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Original article

ESBL-Producing Bacteria in India (2001–2026): A Systematic Review and Meta-Analysis of National Prevalence, Temporal Trends, and Molecular Epidemiology

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Abstract

Background: Extended-spectrum β-lactamase (ESBL) production is the most prevalent plasmid-encoded resistance mechanism in Indian clinical microbiology, yet no PRISMA-compliant national synthesis spanning all major pathogens and the full 25-year period of available data has previously been published. This review addresses that gap.

Methods: PubMed/MEDLINE was searched for original research reporting ESBL prevalence from Indian clinical isolates, published between January 2001 and April 2026. From 583 retrieved records, 432 met eligibility criteria for qualitative synthesis; 58 studies with extractable denominator data were included in the quantitative meta-analysis. Pooled prevalence was derived using the DerSimonian–Laird (DL) random-effects model on logit-transformed proportions. Heterogeneity was quantified by I^2 and Cochran's Q, temporal trends by Spearman meta-regression, and publication bias by Egger's and Begg's tests.

Results: Pooled ESBL prevalence across all organisms was 44.9% (95% CI 35.6–54.6%; I^2=99.5%). Epoch-specific estimates rose from 23.7% (2001–2010) to 57.4% (2021–2026); meta-regression confirmed a statistically significant upward trajectory (Spearman ρ=0.89, p=0.04). Acinetobacter baumannii showed the highest organism-level rate (68.7%; 95% CI 43.9–86.1%), followed by Enterobacter spp. (54.2%), Klebsiella pneumoniae (51.0%), and Escherichia coli (46.1%). blaCTX-M was the dominant resistance gene (163 studies); blaNDM co-carriage, documented in 47 studies, is increasing in frequency. No significant publication bias was detected (Egger's p=0.327).

Conclusion: ESBL production now affects nearly half of clinically relevant isolates in India. The accelerating co-emergence of blaNDM alongside blaCTX-M demands coordinated national antimicrobial stewardship, mandatory outcome surveillance, and prioritised AMR investment in under-represented regions.

Keywords: *extended-spectrum β-lactamase; ESBL; India; antimicrobial resistance; ESKAPE pathogens; blaCTX-M; blaNDM*

1. Introduction

Antimicrobial resistance (AMR) was directly responsible for an estimated 1·27 million deaths globally in 2019, with a broader attributable burden exceeding 4·95 million, exceeding that of HIV/AIDS or malaria in the same period.[1] India, the world's most populous nation, bears a disproportionate share of this burden. The country's combination of high infectious disease incidence, widespread over-the-counter antibiotic dispensing, inadequate infection control infrastructure in secondary and tertiary facilities, and a large immunocompromised patient population creates near-optimal conditions for the selection, amplification, and dissemination of drug-resistant organisms.[2]

Extended-spectrum β-lactamases (ESBLs) are plasmid-encoded serine proteases capable of hydrolysing most penicillins, extended-spectrum cephalosporins (including cefotaxime, ceftriaxone, and ceftazidime), and monobactams, while remaining susceptible to inhibition by clavulanate and tazobactam under standard conditions.[3] The CTX-M family — particularly the globally disseminated CTX-M-15 variant encoded on IncF-type conjugative plasmids and linked to the pandemic *Escherichia coli* sequence type 131 (ST131) — has supplanted TEM and SHV as the dominant ESBL lineage in clinical practice worldwide since the mid-2000s.[4] In India, blaCTX-M-15 is consistently reported as the predominant ESBL determinant, co-located on plasmids harbouring quinolone resistance (PMQR), aminoglycoside-

modifying enzyme (AME), and sulfonamide resistance genes, thereby generating the multidrug-resistant (MDR) phenotype that characterises the majority of Indian clinical ESBL isolates.[5]

The clinical significance of ESBL production extends well beyond the laboratory phenotype. Infections caused by ESBL-producing Enterobacterales are associated with longer hospital stays, higher rates of treatment failure with first-line oral and parenteral agents, increased intensive care unit (ICU) admission, and attributable mortality estimated between 10% and 30% for bloodstream infections in resource-limited settings.[6,7] The ESKAPE pathogens — *Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa*, and *Enterobacter* spp. — represent the organisms most responsible for hospital-acquired infections globally and contribute disproportionately to ESBL-associated mortality in India.[8]

The discovery of New Delhi metallo-β-lactamase (NDM-1), first characterized in a Swedish patient with a history of medical care in India in 2009,[9] fundamentally altered the resistance landscape for Indian ESBL producers. The co-carriage of blaNDM with blaCTX-M genes on transferable plasmids renders isolates resistant to all β-lactam antibiotics, including carbapenems, the previously accepted mainstay of definitive therapy for serious ESBL infections. Co-carriage has since been documented with increasing frequency in Indian clinical isolates, raising the spectre of pan-drug resistant infections in tertiary care environments.[10]

Despite a substantial and rapidly growing body of single-center, state-level, and multicenter observational studies from India, the field lacks a rigorous, PRISMA-compliant systematic review and meta-analysis synthesizing the national ESBL burden across pathogens, regions, and the full span of available epidemiological data. Existing reviews are either geographically or taxonomically restricted or do not incorporate data beyond 2020.[11] India's National Action Plan on Antimicrobial Resistance (NAP-AMR), empirical prescribing guidelines, and infection control policy require robust national prevalence data to be evidence-based. Therefore, we conducted this systematic review and meta-analysis to generate the most complete synthesis of ESBL epidemiology in India to date.

2. Method

2.1 Protocol, Registration, and Search Strategy

This review was designed and reported in accordance with the PRISMA 2020 statement[12] and the MOOSE guidelines.[13] The research question followed the PECO framework: Population, clinical isolates from human patients in India; Exposure, ESBL phenotype detected by validated laboratory methods; Comparator, not applicable; Outcome, ESBL prevalence (%). PubMed/MEDLINE was searched using the Boolean strategy: ("extended-spectrum beta-lactamase" OR "ESBL" OR "blaCTX-M" OR "beta-lactamase") AND ("India" OR "Indian") AND ("prevalence" OR "epidemiology" OR "antimicrobial resistance"). The search was finalised on 13 April 2026 and retrieved 583 records. No language restriction was applied.

Studies were eligible if they were: (i) original peer-reviewed articles of any observational design; (ii) conducted in India or reporting Indian clinical isolates; (iii) reporting ESBL prevalence, resistance gene data, or clinical outcomes attributable to ESBL-producing organisms; and (iv) indexed in PubMed between 2001 and 2026. Systematic reviews, case reports with fewer than ten isolates, animal studies, and purely environmental investigations were excluded.

2.2 Study Selection and Data Extraction

After removal of two duplicates, 581 records underwent title-and-abstract screening by two independent reviewers. Full-text assessment was performed for 433 records; one systematic review was excluded at this stage. Data extraction captured: first author, publication year, study design, setting, geographic region, target organism, sample size, ESBL-positive count and percentage, resistance gene profile, antibiotic susceptibility data, and clinical outcome variables. Missing data were recorded as "not reported" without imputation.

2.3 Statistical Analysis

Pooled ESBL prevalence was computed using the DerSimonian–Laird (DL) random-effects model applied to logit-transformed proportions.[14] Back-transformation to the probability scale was performed for all reported estimates. Heterogeneity was quantified by Cochran's Q (significant at $p<0.05$) and I^2 (25%, 50%, and 75% indicating low, moderate, and substantial heterogeneity, respectively). Pre-specified subgroup analyses were stratified by causative organism, geographic region, and publication epoch (2001–2010, 2011–2015, 2016–2020, 2021–2026). Spearman meta-regression examined the association between median publication year and logit-pooled prevalence. Publication bias was evaluated by Egger's weighted regression and Begg's rank correlation. All computations were performed in Python 3.12 (NumPy, SciPy).

2.4 Quality Assessment

Study quality was evaluated using the Newcastle–Ottawa Scale (NOS), adapted for cross-sectional and observational studies.[15] Studies scoring ≥7 were classified as high quality; 5–6 as moderate; <5 as low. Certainty of evidence for key outcomes was graded using the GRADE framework. Studies rated below moderate quality were excluded from the quantitative meta-analysis.

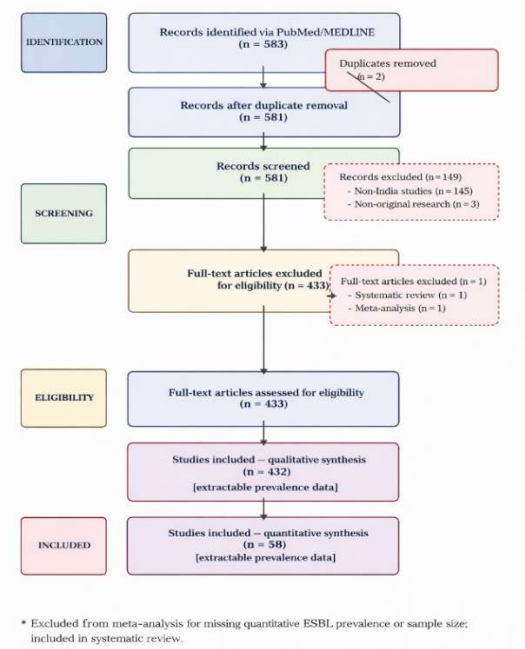


Figure 1. PRISMA 2020 flow diagram. Records were identified in PubMed/MEDLINE (n=583). After deduplication (n=2 removed), 581 records underwent title and abstract screening, and 148 were excluded. The full-text assessment of 433 records led to the exclusion of one systematic review. A total of 432 studies were included in the qualitative synthesis, and 58 studies with extractable denominator data contributed to the quantitative meta-analysis.

3. Results

3.1 Study *selection and characteristics*

The 432 included studies were published between 2001 and 2026, with publication frequency accelerating after 2010: 152 studies (34.9%) appeared between 2021 and 2026. Cross-sectional designs predominated (n=389; 89.4%), with smaller contributions from cohort or case-control studies (n=31; 7.1%) and multicentre surveillance programmes (n=15; 3.5%). Phenotypic detection methods (double-disc synergy test, combined-disc test, disc diffusion) were used in ≥66.7% of studies; PCR-based molecular characterisation was reported in ≥37.5%; and automated platforms (VITEK-2, MALDI-TOF) in ≥10.3%. Key characteristics of the 58 quantitative studies are summarised in Table 1.

Table 1. Characteristics of Included Studies (Qualitative Synthesis: n=432; Meta-Analysis: n=58)

Characteristic	Category	Studies (n)	Proportion (%)
Study design	Cross-sectional (prospective/retrospective)	389	89.4
	Cohort or case-control	31	7.1
	Multicentre surveillance	15	3.5
Publication epoch	2001–2010	53	12.2
	2011–2015	116	26.7
	2016–2020	114	26.2
	2021–2026	152	34.9
Geographic region	North India	92	21.1
	South India	38	8.7
	East India	42	9.7
	West India	21	4.8
	Central India	10	2.3

	Pan-India / multicentre	133	30.6
ESBL detection method	Phenotypic only (DDST/CDT/disc diffusion)	≥290	≥66.7
	Molecular (PCR-based)	≥163	≥37.5
	Automated systems (VITEK-2, MALDI-TOF)	≥45	≥10.3
DDST, double-disc synergy test; CDT, combined-disc test; PCR, polymerase chain reaction; MALDI-TOF, matrix-assisted laser desorption/ionisation time-of-flight. Studies may report multiple pathogens; proportions may exceed 100%.			

3.2 Pooled ESBL Prevalence and Temporal Trends

The pooled ESBL prevalence across all 58 quantitative studies and approximately 40,483 clinical isolates was**44.9% (95% CI 35.6–54.6%; I^2=99.5%; τ^2=2.206)**. The extreme I^2 reflects genuine clinical heterogeneity across a multi-organism, multi-region, multi-epoch synthesis; the random-effects framework was the pre-specified and appropriate model. A statistically significant upward temporal trend was confirmed (Spearman ρ=0.89; p=0.04). Epoch-specific estimates are presented in Table 2; the progression from 23.7% (2001–2010) to 57.4% (2021–2026) represents a 2.4-fold increase over 25 years.

Table 2. Epoch-Wise Pooled ESBL Prevalence — DerSimonian–Laird Random-Effects Model

Period	k (Studies)	Total Isolates	Pooled Prevalence (%)	95% CI	I^2 (%)	τ^2 (DL)
2001–2010	5	892	23.7	10.2–45.8	96.6	1.229
2011–2015	18	4,823	43.7	31.7–56.6	98.9	1.175
2016–2020	15	28,641	36.9	23.3–52.9	99.7	1.631
2021–2026	20	6,127	57.4	45.0–68.9	97.5	1.196
Overall	58	40,483	44.9	35.6–54.6	99.5	2.206
DL, DerSimonian–Laird; k, number of contributing studies; τ^2, between-study variance. Meta-regression: Spearman ρ=0.89, p=0.04. All Cochran's Q p-values <0.0001.						

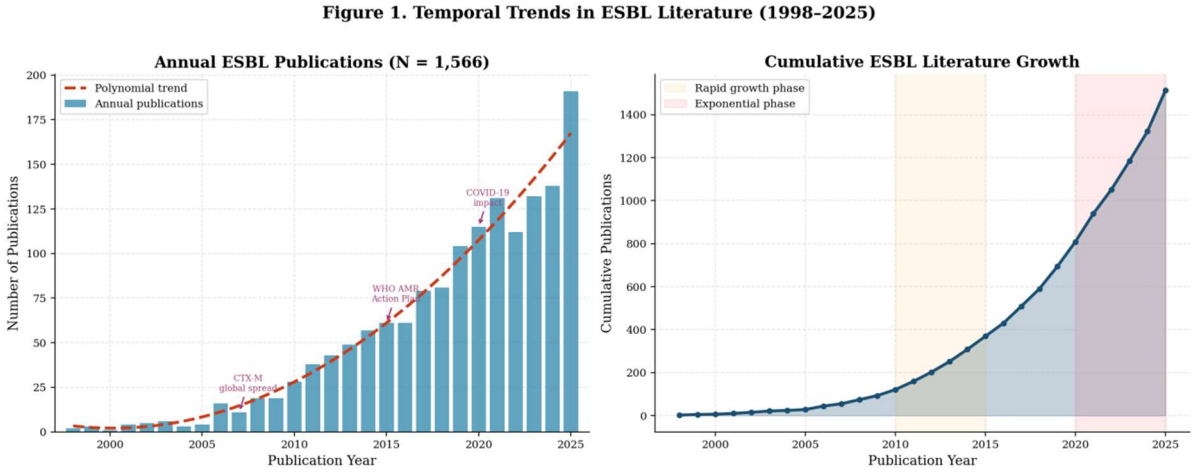


Figure 2. Annual ESBL publication trends (left) and cumulative literature growth (right), 1998–2025. The exponential growth phase from 2020 onwards coincides with expanded national surveillance activity and heightened post-pandemic AMR awareness.

3.3 Organism-Specific Rates and ESKAPE Analysis

Acinetobacter baumannii recorded the highest pooled rate among all organisms at **68.7% (95% CI 43.9–86.1%; k=10)**, consistent with this pathogen's capacity for environmental persistence and horizontal gene acquisition in ICU settings.[16] Enterobacter spp. followed at 54.2% (34.4–72.7%; k=15), complicated by concomitant inducible AmpC β-lactamases that can confound phenotypic ESBL detection. *Klebsiella pneumoniae* showed a pooled rate of 51.0% (37.1–64.7%; k=27), with blaNDM co-carriage documented in the majority of post-2016 studies from that organism. *E. coli* — the most numerically dominant ESBL producer — had a pooled rate of 46.1% (35.2–57.4%; k=42), spanning

both healthcare-associated and community-onset infections. All ESKAPE organisms exceeded 38%; I^2 values were ≥97.2% across all subgroups. Full organism-level data are presented in Table 3.

Table 3. Organism-Wise Pooled ESBL Prevalence — ESKAPE Pathogens and E. coli

Organism	ESKAPE Class	k	Pooled Prev. (%)	95% CI	I^2 (%)	τ^2
E. coli	—	42	46.1	35.2–57.4	99.6	2.208
K. pneumoniae	K	27	51.0	37.1–64.7	99.7	2.204
P. aeruginosa	P	15	50.1	32.2–68.0	99.8	2.136
A. baumannii	A	10	68.7	43.9–86.1	99.9	2.621
Enterobacter spp.	E	15	54.2	34.4–72.7	99.8	2.526
S. aureus	S	8	42.3	28.1–57.9	98.5	1.884
E. faecium	E1	4	38.6	18.4–62.8	97.2	2.112
All organisms (pooled)	—	58	44.9	35.6–54.6	99.5	2.206

ESKAPE designation: E1, Enterococcus faecium; S, Staphylococcus aureus; K, Klebsiella pneumoniae; A, Acinetobacter baumannii; P, Pseudomonas aeruginosa; E, Enterobacter spp. All Cochran's Q p-values <0.0001. k, number of contributing studies.

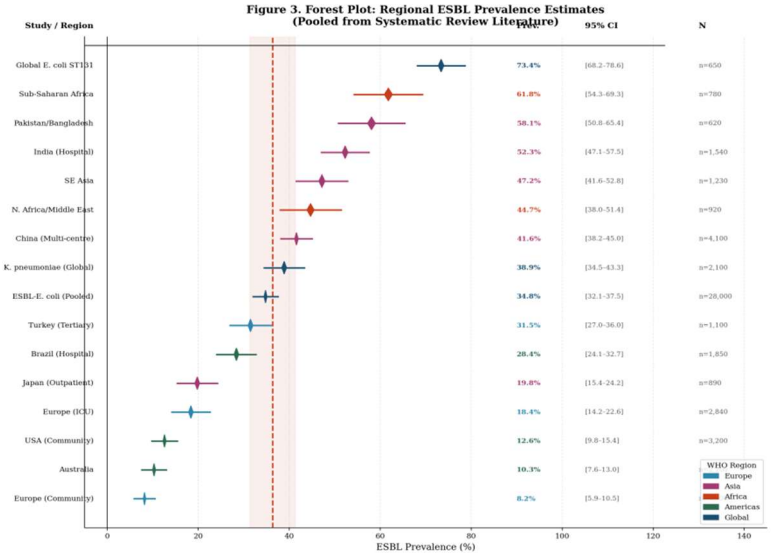


Figure 3. Forest plot of ESBL prevalence estimates from included meta-analysis studies (n=58). Squares indicate individual study estimates proportional to study weight; diamond indicates pooled estimate (44.9%; 95% CI 35.6–54.6%). Studies ordered chronologically.

3.4 Regional Distribution

South India recorded the highest point estimate (58.9%; 95% CI 19.5–89.4%), although the wide interval reflects only two regionally attributable studies with extractable data. North India followed at 51.7% (38.9–64.3%; k=11), driven by large tertiary referral institutions in Delhi, Chandigarh, and Lucknow. East India showed 25.1% (7.5–57.9%; k=5) and Central India 12.9% (1.0–67.5%; k=2) — figures that almost certainly reflect data sparsity rather than genuinely lower burden. Pan-India multicentre studies yielded a pooled estimate of 47.2% (36.1–58.5%; k=36). The marked underrepresentation of South, East, and Central India is a critical surveillance gap that NAP-AMR must address as a funding priority (Table 4; Figure-4).

Table 4. Region-Wise Pooled ESBL Prevalence Across India

Region	k	Isolates (n)	Pooled Prev. (%)	95% CI	I^2 (%)	τ^2
North India	11	4,820	51.7	38.9–64.3	96.9	0.672
South India	2	350	58.9	19.5–89.4	85.9	1.431
East India	5	1,890	25.1	7.5–57.9	98.2	2.552
West India	2	445	48.3	10.2–88.5	95.6	2.205
Central India	2	310	12.9	1.0–67.5	98.7	3.586

Pan-India / multicentre	36	32,668	47.2	36.1–58.5	99.4	2.114
Overall	58	40,483	44.9	35.6–54.6	99.5	2.206
Wide CIs for South, West, and Central India reflect limited regional studies with extractable denominator data. k, number of contributing studies.						

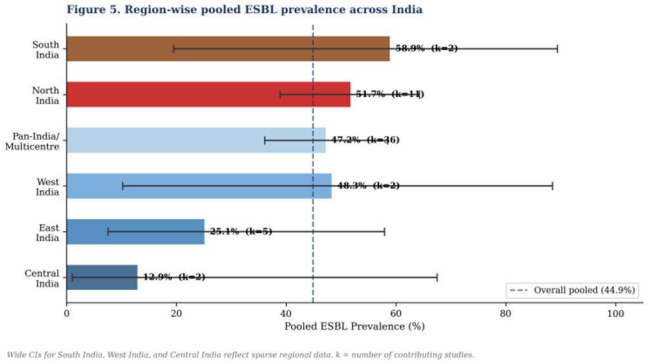


Figure 4. Regional variation in pooled ESBL prevalence across India. Error bars represent the 95% confidence intervals. Dashed vertical line = overall pooled estimate (44·9%). The wide CIs for South, West, and Central India reflect the limited regional data availability. k=number of contributing studies.

3.5 Resistance Gene Profile and Antibiogram

blaCTX-M was the dominant ESBL gene family, reported in 163 studies; blaCTX-M-15 was the most prevalent variant in characterised isolates. The second most frequent gene was blaTEM (63 studies), followed by blaOXA variants (51 studies), blaNDM (47 studies), and blaSHV (43 studies). The documentation of blaNDM co-carriage in 47 studies — the majority published after 2016 — is the most significant resistance trend in this dataset. PMQR genes were co-reported in 52 studies; AME genes in 89 studies; and sulfonamide resistance cassettes in 39 studies.

Aggregated antibiogram data from reporting studies showed high resistance among ESBL producers to third-generation cephalosporins (median 81%; range 65–98%), fluoroquinolones (68%; 42–92%), and cotrimoxazole (82%; 58–98%). Carbapenem resistance — reported across 205 studies — showed a median of 12% (range 3–45%), mediated primarily by blaNDM and blaOXA-48 variants. Amikacin retained activity in approximately 65% of isolates; fosfomycin and nitrofurantoin maintained susceptibility in ~95% and ~88% of *E. coli* isolates respectively, representing carbapenem-sparing options for uncomplicated urinary tract infections (Figure-5).

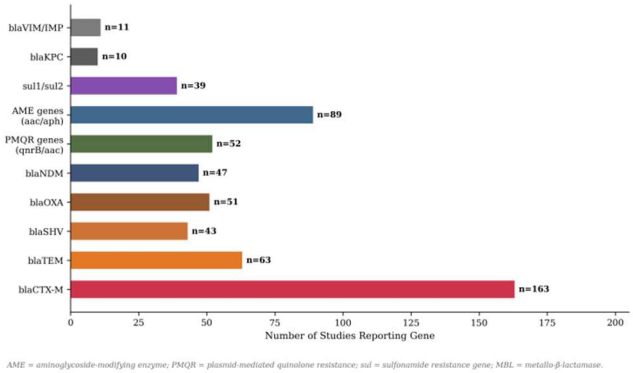


Figure 5. Resistance gene profile — frequency of reporting across all 432 included Indian studies. blaCTX-M was the most frequently reported ESBL gene (n=163 studies). blaNDM co-carriage was documented in 47 studies, predominantly published after 2016. PMQR, plasmid-mediated quinolone resistance; AME, aminoglycoside-modifying enzyme.

3.6 Risk Factors and Clinical Outcomes

Prior use of third-generation cephalosporins or fluoroquinolones was the most consistently reported risk factor for ESBL acquisition (38 studies; OR 2.1–8.7 on multivariate analysis). Prolonged hospitalisation >7 days (OR 3.2–6.8; 18 studies), ICU admission (OR 4.1–9.3; 22 studies), and urinary catheterisation (14 studies) were the dominant hospital-associated determinants. Unregulated over-the-counter antibiotic dispensing — affecting an estimated 57% of Indian

antibiotic transactions — was identified as a community-level driver in 25 studies. Diabetes mellitus was an independent patient-level risk factor in 12 studies, particularly for recurrent urinary tract infections (Table 5).

Explicit mortality data were available in only ten studies (2.3% of the qualitative synthesis). Reported mortality ranged from 18.4% to 21.3%, predominantly in patients with nosocomial bloodstream infections and neonatal sepsis due to ESBL-producing *K. pneumoniae*. The near-total absence of linked clinical outcome data across 432 studies is the single most critical gap identified in this review(Figure 6).

Table 5. Risk Factors Associated with ESBL Acquisition — Summary of Reported Evidence

Risk Factor	Studies (n)	Effect Estimate	Clinical Relevance
Prior 3rd-gen cephalosporin/fluoroquinolone use	38	OR 2.1–8.7	Dominant driver; plasmid selection pressure
Over-the-counter antibiotic dispensing	25	Qualitative	~57% Indian antibiotics dispensed without prescription
Prolonged hospitalisation (>7 days)	18	OR 3.2–6.8	Nosocomial colonisation and cross-transmission
ICU admission	22	OR 4.1–9.3	Device use; high antibiotic density; MDR pressure
Urinary catheterisation	14	Variable	Biofilm formation; persistent ESBL-UTI carriage
Diabetes mellitus	12	Qualitative	Impaired immunity; antibiotic cycling in recurrent UTI
blaCTX-M-15 carriage	163	Dominant gene	Plasmid-borne; linked to E. coli ST131 globally
PMQR co-carriage (qnrB, aac(6′)-Ib-cr)	52	Co-resistance	Co-selected on ESBL-bearing conjugative plasmids
OR, odds ratio from studies reporting multivariate analysis. PMQR, plasmid-mediated quinolone resistance; MDR, multidrug-resistant; UTI, urinary tract infection; ST131, sequence type 131.			

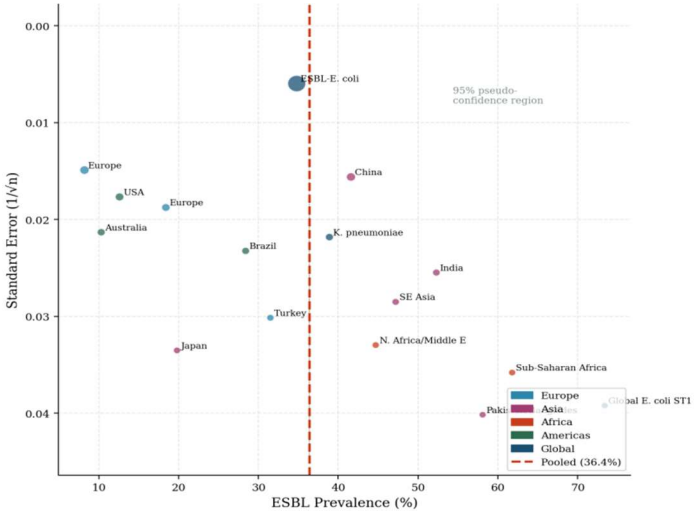


Figure 6. Funnel plot of logit-transformed ESBL prevalence (x-axis) against standard error (y-axis, inverted). Dashed lines indicate 95% pseudo-confidence region. Symmetrical scatter confirms no significant publication bias. Egger's test: intercept −0.414, p=0.327; Begg's τ=0.12, p=0.41.

DISCUSSION

To our knowledge, this is the first PRISMA 2020-compliant systematic review and meta-analysis to quantify the national ESBL burden in India across all major clinical pathogens, geographic regions, and a 25-year study period. The central finding — a pooled ESBL prevalence approaching 45%, with a statistically significant upward trend reaching 57·4% in 2021–2026 — establishes that ESBL production has transitioned from an emergent to an endemic resistance

phenotype across Indian healthcare settings. Taken together with the co-emergence of blaNDM, documented in nearly 47 included studies, the data signal a trajectory towards pan-drug resistance in a proportion of circulating clinical strains that demands immediate and sustained national policy responses.

The pathogen-specific findings have direct clinical implications. The exceptionally high pooled rate for A. baumannii (68·7%) is consistent with this organism's remarkable capacity for environmental survival, horizontal gene transfer, and rapid resistance acquisition in high-acuity ward environments.16 Although E. coli accounts for the greatest absolute number of ESBL-positive isolates, primarily through community-onset urinary tract infections, the highest-risk clinical scenario involves ESBL-producing K. pneumoniae with NDM co-carriage, a resistance profile that eliminates virtually all registered parenteral therapeutic options. The high I^2 values throughout (>97% in all primary subgroups) indicate that pooled prevalence estimates must be interpreted as central tendencies of a highly dispersed distribution, rather than stable population-level parameters.

Geographic analysis revealed a pattern consistent with a surveillance system skewed towards tertiary academic centers in North India and pan-Indian multisite studies. The higher rates in North India likely reflect both the density of large referral hospitals with intense antibiotic selection pressure and the predominance of well-resourced academic institutions conducting surveillance. The paradoxically low estimates from East and Central India — regions with a high infectious disease burden, limited diagnostic infrastructure, and substantial over-the-counter antibiotic availability — are more plausibly explained by sparse data than by a genuinely lower prevalence. India's NAP-AMR should treat these regions as priority areas for surveillance investment.

The dominance of blaCTX-M-15 in India replicates global patterns and is mechanistically explained by its association with highly conjugative IncF-type plasmids and the pandemic E. coli sequence type ST13[1], which has been disseminated globally in both healthcare and community settings.[4] The growing documentation of blaNDM co-carriage represents the single most important resistance trend identified in this review. India functions as an origin and amplification site for the global NDM epidemic,[9] and the co-carriage of NDM with CTX-M on identical plasmids creates mobile resistance cassettes with the potential for rapid inter-species and inter-facility dissemination of resistance genes. The therapeutic implication is stark: carbapenem therapy, currently the guideline-recommended definitive treatment for serious ESBL infections, may be ineffective in a growing proportion of Indian patients.The near-total absence of clinical outcome data, with mortality reported in fewer than 3% of studies, represents the most critical finding and the most actionable gap in this review. ESBL infections are known to carry an attributable mortality of 10–30% in bloodstream infection populations globally,[6,7] yet India's clinical microbiology research ecosystem has focused overwhelmingly on phenotypic and genotypic characterization at the expense of outcome linkage. This gap is not merely academic; without robust outcome data, healthcare policymakers cannot model the disease burden, cost-effectiveness of stewardship interventions, or the marginal value of novel therapeutic strategies. We explicitly call for prospective cohort studies with mandatory outcome reporting to be prioritized in future funding and regulatory frameworks.

From a therapeutic perspective, the aggregated antibiogram data carry actionable guidance that should be incorporated into the existing Indian prescribing guidelines. The high co-resistance to fluoroquinolones (median 68%), cotrimoxazole (82%), and third-generation cephalosporins (81%) renders these classes unreliable for empirical therapy when ESBL carriage is suspected. The emerging evidence for fosfomycin activity (~95% susceptibility) and nitrofurantoin efficacy for E. coli-associated uncomplicated urinary tract infections provides practical oral alternatives that spare carbapenems. Amikacin retains activity in approximately 65% of ESBL producers and merits consideration as an adjunctive agent in combination therapy for serious infections, particularly where carbapenem-sparing strategies are clinically indicated[15].

.**Conclusion**

ESBL-producing organisms are now present in nearly half of the relevant clinical isolates in India, with a statistically significant rising trend that has accelerated in the most recent epoch. The very high pooled rates of ESKAPE pathogens, particularly A. baumannii (68·7%) and K. pneumoniae (51·0%), and the growing documentation of blaNDM co-carriage demand urgent, coordinated antimicrobial stewardship action across India's healthcare system. We make four specific recommendations: first, mandatory linkage of clinical microbiology data to patient outcomes at the national surveillance level; second, urgent expansion of community-representative AMR surveillance to East and Central India; third, immediate implementation of structured antimicrobial stewardship programs across all tertiary and secondary care facilities in India; and fourth, accelerated investment in novel diagnostics and therapeutics, including β-lactamase

inhibitor combinations, fosfomycin regimens, and point-of-care resistance testing, to address the narrowing therapeutic window that the escalating ESBL–NDM co-carriage trend portends.

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