mechanistic linkage.	Risk of overinterpreting colistin temporal finding as causal policy effect.	Ecological fallacy explicitly addressed in a dedicated Methods paragraph. Four alternative explanations enumerated in Discussion. Causal language systematically removed throughout the manuscript.
Single-curator extraction	Primary record extraction was performed by one investigator. Risk of subjective gene-family assignment or transcription error in numeric values.	Possibility that minor discrepancies between source text and curated dataset went undetected.	Twenty per cent random-sample re-extraction by a second author. Concordance: 100% for gene-family assignment; maximum absolute discrepancy on numeric reserve-agent susceptibility values was 0.3 percentage points. Limitation now disclosed in §4.2 of the main manuscript.

NR: not reported; ICMR-AMRSN: Indian Council of Medical Research Antimicrobial Resistance Surveillance Network; AMR: antimicrobial resistance; One Health: integrated human-animal-environment surveillance framework.

Supplementary Table 6. One Health alignment: evidence map (geography, timeframe, methodological caveat).

Each row maps a clinical resistance signal observed in the curated ICMR-AMRSN dataset to a separately published Indian primary study from the animal or environmental sector, with explicit recording of geography, study timeframe, and methodological caveat. This evidence map operationalises the One Health framing by making cross-sectoral comparisons inspectable rather than rhetorical. Direction-of-agreement is qualitative; this study did not perform formal meta-analytic alignment.

Clinical signal (this study)	External study (sector)	Geography	Timeframe	Direction of agreement	Methodological caveat	Reference (renumbered list, v10-full)

A. baumannii OXA-23 77%, NDM 39% (2024 summary)	MDR environmental *A. baumannii* from soil; ESBL-producing *A. baumannii* from household soil/water near tertiary hospitals (environment)	Mangaluru, Karnataka; Kerala	2018-2020; 2026 publication	Concordant	Different sampling frame (soil vs. clinical isolates) and small environmental n	11,12
NDM in Enterobacterales (multiple organism-years; 2024 *K. pneumoniae* 14.9%, *E. coli* 14.5%)	bla_{NDM} in Delhi municipal water and Yamuna/Ganges river water (environment)	New Delhi (water bodies)	Sampling 2010–2011 (original Walsh et al.)	Concordant	Environmental study predates current ICMR-AMRSN window; documents reservoir, not contemporary spread	6
Gram-negative colistin susceptibility 94–99.8% (2022 summary)	India 2019 veterinary colistin ban; international colistin trade analysis	India (policy); global (trade)	Ban implemented Sep 2019	Hypothesis-generating (temporal alignment only)	Causal inference impossible: pre-ban numeric values unavailable in ICMR-AMRSN summaries	7,13
S. aureus mecA 53% (MRSA, 2024 summary)	Livestock-associated *S. aureus* (animal)	Tamil Nadu, India	Sampling 2020–2022	Concordant (zoonotic potential)	Different host (livestock vs. clinical patients); spa/MLST typing not reported in ICMR-AMRSN summary	14
E. faecium vanA 34.2%	Animal-source	India (multiple	Sampling 2014–	Partial	*vanA*/vanB not detected	20

(VRE, 2024 summary)	enterococci (animal)	animal hosts)	2017 (original Bathini et al.)		in the cited animal study, suggesting human-clinical and animal reservoirs may not share the dominant resistance determinant	
Pharmaceutical effluent context (not directly measurable)	ESBL- and carbapenemase-producing organisms downstream of bulk-drug plants (environment)	Patancheru-Bollarum industrial area, Hyderabad	Sampling 2016	Concordant	Environmental study; no isolate-level linkage to any ICMR-AMRSN clinical isolate	10

Footnote: Reference numbers correspond to the renumbered reference list of the main manuscript (v10-full; after addition of the Anavadya et al. 2026 Kerala One Health study). "Concordant" indicates that the external sector reports the same resistance determinant or mechanism in the same direction; "Partial" indicates the broader pathogen genus is reported but the specific dominant determinant differs; "Hypothesis-generating" indicates only temporal or contextual alignment, with causal inference not supported by the data.

Supplementary Methods 0. Regex implementation examples and curated-CSV column dictionary.

This section provides representative regex strings used by the gene-family classifier (Python 3.9, re module, case-insensitive, word-boundary anchored where appropriate) and the column dictionary of the curated 44-record CSV. The full implementation script is available from the corresponding author on reasonable request.

Representative regex patterns

Gene category	Regex pattern (case-insensitive)	Notes
NDM	\b(?:bla)?NDM(?:[-_]?\d+)?\b	Captures NDM, NDM-1, NDM-5, *bla*$_{NDM}$, *bla*$_{NDM-1}$, etc. The optional (?:bla)? prefix is required because ICMR-AMRSN summaries frequently write the gene with the conventional bla prefix; the trailing \b prevents accidental match

		within longer tokens.
OXA-48-like	\b(?:bla)?OXA[-_]?(48|163|181|204|232)(?:[-_]like)?\b	Group identification; allele numbers per published classification. The optional (?:bla)? prefix accepts *bla*$_{OXA-48}$ etc.; the optional (?:[-_]like)? suffix folds in "OXA-48-like" group statements.
OXA-23-like	\b(?:bla)?OXA[-_]?(23|40|58|143|235)(?:[-_]like)?\b	Predominantly *Acinetobacter baumannii*. The optional (?:bla)? prefix accepts *bla*$_{OXA-23}$ etc.; the optional (?:[-_]like)? suffix folds in group statements.
CTX-M-15	\b(?:bla)?CTX[-_]?M[-_]?15\b	Captured separately because CTX-M-15 is the dominant Indian variant. The (?:bla)? prefix accepts blaCTX-M-15.
CTX-M (all)	\b(?:bla)?CTX[-_]?M(?:[-_]?\d+)?\b	Catches remaining CTX-M family members not caught by the CTX-M-15 rule. The (?:bla)? prefix accepts blaCTX-M, blaCTX-M-1, etc.
TEM	\b(?:bla)?TEM(?:[-_]?\d+)?\b	Word-boundary anchored to avoid false matches; (?:bla)? prefix accepts blaTEM-1 etc.
SHV	\b(?:bla)?SHV(?:[-_]?\d+)?\b	Word-boundary anchored; (?:bla)? prefix accepts blaSHV-12 etc.
VIM	\b(?:bla)?VIM(?:[-_]?\d+)?\b	MBL family; (?:bla)? prefix accepts *bla*$_{VIM-2}$ *etc*.
IMP	\b(?:bla)?IMP(?:[-_]?\d+)?\b	Word-boundary anchored to avoid matching "imipenem" or "IMPACT"; (?:bla)? prefix accepts blaIMP-1 etc.
KPC	\b(?:bla)?KPC(?:[-_]?\d+)?\b	Uncommon in India; included for completeness. (?:bla)? prefix accepts bla .
mecA	\bmecA\b	MRSA marker; exact case-insensitive token. The trailing \b prevents partial matches such as *mecAB*.
vanA	\bvanA\b	VRE marker; exact case-insensitive token.
MCR-1	\b(?:mcr|MCR)[-_]?1\b	Plasmid-mediated colistin-

		resistance allele MCR-1 specifically. By design, the trailing \b excludes longer alleles such as mcr-10 and mcr-11; broader MCR family capture is not implemented in this dataset because no other mcr allele was reported in any curated ICMR-AMRSN summary record.

Note on regex correctness: The patterns above were validated against representative gene labels extracted from the source ICMR-AMRSN PDFs (*n*=34 *bla*-prefixed tokens identified during validation). The optional (?:bla)? prefix on beta-lactamase patterns ensures that conventionally-written tokens such as "*bla*$_{\text{NDM-1}}$", "*bla*$_{\text{OXA-48}}$", and "*bla*$_{\text{CTX-M-15}}$" are captured. The *mcr-1* pattern is intentionally allele-specific; broader MCR-family capture would require a distinct pattern and is not implemented because no other mcr allele was reported in any curated organism-year record. All patterns are applied case-insensitively (re.IGNORECASE).

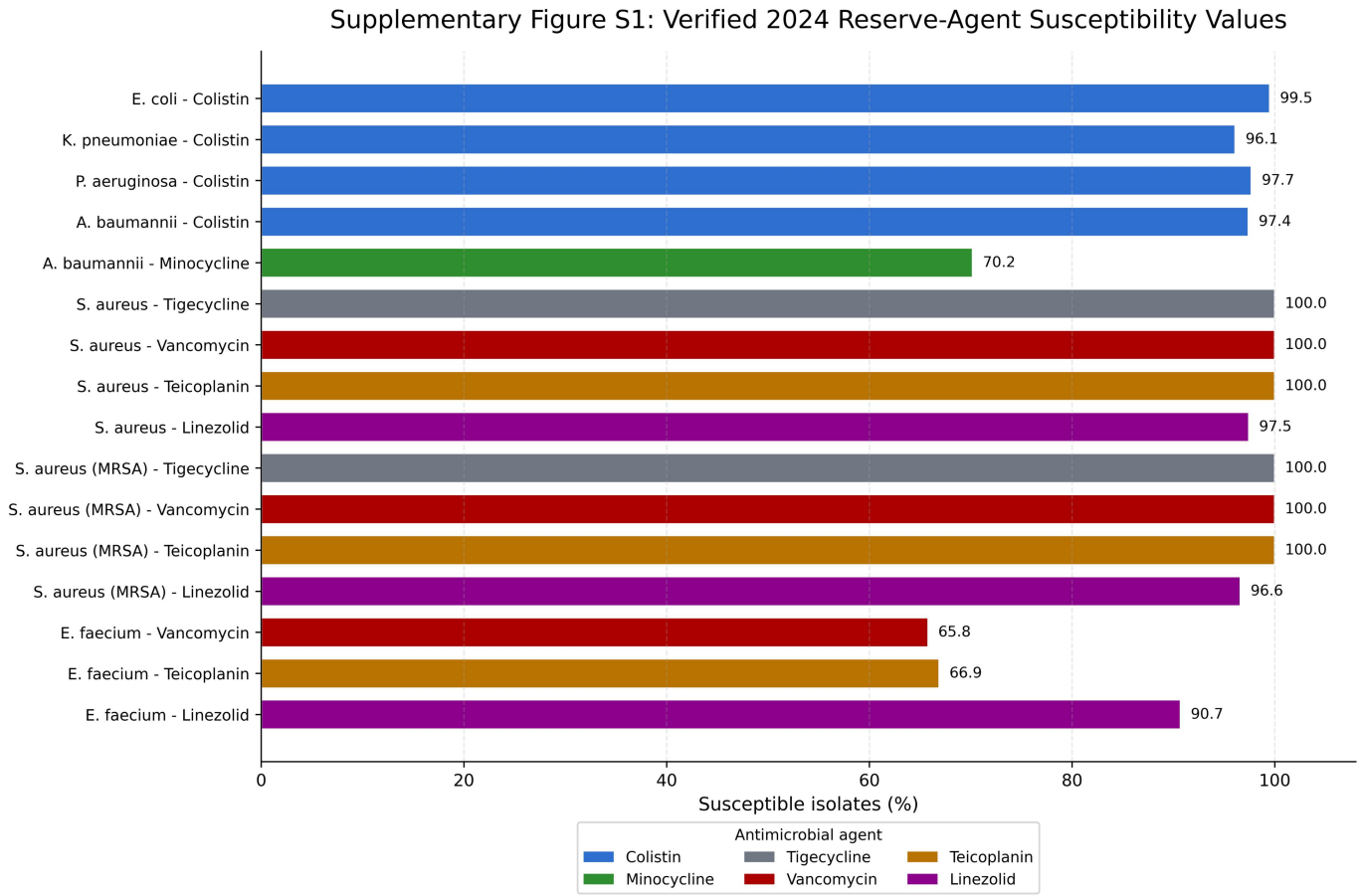
Numeric prevalence extraction: a percentage value occurring within 0–20 characters after a matched gene token (regex r"(\d+\.?\d*)\s*%") was captured as the reported prevalence. When no percentage was present in the source text, the gene was recorded as “Detected” rather than assigned a numeric prevalence.

Curated-CSV column dictionary

Column	Type	Description
record_id	integer	Unique row identifier (1–44).
organism	string	One of: *Escherichia coli*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Enterococcus faecium*.
report_year	integer	Four-digit publication year of the source ICMR-AMRSN annual report (2017–2024).
specimen_type	string	Clinical specimen category as reported in source summary (blood, respiratory, urine, etc.).
clinical_setting	string	ICU / non-ICU / not specified.
total_isolates	integer or null	Number of culture-positive isolates if reported; null where not stated.
gene_summary_text	string	Verbatim gene-related text

		extracted from the source summary.
gene_categories_detected	list of strings	Output of regex classifier (*e.g.*, ["NDM", "OXA-23-like"]).
gene_prevalence_pct	dict[gene -> float] or null	Captured numeric prevalence values per gene where available.
susceptibility_summary_text	string	Verbatim susceptibility text extracted from the source summary.
reserve_agent_susceptibility_pct	dict[agent -> float] or null	Captured numeric susceptibility percentages for agents of interest.
source_pdf_filename	string	Filename of the ICMR-AMRSN annual report PDF used as the source document.
source_page_number	integer	Page number within the source PDF where the data point was located.
curation_notes	string	Free-text annotation by the curator (*e.g.*, “co-carriage reporting”).

Supplementary Figure 1 and Supplementary Table 7



Primary 2024 values only. Source strata differ by pathogen; Gram-negative colistin is reported as intermediate susceptibility in the source report.

Supplementary Figure 1. Verified 2024 reserve-agent susceptibility values from ICMR-AMRSN 2024 source tables. Bars show percentage susceptible isolates for extractable pathogen-agent combinations in the primary 2024 aggregate; source strata differ by pathogen as listed in Supplementary Table 7. For Gram-negative organisms, colistin values are the source-reported intermediate-susceptibility percentages. MRSA, methicillin-resistant *Staphylococcus aureus*.

Supplementary Table 7. Verified 2024 reserve-agent susceptibility values used for Supplementary Figure 1.

Pathogen	Source stratum	Agent	Susceptible isolates (%)	Source table/page	Note
E. coli	All specimens except urine/faeces	Colistin	99.5	ICMR-AMRSN 2024, Table 2.1/2.2, physical pp. 56-57	Colistin reported as percentage intermediate susceptibility.
K. pneumoniae	All specimens except urine/faeces	Colistin	96.1	ICMR-AMRSN 2024, Table 2.1/2.3, physical pp. 56-58	Colistin reported as percentage intermediate susceptibility.
P. aeruginosa	All specimens except faeces	Colistin	97.7	ICMR-AMRSN 2024, Table 3.1, physical p. 102	Colistin reported as percentage intermediate susceptibility.
A. baumannii	All specimens except faeces	Colistin	97.4	ICMR-AMRSN 2024, Table 3.4, physical p. 105	Colistin reported as percentage intermediate

					susceptibility of *Acinetobacter* spp.
A. baumannii	All specimens except faeces	Minocycline	70.2	ICMR-AMRSN 2024, Table 3.4, physical p. 105	Minocycline value from the same 2024 Table 3.4 source stratum as the *A. baumannii* colistin value.
S. aureus	All samples, year-wise trend	Tigecycline	100.0	ICMR-AMRSN 2024, Table 6.4, physical p. 148	All *S. aureus*, not limited to MRSA.
S. aureus	All samples, year-wise trend	Vancomycin	100.0	ICMR-AMRSN 2024, Table 6.4, physical p. 148	All *S. aureus*, not limited to MRSA.
S. aureus	All samples, year-wise trend	Teicoplanin	100.0	ICMR-AMRSN 2024, Table 6.4, physical p. 148	All *S. aureus*, not limited to MRSA.
S. aureus	All samples, year-wise trend	Linezolid	97.5	ICMR-AMRSN 2024, Table 6.4, physical p. 148	All *S. aureus*, not limited to MRSA.
S. aureus (MRSA)	All samples, year-wise MRSA trend	Tigecycline	100.0	ICMR-AMRSN 2024, Table 6.9, physical pp. 153-154	MRSA subset.
S. aureus (MRSA)	All samples, year-wise MRSA trend	Vancomycin	100.0	ICMR-AMRSN 2024, Table 6.9, physical pp. 153-154	MRSA subset.
S. aureus (MRSA)	All samples, year-wise MRSA trend	Teicoplanin	100.0	ICMR-AMRSN 2024, Table 6.9, physical pp. 153-154	MRSA subset.
S. aureus (MRSA)	All samples, year-wise MRSA trend	Linezolid	96.6	ICMR-AMRSN 2024, Table 6.9, physical pp. 153-154	MRSA subset.
E. faecium	All specimens except urine	Vancomycin	65.8	ICMR-AMRSN 2024, Table 6.12, physical p. 157	Matches 34.2% VRE statement in the 2024 report (100 - 34.2 = 65.8).
E. faecium	All specimens except urine	Teicoplanin	66.9	ICMR-AMRSN 2024, Table 6.12, physical p. 157	All-specimen *E. faecium* value from ICMR-AMRSN 2024 Table 6.12.
E. faecium	All specimens except urine	Linezolid	90.7	ICMR-AMRSN 2024, Table 6.12, physical p. 157	All-specimen *E. faecium* value from ICMR-AMRSN 2024 Table 6.12.