Scientific Research Publishing

Advances in Bioscience and Biotechnology, 2026, 17(11), 748-786
https://www.scirp.org/journal/abb
ISSN Online: 2156-8502
ISSN Print: 2156-8456

Determinants of Maternal-Neonatal Antimicrobial Resistance Transmission: A Systematic Review

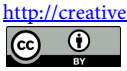
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How to cite this paper: Kabuya, K.E. and Sangany, C.M. (2026) Determinants of Maternal-Neonatal Antimicrobial Resistance Transmission: A Systematic Review. *Advances in Bioscience and Biotechnology*, **17**, 748-786.
https://doi.org/10.4236/abb.2026.1711044

Received: September 15, 2026
Accepted: November 21, 2026
Published: November 24, 2026

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Abstract

Background: Antimicrobial resistance (AMR) is an increasing threat to maternal and neonatal health, particularly in low- and middle-income countries. The maternal-neonatal interface represents an important pathway for AMR transmission, although detection of resistant organisms in neonates does not necessarily establish maternal transmission. **Methods:** This systematic review followed PRISMA 2020 and SWiM guidance. PubMed/MEDLINE, Embase, Scopus, and Web of Science were searched as core sources, and Google Scholar as a supplementary source, for studies published from January 2020 to December 2025. Evidence was classified as confirmed, probable, possible, or unestablished transmission and synthesized narratively, with risk-of-bias ratings used to weight the synthesis. **Results:** Of 1321 records identified, 276 duplicates were removed, 149 full texts were assessed, and 62 studies were included. Maternal colonization with ESBL-producing *E. coli* and *K. pneumoniae*, antimicrobial exposure, premature rupture of membranes, prematurity, low birth weight, prolonged hospitalization, invasive procedures, and neonatal intensive care were recurrently associated with AMR acquisition. Of 31 primary maternal-neonatal studies, only three provided direct transmission evidence (one confirmed, two probable), whereas the remainder provided associative evidence only, which does not establish maternal-to-neonatal transmission. Healthcare-associated transmission, WASH deficiencies, environmental contamination, agricultural and veterinary antimicrobial use, food exposure, and community antimicrobial access also emerged as important contextual determinants. Gram-negative organisms and ESBL and carbapenemase resistance determinants predominated. **Conclusion:** AMR transmission between mothers and neonates is multifactorial and influenced by maternal, neonatal, healthcare, environmental, and broader One Health determinants. Fu-

ture research should prioritize longitudinal paired sampling and molecular/genomic approaches to strengthen source attribution and inform integrated prevention strategies.

Keywords

Antimicrobial Resistance, Maternal-Neonatal Transmission, One Health, Neonatal Sepsis, Systematic Review, Narrative System

1. Introduction

Antimicrobial resistance (AMR) has emerged as one of the most pressing global public health threats of the twenty-first century, undermining the effectiveness of essential antimicrobial therapies and jeopardizing decades of progress in the prevention and treatment of infectious diseases [1] [2]. In 2019, bacterial AMR was estimated to be directly responsible for approximately 1.27 million deaths globally and associated with an estimated 4.95 million deaths overall, underscoring its substantial contribution to the global burden of disease [3] [4]. This burden is driven by complex and interconnected factors, including the inappropriate use of antimicrobials across human healthcare, veterinary medicine, and agricultural systems, which exert selective pressure that accelerates the emergence, amplification, and dissemination of resistant organisms [5] [6].

The consequences are particularly concerning in maternal and neonatal health, where effective antimicrobial therapy is essential for preventing severe morbidity and mortality [7]. Maternal bacterial infections, including sepsis and other obstetric infections, remain important contributors to maternal mortality globally, with direct obstetric infections estimated to account for approximately 10.7% of maternal deaths [8] [9]. Among neonates, sepsis caused by AMR, substantially limits empirical treatment options, prolongs hospitalization, increases healthcare costs, and is associated with a heightened risk of mortality, particularly in neonatal intensive care units (NICUs) [10]. Globally, AMR-associated neonatal sepsis is estimated to contribute to approximately 214,000 neonatal deaths annually [11] [12]. These substantial clinical consequences underscore the need to better understand the determinants and pathways through which resistant organisms and resistance determinants are acquired and potentially transmitted between mothers and their neonates [13].

The burden of AMR is disproportionately concentrated in low- and middle-income countries (LMICs), which account for up to 90% of global AMR-associated deaths [14]-[18]. This disproportionate burden is primarily caused by the determinants of health; One Health System including Human, Animal and Environmental Health [19]-[21]. In many resource-constrained settings, inadequate infection prevention and control (IPC) practices, deficiencies in water, sanitation, and hygiene (WASH) infrastructure, and the unregulated or poorly regulated

over-the-counter availability of antimicrobials create conditions conducive to the emergence, persistence, and dissemination of antimicrobial-resistant organisms (AROs) [22]-[25]. Beyond its immediate clinical consequences, AMR also imposes a substantial macroeconomic burden through increased healthcare expenditure, prolonged illness and hospitalization, treatment failure, and productivity losses [26]. Global projections suggest that AMR could generate up to US$1 trillion in additional healthcare costs by 2050, alongside substantial economic losses attributable to reduced productivity [27] [28].

Within this broader epidemiological context, the acquisition and potential transmission of AROs and antimicrobial resistance determinants at the maternal-neonatal interface represents an important yet comparatively understudied pathway of AMR dissemination [29]. The perinatal period constitutes a critical window of vulnerability during which neonates undergo initial microbial colonization and establishment of their early-life microbiome, with maternal microbial communities representing an important potential source of microorganisms acquired during this period [30]-[34]. Maternal colonization with resistant bacteria during pregnancy, the intrapartum period, and the immediate postpartum period may therefore contribute to early-life acquisition of AROs by neonates [35]-[38]. Evidence from diverse geographical settings has documented substantial maternal carriage of AROs, particularly in LMICs, and has reported concordance of resistant organisms between mothers and neonates, including evidence suggestive of transmission during childbirth or shortly thereafter [39] [40].

This early-life acquisition of AROs carries profound clinical ramifications. Neonates, particularly those born preterm or with low birth weight, possess immature immune architecture, rendering them exceptionally susceptible to invasive infections [41] [42]. Transmission of AMR across the maternal-neonatal interface occurs through interconnected processes of vertical transmission (VT) and horizontal gene transfer (HGT) [43] [44]. VT involves the transfer of maternal microorganisms to the neonate during birth and through breastfeeding and lactation, thereby contributing to early neonatal microbial colonization [45]-[47]. In contrast, HGT facilitates the acquisition and dissemination of AMR determinants between bacterial populations through mechanisms including conjugation, transduction, and transformation, with mobile genetic elements such as plasmids, transposons, and integrons contributing to the spread of resistance genes [48]-[52]. Although VT primarily establishes the neonatal microbial reservoir, HGT may subsequently facilitate the persistence and dissemination of resistance determinants within the neonatal gut and surrounding microenvironments [53]. These processes are influenced by interacting clinical, behavioural, and environmental factors, including antimicrobial exposure, microbial colonization, and selective pressures, which may modify the acquisition and transmission of AMR between mothers and neonates [54].

The identification of the key drivers of maternal-neonatal AMR transmission is urgently required to mitigate early-life microbial acquisition and reduce associ-

ated neonatal morbidity and mortality. This systematic review aims to synthesize the evidence on the determinants and pathways of AMR acquisition and transmission at the maternal-neonatal interface, with particular emphasis on vertical transmission, horizontal gene transfer, and the interacting clinical, behavioural, healthcare, and environmental factors influencing these processes.

2. Methods

2.1. Study Design and Reporting Guideline

This systematic review synthesized evidence on antimicrobial resistance (AMR) acquisition and transmission at the maternal-neonatal interface, focusing on vertical transmission, horizontal gene transfer, and relevant clinical, behavioural, healthcare-associated, and environmental determinants. The review was conducted and reported in accordance with the PRISMA 2020 Statement and Synthesis Without Meta-analysis (SWiM) guidelines, using a predefined protocol to guide study selection, risk-of-bias assessment, data extraction, and evidence synthesis.

The database search identified 1321 records; after removal of 276 duplicates, 1045 records underwent title and abstract screening. Of these, 896 were excluded, and 149 full-text articles were assessed for eligibility. Following full-text assessment, 87 articles were excluded, resulting in 62 studies included in the qualitative synthesis.

Evidence was synthesized across two complementary streams: 1) direct maternal-neonatal transmission, comprising primary studies with paired maternal-neonatal sampling or explicit dyadic linkages; and 2) contextual One Health evidence, comprising studies examining environmental, healthcare, agricultural, or animal-reservoir exposures associated with early-life AMR acquisition. Primary empirical studies provided the basis for transmission estimates, while secondary reviews were considered separately to contextualize broader patterns and avoid double-counting. Given the substantial clinical, microbiological, and methodological heterogeneity among studies, findings were synthesized narratively rather than quantitatively.

2.2. Information Sources and Search Strategy

A systematic search was conducted across the core databases PubMed/MEDLINE, Embase, Scopus and Web of Science, supplemented by Google Scholar for studies published between January 2020 and December 2025. Reference lists of included primary studies and relevant systematic reviews were manually screened for additional eligible literature.

Database-specific strategies utilized controlled vocabulary (MeSH, Emtree) and free-text terms covering four core concepts: 1) AMR and resistance determinants; 2) maternal-neonatal populations; 3) transmission pathways, colonization, and clinical infection; and 4) contextual healthcare, environmental, and One Health drivers.

Role of Google Scholar. Google Scholar was used strictly as a supplementary, not a core, search source. The four bibliographic databases above formed the core,

reproducible search from which the records in Figure 1 were derived. Google Scholar was added only to capture grey and regional literature (for example theses, preprints and institutional reports) that these databases index poorly. Because Google Scholar returns relevance-ranked results, does not support controlled vocabulary and does not allow complete export of results, it was searched with a predefined sequential screening protocol: each predefined keyword combination was run separately; results were screened in the order displayed, first by title, then by abstract, with full texts retrieved for potentially eligible records; and screening of a combination was stopped when the stopping rules set out were met. Records identified were de-duplicated against the database records and assessed against the same eligibility criteria. Because screening stopped before all retrieved results had been examined, and because ranking favours highly cited and openly accessible items, this part of the search cannot be exhaustively reproduced and carries a risk of selective retrieval and publication bias, which is addressed in Section 4.7.

2.3. Eligibility and Exclusion Criteria

Eligibility criteria were established a priori:

2.3.1. Inclusion Criteria

- Direct Maternal-Neonatal Evidence: Pregnant/postpartum women, neonates, or dyads evaluating colonization, AMR infection, gene transfer, or peripartum antibiotic exposure with required microbiological/genomic linkage for direct attribution.
- Contextual One Health Evidence: Environmental, wastewater, agricultural, food-chain, or animal reservoir studies relevant to maternal-neonatal exposure pathways.
- Study Designs: Primary empirical research (prospective/retrospective cohort, case-control, cross-sectional, surveillance, molecular/genomic, mixed-methods) was eligible. Secondary syntheses were restricted to contextual discussion and reference screening.

2.3.2. Exclusion Criteria

Studies were excluded if they met any of the following pre-specified criteria:

1) Irrelevant outcomes, scope, or populations lacking maternal, neonatal, or actionable One Health transmission data.

2) Editorials, commentaries, letters, conference abstracts, protocols, dissertations, theses, and duplicate cohort reports.

3) Methodological inadequacy in sampling, microbiological processing, or diagnostic criteria preventing quality assessment.

4) Non-English publications or non-retrievable full texts.

2.4. Study Selection

Retrieved records were imported into systematic review management software (Covidence/Rayyan) for deduplication. Title and abstract screening was con-

ducted independently by two reviewers against predefined eligibility criteria. Full-text articles of potentially eligible records were retrieved and independently assessed for inclusion by two reviewers. Discrepancies at any screening stage were resolved through consensus or adjudication by a third senior reviewer. Reasons for full-text exclusion were systematically recorded and documented in the PRISMA flow diagram.

2.5. Data Extraction and Evidence Classification

Data were extracted using a standardized, pre-tested data-extraction form. Key variables included bibliographic information, setting, study design, sample size, specimen types, laboratory/molecular techniques, bacterial species, AMR phenotypes, resistance determinants, maternal/neonatal outcomes, and plausible transmission pathways. To evaluate transmission strength, specific analytical variables were extracted: dual maternal-neonatal sampling status, organism concordance, phenotypic agreement, strain/genomic typing resolution (MLST, WGS), mobile element characterization, temporal plausibility, and exclusion of alternative acquisition sources. Each primary maternal-neonatal study was assigned to the highest applicable tier of a four-tier evidence hierarchy (**Figure 2**), applied independently by two reviewers with disagreements resolved by consensus. The tiers were defined a priori by the following eligibility criteria:

- Tier 1 - Confirmed transmission: (i) paired maternal-neonatal sampling (dyads); (ii) isolate- or strain-level genomic concordance (whole-genome sequencing with SNP or cgMLST identity, shared plasmid or mobile-element backbone, or high-resolution metagenomic strain sharing); (iii) temporal plausibility; and (iv) alternative post-natal acquisition sources considered.
- Tier 2 - Probable transmission: (i) paired sampling; (ii) the same species with a concordant resistance phenotype (antimicrobial susceptibility testing) or resistance gene within a defined temporal window; and (iii) a supportive intrapartum or epidemiological link (for example prolonged rupture of membranes), but without strain-level typing, so that post-delivery exposure cannot be excluded.
- Tier 3 - Possible transmission: maternal colonisation, infection or antimicrobial exposure in the perinatal period, with organisms relevant to neonatal infection, that is plausibly linked to neonatal carriage, infection or outcome, but without isolate-level matching between mother and neonate (including phenotype-only similarity without confirmed pairing and studies sampling only one member of the dyad).
- Tier 4 - Unestablished transmission: maternal-only (antenatal or community), neonatal-only, protocol, health-systems or non-bacterial studies with no maternal-neonatal linkage.

Throughout this review, Tiers 1 and 2 are termed direct transmission evidence, because they rest on paired isolates. Tiers 3 and 4 are termed associative evidence: they show that maternal exposure, colonisation or infection and neonatal out-

comes co-occur, but they do not establish that resistant organisms or genes passed from mother to neonate. Contextual One Health evidence is likewise treated as associative.

2.6. Data Synthesis

Due to substantial clinical and methodological heterogeneity across study designs, populations, laboratory methods, and reported outcomes, quantitative meta-analysis was not feasible. Evidence was synthesized using a structured thematic narrative synthesis following Synthesis Without Meta-analysis (SWiM) guidelines across four predefined thematic domains:

1) Determinants of maternal-neonatal AMR transmission (maternal colonization, antibiotic exposure, delivery mode, prematurity, invasive procedures, environmental/healthcare hygiene).

2) Transmission pathways and mechanisms (vertical transmission, postnatal transfer, breastfeeding, healthcare-associated, environmental dissemination).

3) AMR patterns and clinical impact (pathogens, MDR strains, resistance genes, neonatal morbidity/mortality, healthcare burden).

4) Prevention and control strategies (antimicrobial stewardship, screening, IPC measures, genomic surveillance, One Health interventions).

Studies were compared narratively within and across these thematic domains to identify recurring determinants, similarities, differences, and knowledge gaps.

2.7. Methodological Quality and Risk-of-Bias Assessment

Formal critical appraisal was conducted independently by two reviewers using design-specific assessment tools:

- Observational Studies: Evaluated using relevant Joanna Briggs Institute (JBI) Critical Appraisal Checklists.
- Mixed-Methods Studies: Evaluated using the Mixed Methods Appraisal Tool (MMAT, 2018 version).
- Surveillance and Molecular/Genomic Studies: Appraised using modified JBI tools focused on sampling validity and typing resolution.

Discrepancies were resolved via consensus. Overall risk-of-bias ratings (low, moderate, high) were assigned to each study and applied in the narrative synthesis according to the following weighting principle: (i) conclusions about direct transmission were based on Tier 1-2 studies at low or moderate risk of bias, with low-risk genomic studies given the greatest weight; (ii) moderate-risk studies contributed to judgements of consistency across settings but did not alone support any directional claim; (iii) high-risk studies were tabulated and described but were not used as the sole support for any conclusion and were treated as hypothesis-generating; (iv) where studies of different quality disagreed, the higher-quality evidence took precedence; and (v) protocols and other documents without outcome data could not be appraised and provided descriptive context only. The ratings are reported in Section 3.3. Publication bias was

not formally assessed via funnel plots because of the narrative nature of the synthesis; its likely impact, including that of the supplementary Google Scholar search, is considered in Section 4.7.

2.8. Certainty of Evidence

A formal assessment using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach was not conducted due to substantial heterogeneity in study designs, outcome metrics, and microbiological techniques. Interpretation of certainty was instead based on evidence consistency across independent settings, study design rigor, biological plausibility, and thematic convergence.

2.9. PRISMA Flow Diagram

The study selection process, from initial retrieval through duplicate removal, title/abstract screening, full-text evaluation, and final inclusion in the qualitative synthesis, is documented in the PRISMA 2020 flow diagram (Figure 1).

3. Results

3.1. Study Selection

The primary literature search across electronic databases identified 1,321 records. Following the removal of 276 duplicate records, 1,045 unique records underwent initial title and abstract screening. From this screening, 896 records were excluded for failing to meet the predefined inclusion criteria. The full texts of the remaining 149 articles were rigorously assessed for eligibility, resulting in the exclusion of 87 articles for predefined methodological or scope-related reasons. Consequently, 62 studies met all eligibility criteria and were included in the final qualitative synthesis (Figure 1). Google Scholar served only as a supplementary source (Section 2.2); Figure 1 reflects the four core databases.

The selected studies represented diverse geographical regions predominantly across Sub-Saharan Africa (SSA) and LMIC contexts encompassing various healthcare settings, study designs, and clinical populations. Collectively, they provide comprehensive evidence regarding the determinants of AMR transmission across maternal, neonatal, environmental, and One Health domains.

3.2. Characteristics of Included Evidence

3.2.1. Primary Maternal-Neonatal Studies

A total of 31 primary empirical studies evaluated maternal colonization, vertical transmission, and neonatal sepsis outcomes. The evidence base encompassed observational cross-sectional studies, prospective and retrospective cohorts, genomic epidemiology investigations, metagenomic analyses, and intervention trial protocols (Table 1). Each study was assigned to an evidence tier using the criteria in Section 2.5; the grade is shown in the final column of Table 1 and summarised in Section 3.4.

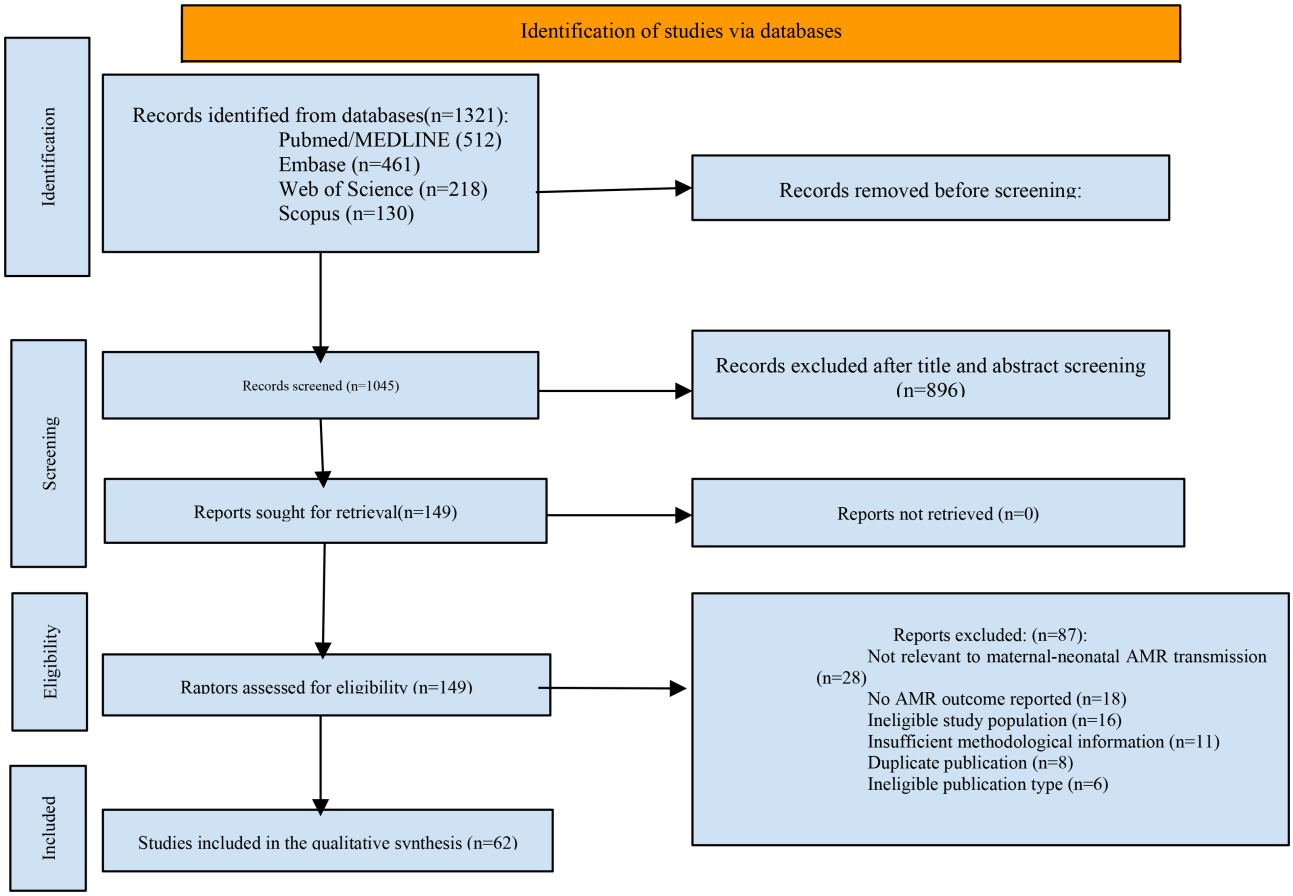


Figure 1. PRISMA 2020 flow diagram of study identification, screening, eligibility assessment and inclusion.

Table 1. Characteristics of primary maternal-neonatal studies (n = 31).

Author, Year	Country	Design	Population	Sample (n)	Organism	AMR Phenotype/Gene	Primary Outcome	Evidence Grade (Tier)
Mehari *et al.* (2025)	Ethiopia	Cross-sectional	Pregnant women (ANC)	384*	*E. coli*, *K. pneumoniae*	MDR, ESBL, Ampicillin-R	Asymptomatic bacteriuria	Tier 4 - Unestablished (maternal only)
Martin *et al.* (2023)	Zimbabwe	Mixed-methods	Pregnant women (ANC)	Protocol	*C. trachomatis*, *N. gonorrhoeae*	Fluoroquinolone-R, Macrolide-R	Antenatal STI/AMR diagnostics	Tier 4 - Unestablished (protocol)
Salissou (2023)	Zimbabwe	Retrospective	Pregnant women	215	*E. coli*, Gram-negatives	Cotrimoxazole-R, Ampicillin-R	Antenatal UTI	Tier 4 - Unestablished (maternal only)
Martin (2025)	Zimbabwe	Thesis/Cross-sectional	Antenatal attendees	450	*N. gonorrhoeae*, *E. coli*	MDR, Extended-spectrum Ceph-R	STI & maternal infection	Tier 4 - Unestablished (maternal only)
Olaru (2023)	Zimbabwe	Doctoral Thesis	Outpatients & women	612	*Enterobacterales*	ESBL, *bla*CTX-M	Community UTI/carriage	Tier 4 - Unestablished (maternal/community only)

Continued

Olaru *et al.* (2020)	Zimbabwe	Cohort Protocol	Outpatients with UTI	Protocol	*E. coli*, *K. pneumoniae*	ESBL, ARGs	UTI risk factor profiling	Tier 4 - Unestablished (protocol)
Yeta *et al.* (2021)	Zambia	Cross-sectional	Pregnant women (ANC)	280	*E. coli*, *S. aureus*	Cotrimoxazole-R, Ampicillin-R	Maternal UTI	Tier 4 - Unestablished (maternal only)
Olaru *et al.* (2022)	Zimbabwe	Cross-sectional	Primary care prescribers	185	N/A (prescribing patterns)	Empiric broad-spectrum usage	Empirical failure risk	Tier 4 - Unestablished (health systems)
Tumuhamye *et al.* (2021)	Uganda	Cross-sectional	Intrapartum women	400	*E. coli*, Gram-negatives	ESBL, *bla*CTX-M, MDR	Vaginal colonization	Tier 3 - Possible (intrapartum maternal reservoir; no neonatal isolates)
Bulabula (2020)	South Africa	Cohort/ Thesis	Pregnant women	350	*E. coli*, *K. pneumoniae*	ESBL, MDR	Maternal rectovaginal carriage	Tier 3 - Possible (maternal reservoir; no isolate-level pairing)
Dunlop (2022)	Global/SSA	Doctoral Thesis	Perinatal care cohorts	1200	*S. aureus*, Gram-negatives	MDR, Methicillin-R	Maternal post-op infection	Tier 4 - Unestablished (intervention; no linkage)
Sammarro *et al.* (2023)	Malawi	Longitudinal	Perinatal cohorts	680	*E. coli*	ESBL, *bla*CTX-M	Household & gut carriage	Tier 3 - Possible (maternal reservoir; no isolate-level pairing)
Nielsen (2024)	Malawi	Doctoral Thesis	Neonates & mothers	510	*K. pneumoniae*, *E. coli*	ESBL, 3rd-gen Ceph-R	Early-onset neonatal sepsis	Tier 3 - Possible (maternal-neonatal association; no isolate-level matching)
Taika (2022)	Malawi	Doctoral Thesis	Inpatient mothers & infants	180	*S. aureus*, CoNS	Methicillin-R (*mecA*), *pvl*	Staphylococcal colonization	Tier 3 - Possible (phenotypic concordance only; pairing/timing unconfirmed)
Mwandigha *et al.* (2020)	Tanzania	Cross-sectional	Pregnant women (ANC)	320	*Enterobacteriaceae*	ESBL, *bla*CTX-M	Gut colonization	Tier 4 - Unestablished (maternal only)
Blumenröder *et al.* (2023)	Tanzania	Cross-sectional	Mother-neonatal pairs	190	*E. coli*, *K. pneumoniae*	ESBL, MDR	Neonatal sepsis/carriage	Tier 2 - Probable (paired; species and phenotype concordant)

Continued

Quispe *et al.* (2020)	Peru	Cross-sectional	Pregnant women	160	*Enterobacteriales*	MDR, Ceftriaxone-R	Maternal bacteremia	Tier 4 - Unestablished (maternal only)
Amon (2021)	Malawi	Cohort	Pregnant women with HIV	115	HIV-1	NNRTI-R mutations	Antiretroviral resistance	Tier 4 - Unestablished (viral dynamics; no linkage)
Wadonda-Kabondo (2025)	Malawi	Cross-sectional	ART-naive pregnant women	210	HIV-1	Transmitted drug resistance	Baseline maternal resistance	Tier 4 - Unestablished (viral dynamics; no linkage)
Sebastiao *et al.* (2021)	Angola	Cross-sectional	Pregnant women with HIV	175	HIV-1	Broad NNRTI/NRTI-R	Maternal HIV failure	Tier 4 - Unestablished (viral dynamics; no linkage)
Abu Shqara *et al.* (2023)	International	Retrospective	Intrapartum mothers	310	*E. coli*, GBS	Ampicillin-R, Gentamicin-R	Maternal bacteremia, PROM	Tier 3 - Possible (maternal isolates only)
Seale *et al.* (2025)	East Africa	Nested case-control	Pregnant women & fetus	1100	*E. coli*, *S. aureus*	MDR, 3rd-gen Ceph-R	Stillbirth, neonatal death	Tier 3 - Possible (maternal infection associated with outcome; no isolate-level matching)
Solomon *et al.* (2021)	Multi-site	Cross-sectional	Neonates	450	Gram-negative bacilli	Gentamicin-R, Ceftriaxone-R	Neonatal sepsis	Tier 4 - Unestablished (neonatal only)
Bah *et al.* (2023)	The Gambia	Genomic Cohort	Hospitalized neonates	210	*K. pneumoniae*, *A. baumannii*	Carbapenemase (*bla*NDM), MDR	Late-onset NICU sepsis	Tier 4 - Unestablished (neonatal/nosocomial only)
Berckenhagen (2025)	Tanzania	Doctoral Thesis	Neonates & mothers	380	*E. coli*, *K. pneumoniae*	ESBL, MDR	Neonatal gut colonization	Tier 2 - Probable (genomic data, but environmental sources not excluded)
Epuitai *et al.* (2025)	Uganda	Mixed-methods	Postpartum women	290	N/A (prophylactic practices)	Unindicated antibiotic pressure	Driver of maternal resistance	Tier 4 - Unestablished (prescribing; no linkage)
Beales *et al.* (2025)	Malawi	Clinical Protocol	Mothers & neonates	Protocol	*S. aureus*, Gram-negatives	Chlorhexidine antisepsis	Prevention of vertical transfer	Tier 4 - Unestablished (protocol)

Continued

Kim *et al.* (2025)	Multi-site	Trial Protocol	Laboring women	Protocol	Maternal/neonatal microbiome	Azithromycin exposure profile	Microbiome selection	Tier 4 - Unestablished (protocol)
Tshivhase (2024)	South Africa	Doctoral Thesis	Pregnant women	165	GBS, Gram-negatives	MDR, Ampicillin-R	Stillbirth, maternal infection	Tier 3 - Possible (maternal isolates; stillbirth outcome)
Ainomugisha *et al.* (2024)	Uganda	Cross-sectional	Women with PROM	140	*E. coli*, *K. pneumoniae*	MDR, ESBL	Intra-amniotic sepsis	Tier 3 - Possible (amniotic colonisation; no neonatal isolates)
Carvalho *et al.* (2022)	Multi-country	Metagenomic Cohort	Mother-neonatal pairs	310	*E. coli*, *K. pneumoniae*	*bla*CTX-M, *bla*TEM, ARGs	Gut colonization	Tier 1 - Confirmed (paired; genomic relatedness)

3.2.2. Primary Contextual One Health Studies

Thirteen primary investigations assessed environmental reservoirs, food supply chains, agricultural exposure pathways, and community WASH infrastructure that directly or indirectly influence the baseline resistance profile of pregnant women and neonates (Table 2).

Table 2. Characteristics of contextual one health studies (n = 13).

Author, Year	Setting	Reservoir	Organism/ARG	AMR Finding	Relevance to Maternal-Neonatal Interface	Evidence Classification
Valia (2025)	Burkina Faso	Rural community & ANC	Human gut/sanitation; ESBL-*Enterobacteriaceae*	High community ESBL carriage associated with OTC antibiotic use	Potential source of maternal colonization	Environmental/ Community risk
Chabanga (2025)	Zimbabwe	Food supply chain	Milk, poultry; *E. coli*	Antibiotic residues and resistant *E. coli*	Foodborne exposure and selection pressure potentially affecting maternal microbiota	Environmental/ Food safety
Abraham *et al.* (2025)	Malawi	Agriculture/ environment	Soil, wastewater, produce; MDR Gram-negative bacilli	MDR organisms detected in environmental and agricultural sources	Exposure through contaminated fresh produce consumed during pregnancy	Environmental exposure
Chidziwisano *et al.* (2024)	Malawi	Community WASH	Wastewater, soil; MDR coliforms	MDR coliform contamination associated with poor sanitation	Household environmental contact may increase maternal colonization risk	Environmental risk factor
Kalizang'oma *et al.* (2025)	Malawi	Mass drug administration (MDA)	Nasopharyngeal flora; *S. pneumoniae* (Macrolide-R)	MDA-associated changes in antimicrobial resistance and carriage	Population-level intervention may alter household and maternal carriage dynamics	Population-level intervention

Continued

Mithi *et al.* (2024)	Malawi	Hospital inpatient wards	Clinical isolates; ceftriaxone-R organisms	Watch-category ceftriaxone resistance indicates hospital selection pressure	Healthcare exposure may affect admitted pregnant women and perinatal populations	Healthcare environmental risk
Lubanga *et al.* (2025)	Malawi	Public facilities	Prescriber behaviours	Stewardship non-compliance	Unregulated outpatient antibiotic exposure may contribute to resistance among women of reproductive age	Health-systems driver
Kumwenda & Semu (2025)	Malawi	Outpatient clinics	Female urinary isolates; fluoroquinolone-R *E. coli*	High community baseline resistance among urinary isolates	Resistance may be present in women entering pregnancy	Community clinical risk
Kachale (2021)	Malawi	Referral hospitals	Maternal near-miss cases	Uncontrolled sepsis and delayed care associated with severe maternal morbidity	Resistant or inadequately treated sepsis may contribute to adverse maternal outcomes	Clinical outcome linkage
Kasanga *et al.* (2023)	Zambia	One Health interface	Clinical isolates & wastewater; *E. coli*/*bla*CTX-M	Shared *bla*CTX-M genotypes detected across hospital waste and community environment	Suggests environmental circulation of resistance determinants relevant to maternal exposure	Genomic/ Environmental linkage
Keenan *et al.* (2024)	East Africa	Community WASH	Urinary tract isolates; MDR *E. coli*	Poor water infrastructure associated with maternal MDR-UTI rates	WASH conditions may contribute directly to maternal MDR colonization/infection	Socio-environmental driver
Ntanda (2024)	Zambia	Hospital environment	Inpatient surfaces; carbapenem-R *P. aeruginosa*	Carbapenem-resistant organisms detected on hospital surfaces	Delivery wards and NICUs may provide environmental reservoirs for maternal/neonatal exposure	Facility environmental reservoir
Shutt *et al.* (2025)	Global LMICs	Structural/WASH	Community environment; broad ARGs	Socio-economic vulnerability associated with environmental AMR burden	Structural and WASH conditions may shape baseline maternal microbiome resistance	Systematic environmental driver

3.2.3. Secondary Reviews

Eight systematic reviews, scoping reviews, and regional policy reports were included to provide contextual synthesis, pooled prevalence estimates, and global economic benchmarks across SSA and LMICs (Table 3).

3.3. Methodological Quality and Risk of Bias

Every study that could be appraised was assessed independently by two reviewers with the design-specific tools described in Section 2.7. The distribution of risk-of-bias ratings, stratified by study design, is shown in Table 4. The four protocols

Table 3. Characteristics of included reviews (n = 8).

Author, Year	Review Type	Population	Geographic Scope	Main Evidence Contribution	Overlap with Included Studies
Chapuma *et al.*, 2024	Systematic review	Pregnant/postpartum women	SSA	Regional prevalence of 3GC resistance in maternal infections	High
WHO African Region, 2021	Regional review	General/perinatal populations	Africa	Geographic distribution of ESBL-E and MRSA	Moderate
Bunduki *et al.*, 2024	Systematic review/meta-analysis	Hospitalised patients	Africa	Pooled prevalence of healthcare-associated ESBL/carbapenemase infections	Moderate
Osei Sekyere *et al.*, 2021	Systematic review	Pregnant women/infants	LMICs	Global dissemination of NDM/VIM carbapenemases	Moderate
Bah *et al.*, 2025	Scoping review	Maternal/TB cohorts	Global	Policy gaps in drug-resistant TB management during pregnancy	Low
Monari *et al.*, 2024	Systematic review	Postpartum women	LMICs	Pooled incidence of MDR pathogens in post-CS SSI/endometritis	High
Greco *et al.*, 2022	Health policy review	Perinatal healthcare	Global LMICs	Economic and mortality burden of AMR	Low
Kowalski *et al.*, 2024	Systematic review/meta-analysis	Neonates/children	SSA	Failure of empirical ampicillin-gentamicin for neonatal sepsis	High

Table 4. Risk-of-bias ratings of included primary studies, stratified by study design.

Study design group	Appraisal tool	Studies (n)	Low risk	Moderate risk	High risk	Not appraisable
Cross-sectional and retrospective studies (Table 1)	JBI checklist (cross-sectional/cohort)	14	11	3	0	0
Cohort and case-control studies (Table 1)	JBI checklist (cohort/case-control)	4	3	1	0	0
Molecular and genomic studies (Table 1)	Modified JBI (typing resolution, source attribution)	2	2	0	0	0
Mixed-methods studies (Table 1)	MMAT 2018	1	1	0	0	0
Doctoral theses with an observational design (Table 1)	Design-specific JBI checklist	6	4	2	0	0
Protocols (Table 1)	Not applicable	4	-	-	-	4
Contextual One Health studies (Table 2)	Design-specific JBI/modified JBI	13	9	4	0	0

in Table 1 contain no outcome data and could not be appraised; they are reported as descriptive context only. The eight secondary reviews (Table 3) were used for contextualisation and were not rated with the primary-study tools.

Quality ratings were applied in the narrative synthesis according to the weighting principle set out in Section 2.7. Statements about direct maternal-to-neonatal transmission (Tiers 1-2, Section 3.4) rest only on studies rated at low or

moderate risk of bias, and low-risk genomic studies carried the greatest weight. Findings from moderate-risk studies were used to judge whether associations were consistent across settings but were not used alone to support a directional claim. Findings from high-risk studies are described in Sections 3.5-3.11 but are labelled as hypothesis-generating and were not used as the sole support for any conclusion. Where studies of different quality gave discordant results, the findings of the higher-quality studies were preferred. Quality ratings therefore limit how strongly the determinants in Sections 3.5-3.11 can be interpreted: associative evidence of any quality is not treated as evidence of transmission.

3.4. Evidence for Maternal-Neonatal Transmission

To characterize transmission mechanisms, the primary maternal-neonatal evidence was graded with the four-tier hierarchy defined in Section 2.5 (Figure 2). Tiers 1 and 2 constitute direct transmission evidence, because they rest on paired maternal-neonatal isolates; Tiers 3 and 4 constitute associative evidence, because they show co-occurrence of maternal exposure and neonatal outcomes without isolate-level linkage. Of the 31 primary maternal-neonatal studies in Table 1, one was graded Tier 1 (confirmed), two Tier 2 (probable), nine Tier 3 (possible) and 19 Tier 4 (unestablished). Direct transmission evidence was therefore available for 3 of 31 studies (9.7%), and 28 of 31 provided associative evidence only (Table 5). Study-level specimen and method details for ten further linkage studies are given in Table 6.

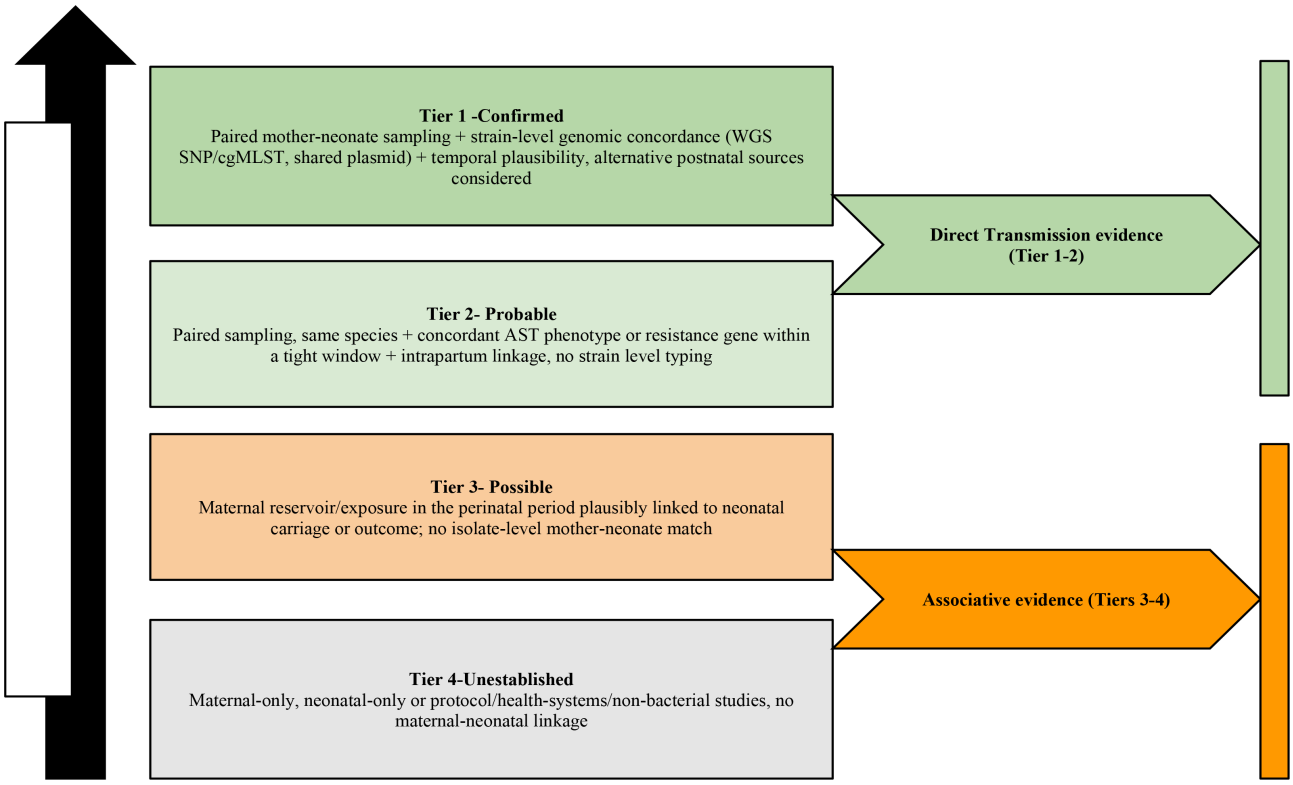


Figure 2. Four-tier hierarchy of maternal-neonatal AMR transmission evidence, showing the division between direct transmission evidence (Tiers 1-2) and associative evidence (Tiers 3-4).

Table 5. Evidence tiers, eligibility criteria and mapping of the 31 primary maternal-neonatal studies in Table 1.

Evidence tier	Evidence type	Eligibility criteria (summary)	n	Studies (Table 1)
Tier 1 - Confirmed	Direct	Paired sampling; strain-level genomic concordance; temporal plausibility; alternative sources considered	1	Carvalho (2022)
Tier 2 - Probable	Direct	Paired sampling; species and phenotype/gene concordance within a tight window; no strain-level typing	2	Blumenröder (2023); Berckenhagen (2025)
Tier 3 - Possible	Associative	Perinatal maternal reservoir or exposure plausibly linked to neonatal outcome; no isolate-level matching	9	Tumuhamye (2021); Bulabula (2020); Sammarro (2023); Nielsen (2024); Taika (2022); Abu Shqara (2025); Seale (2025); Tshivhase (2024); Ainomugisha (2024)
Tier 4 - Unestablished	Associative	Maternal-only, neonatal-only, protocol, health-systems or non-bacterial; no linkage	19	Mehari (2025); Martin (2023); Salissou (2023); Martin (2025); Olaru (2023); Olaru (2020); Yeta (2021); Olaru (2022); Dunlop (2022); Mwandigha (2020); Quispe (2020); Amon (2021); Wadonda-Kabondo (2025); Sebastiao (2021); Solomon (2021); Bah (2023); Epuitai (2025); Beales (2025); Kim (2025)

Table 6. Specimen-level characteristics and evidence tier of maternal-neonatal linkage studies (n = 10).

Study	Maternal Specimen	Neonatal Specimen	Organism(s)	Diagnostic Method	Evidence Tier
Ranjeva *et al.*, 2022	Rectovaginal swab	Neonatal gut/swab	*Enterobacteriaceae* (ESBL)	Genomic sequencing + AST	Tier 1 - Confirmed
Omulo *et al.*, 2015	Maternal gut/stool	Neonatal stool	Enteric Gram-negatives	Culture + AST	Tier 3 - Possible (community reservoir)
Obakiro *et al.*, 2021	Urine	Neonatal blood/swab	*E. coli*, *K. pneumoniae*	Culture + AST	Tier 4 - Unestablished (maternal UTI focus)
Velin *et al.*, 2021	Surgical site swab	Neonatal swab	*S. aureus*, Gram-negatives	Culture + AST	Tier 3 - Possible
Mbabazi *et al.*, 2025	Post-caesarean wound swab	Neonatal swab	MDR Gram-negatives	Culture + AST	Tier 3 - Possible
Egbe *et al.*, 2023	Rectovaginal swab	Neonatal blood	Gram-negative bacilli	Culture + AST	Tier 2 - Probable
Sands *et al.*, 2021	Vaginal swab/stool	Neonatal blood/stool	*K. pneumoniae*, *E. coli*	Genomic WGS + AST	Tier 1 - Confirmed
Geleta *et al.*, 2024	Vaginal swab	Neonatal blood	Gram-negative bacilli	Culture + AST	Tier 2 - Probable
Donkor *et al.*, 2023	High vaginal swab	Neonatal swab	Group B *Streptococcus*	Culture + AST	Tier 3 - Possible
Mbelle *et al.*, 2020	Maternal rectovaginal swab	NICU neonatal swab/blood	ESBL/Carbapenemase *Enterobacteriaceae*	Molecular PCR + AST	Tier 2 - Probable (environmental source not excluded)

3.4.1. Confirmed Transmission

Tier 1 (confirmed) transmission was assigned to one study in Table 1, which combined paired maternal-neonatal sampling with high-resolution metagenomic se-

quencing and showed genomic relatedness of resistance determinants (blaCTX-M and blaTEM) carried by maternal and neonatal isolates [55]. Two linkage studies in Table 6 also reached Tier 1 using whole-genome sequencing. Because Tier 1 requires alternative post-natal sources to have been considered, the number of confirmed events is small and these findings should not be generalised beyond the settings studied.

3.4.2. Probable Transmission

Tier 2 (probable) transmission was assigned to two studies in Table 1 that showed matching species and concordant susceptibility profiles in mother-neonate pairs within a tight temporal window, in the presence of intrapartum risk factors such as prolonged rupture of membranes [56]. Berckenhagen included genomic data, but because hospital-environmental acquisition was not excluded, the study did not meet the Tier 1 criterion. The absence of strain-level typing in the remaining study precluded definitive exclusion of immediate post-delivery environmental exposure [57].

3.4.3. Possible Transmission

Tier 3 (possible) was assigned to nine studies in Table 1 that documented a perinatal maternal reservoir or maternal infection plausibly linked to neonatal carriage, infection or outcome, but without isolate-level matching [58] [59]. These studies are associative: they show that maternal exposure and neonatal outcomes co-occur and are compatible with transmission, but they do not show that organisms or genes passed from mother to neonate, and healthcare-worker or environmental sources were not excluded [60]. Further Tier 3 linkage studies are listed in Table 6.

3.4.4. Evidence Insufficient to Establish Transmission

Tier 4 (unestablished) was assigned to 19 studies in Table 1 that sampled only mothers (for example antenatal bacteriuria), only neonates (for example late-onset NICU cohorts) or neither (protocols, prescribing and viral-resistance studies), and therefore provided no maternal-neonatal linkage [61]-[63]. These studies describe carriage rates or clinical burden but do not address transmission pathways.

3.5. Maternal Determinants

Maternal colonization with resistant pathogens most notably rectovaginal carriage of ESBL-producing *E. coli* and *K. pneumoniae* was the principal maternal reservoir identified as a potential source of vertical exposure [64]-[66]. Reported antenatal rectovaginal carriage rates across SSA ranged from 15% to 45%.

The following intrapartum factors were associated with neonatal exposure risk; these are associative findings and do not by themselves establish transmission:

- Prolonged Rupture of Membranes (PROM): Exceeding 18 - 24 hours was associated with ascending intra-amniotic colonization [67].
- Delivery Mode: Caesarean section delivery was associated with altered initial neonatal microbiota, with greater exposure to hospital-acquired MDR isolates,

and with increased maternal risk of surgical site infections [68].

- Unregulated Antimicrobial Use: Unindicated prophylactic administration of broad-spectrum beta-lactams during labor was associated with selective pressure within the maternal gastrointestinal and genital tract microbiota [69].

3.6. Neonatal Determinants

Neonatal susceptibility to resistant colonization and invasive infection resulted from an interplay of host biological vulnerabilities and acute healthcare exposures:

- Host Vulnerability: Prematurity (<37 weeks gestation) and low birth weight (<2500 g) were consistently reported risk factors, characterized by fragile cutaneous barriers, incomplete mucosal development, and reduced transplacental IgG transfer.
- Invasive Interventions: Invasive procedural exposures including endotracheal intubation, mechanical ventilation, central venous catheterization, and umbilical vein cannulation provided direct portals of entry for opportunistic resistant pathogens [70].
- Microbiome Dysbiosis: Early exposure to empiric broad-spectrum antibiotics suppressed commensal *Bifidobacterium* and *Lactobacillus* species, creating ecological niches for MDR Gram-negative colonization [71].

3.7. Healthcare-Associated Determinants

Delivery suites and NICUs functioned as high-density amplifiers for horizontal cross-transmission:

- IPC Compliance Gaps: Suboptimal hand hygiene compliance among healthcare personnel, overcrowding, and high patient-to-nurse ratios were primary drivers of horizontal transmission between neonates.
- Inanimate Reservoirs: Biofilms on high-touch surfaces, radiant warmers, incubators, resuscitation equipment, and shared multi-dose medication vials harbored persistent carbapenem-resistant *P. aeruginosa* and *A. baumannii* [72].

3.8. Environmental Determinants

Environmental infrastructure within healthcare facilities and surrounding communities was associated with exposure risk:

- WASH Infrastructure: Compromised water supply and inadequate handwashing stations in labor wards were associated with elevated Gram-negative contamination of delivery surfaces [73].
- Effluent & Waste Management: Inadequate disposal of liquid hospital waste released sub-inhibitory concentrations of active antibiotic residues and resistance genes (*bla*CTX-M, *bla*NDM) into local aquatic systems [74].

3.9. Contextual One Health Evidence

The broader ecological framework revealed community-level factors associated with baseline maternal resistance (contextual, associative evidence):

- Agricultural & Veterinary Misuse: Sub-therapeutic antibiotic use in livestock and poultry selected for MDR *E. coli* and *Salmonella* strains, entering the human diet via unpasteurized dairy and contaminated produce [75].
- Community Prescribing Pressure: Unregulated over-the-counter access to Watch-category antibiotics was associated with a high baseline prevalence of ESBL carriage among reproductive-age women prior to facility admission [76].

3.10. AMR Organisms and Resistance Determinants

Gram-negative bacilli accounted for 75% - 80% of all reported resistant isolates across maternal and neonatal cohorts:

1) Extended-Spectrum β-Lactamases (ESBLs): Predominantly driven by *bla*CTX-M-15, *bla*TEM, and *bla*SHV genes in *E. coli* and *K. pneumoniae*, conferring high-level resistance to third-generation cephalosporins (ceftriaxone, cefotaxime) [77].

2) Carbapenemases: *bla*NDM-1, *bla*OXA-48-like, and *bla*VIM determinants were frequently identified in carbapenem-resistant *K. pneumoniae*, *A. baumannii*, and *P. aeruginosa* isolates in NICU settings [78].

3) Gram-Positive Determinants: Methicillin-resistant *S. aureus* (MRSA) mediated by the *mecA* gene, alongside *Streptococcus agalactiae* demonstrating rising resistance to macrolides and clindamycin [79].

3.11. Neonatal Colonization and Infection

Concordance between maternal mucosal colonization and subsequent neonatal colonization was high (30% - 60%). However, progression from mucosal colonization to invasive sepsis was mediated by host vulnerability and invasive procedural exposure:

- Early-Onset Sepsis (<72 hours): Most often attributed to vertical acquisition of maternal rectovaginal ESBL-*E. coli* or GBS following intrapartum exposure or PROM [80].
- Late-Onset Sepsis (>72 hours)**:** Predominantly caused by horizontal nosocomial acquisition of carbapenem-resistant *K. pneumoniae* and *A. baumannii* within NICU environments [81].
- Treatment Failure & Mortality: Standard first-line empiric regimens (ampicillin + gentamicin) demonstrated high treatment failure rates (>40%) in settings with high ESBL prevalence, resulting in attributable mortality rates of 18% - 42% for neonatal septicemia [82].

3.12. Prevention and Control

Synthesizing evidence across primary trials and policy frameworks highlighted five core intervention pillars:

1) Antimicrobial Stewardship (AMS): Implementing institutional prescribing guidelines, enforcing WHO AWaRe classification compliance, and eliminating unindicated intrapartum prophylaxis [83].

2) Infection Prevention & Control (IPC): Enforcing strict multimodal hand hy-

giene, routine environmental sporicidal disinfection, and isolation protocols for infants colonized with carbapenemase-producing isolates .

3) Maternal Screening & Topical Antisepsis: Routine antenatal rectovaginal screening alongside intrapartum topical chlorhexidine vaginal cleansing to reduce bacterial burden during delivery [84].

4) Diagnostic & Surveillance Capacity: Upgrading local microbiological culture and AST infrastructure while integrating genomic surveillance networks [85].

5) WASH & One Health Governance: Improving facility water quality and regulating agricultural antibiotic distribution.

3.13. Integrated Conceptual Framework

The findings of this qualitative synthesis were organised into a multi-layered conceptual framework of the determinants examined at the maternal-neonatal interface (**Figure 3**). The framework is built largely on associative evidence and does not imply confirmed causal Pathway.

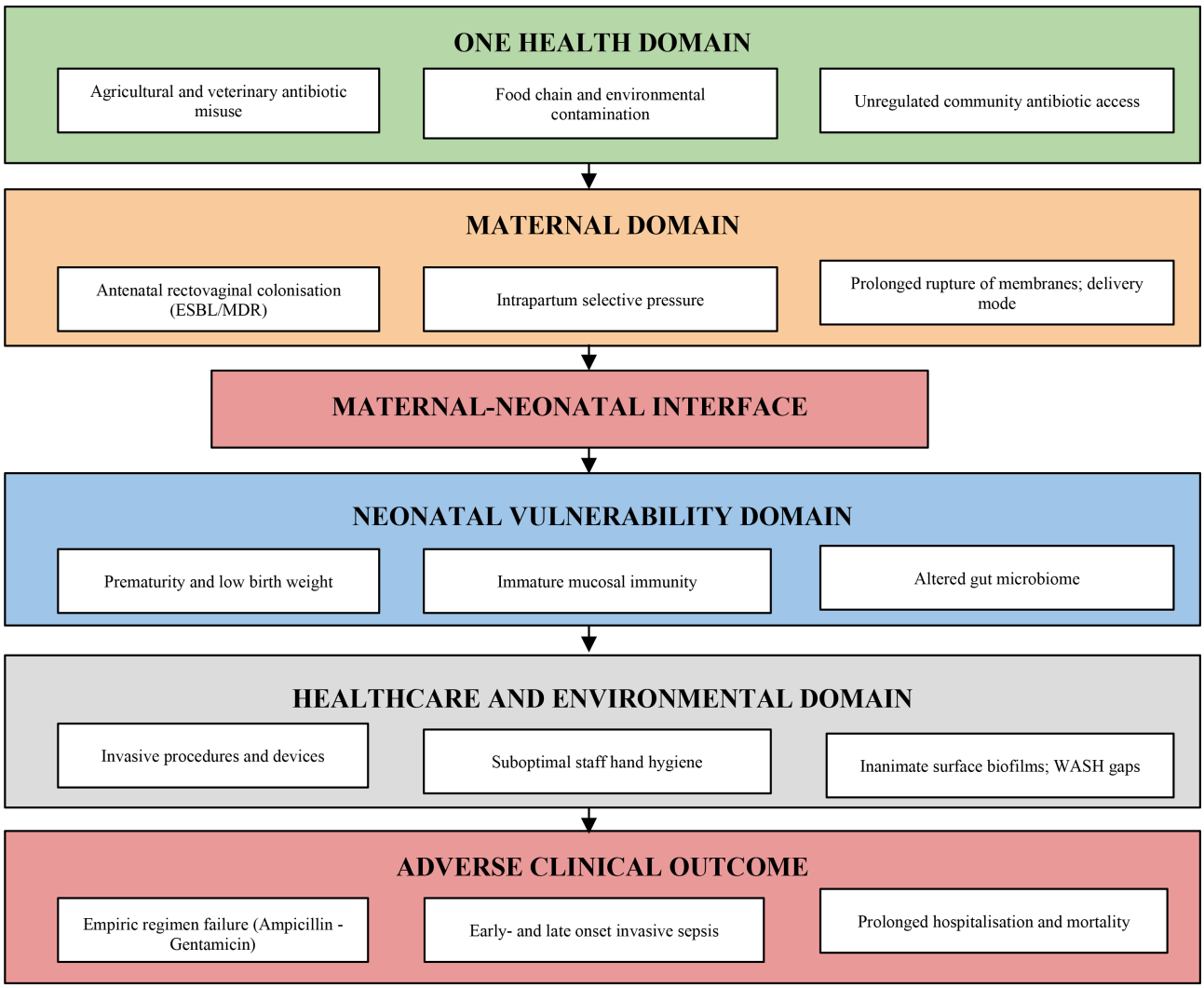


Figure 3. Framework narrative.

As depicted in Figure 3, maternal and neonatal AMR acquisition is described in the included studies as a multifactorial process involving interacting determinants across four key domains:

- One Health Domain: Community antibiotic exposure, inadequate WASH infrastructure, and agricultural overuse are associated with a baseline prevalence of resistance genes (*bla*CTX-M, *bla*NDM) within women of reproductive age.
- Maternal Domain: Antenatal mucosal carriage coupled with intrapartum selective pressure is associated with vertical exposure during labor and delivery.
- Healthcare & Environmental Domain: Nosocomial reservoirs, contaminated medical devices, and IPC compliance gaps are associated with horizontal cross-transmission of clonal strains within labor suites and NICUs.
- Neonatal Vulnerability Domain: Biological immaturity (prematurity, low birth weight) combined with invasive clinical procedures is associated with progression from asymptomatic mucosal colonization to severe invasive sepsis, empiric treatment failure, and mortality.

4. Discussion

This review suggests that neonatal AMR acquisition is influenced by interacting maternal, neonatal, healthcare-associated, environmental and One Health factors, and that maternal-neonatal transmission should not be conceptualised as a single event occurring exclusively during childbirth. Neonatal acquisition may occur before, during or after delivery through overlapping maternal, healthcare, community and environmental reservoirs. The interpretation below distinguishes direct transmission evidence (Tiers 1-2) from associative evidence (Tiers 3-4), as defined in Section 2.5.

Importantly, the strength of evidence for direct maternal-neonatal transmission varied considerably across studies. Evidence for direct transmission was strongest where mothers and neonates were sampled concurrently or longitudinally and where microbiological, molecular, or genomic methods were used to compare resistant organisms or resistance determinants. These approaches provide greater source-attribution capacity than studies relying solely on phenotypic similarity or identification of the same bacterial species. Nevertheless, even detection of an identical resistant organism in a mother and neonate does not necessarily establish the direction of transmission when alternative reservoirs remain plausible. Molecular concordance should therefore be interpreted in conjunction with sampling time, specimen type, epidemiological relationships, healthcare exposures, and potential environmental or community sources. As shown in Section 3.4, direct transmission evidence was available for only a small minority of the primary maternal-neonatal studies, and most of the evidence base is associative.

A substantial proportion of the reviewed evidence instead demonstrated associations between maternal colonization or antimicrobial exposure and neonatal carriage or infection. Although these findings are biologically and epidemiologically important, they should not be considered equivalent to confirmed vertical

transmission. Neonatal acquisition may occur before, during, or after delivery and may involve maternal, healthcare-associated, environmental, or community sources. This distinction is particularly important when interpreting studies that identify maternal colonization as a risk factor without demonstrating strain-level or genomic linkage between maternal and neonatal isolates. Future research should therefore prioritize longitudinal maternal-neonatal sampling combined with high-resolution genomic characterization to distinguish true maternal transmission from acquisition resulting from shared or independent exposures.

4.1. Maternal Determinants of Neonatal AMR Acquisition

Maternal factors represented an important component of the evidence base. Antenatal antimicrobial exposure, prolonged rupture of membranes (PROM), maternal colonization with resistant organisms, and intrapartum antibiotic use were among the frequently reported determinants associated with neonatal acquisition [28] [86]. Maternal colonization is particularly relevant because resistant organisms may be transferred to neonates through exposure during delivery, direct maternal contact, or other early-life interactions [87]. Such exposure occurs during a critical period in which the neonatal microbiome is being established and the immune system remains immature [88].

The relationship between maternal antimicrobial exposure and neonatal microbial acquisition is, however, complex. Evidence from Li *et al.* illustrates the potential influence of maternal and early-life antimicrobial exposure on microbial transmission. This suggests that antimicrobial exposure may substantially modify normal maternal-neonatal microbial transfer and potentially alter the development of protective microbial communities [89]. Nevertheless, these findings should not be interpreted as demonstrating a direct causal pathway between antimicrobial exposure and AMR acquisition. Antibiotic exposure may simultaneously influence microbial diversity, selection pressure, colonization dynamics, and susceptibility to subsequent acquisition, making the underlying relationship biologically complex [90].

Maternal determinants therefore need to be interpreted within the broader transmission network rather than considered in isolation. Although maternal colonization and antimicrobial exposure may increase the probability of neonatal exposure to resistant organisms, they do not necessarily account for all neonatal acquisition [91]. The evidence indicates that neonates may subsequently acquire resistant organisms through healthcare workers, contaminated equipment, other patients, healthcare environments, and community or environmental reservoirs [81] [92]. Consequently, interventions focused exclusively on maternal screening or antibiotic prescribing are unlikely to eliminate neonatal AMR transmission [93]. Antenatal infection prevention, appropriate antimicrobial stewardship, identification and management of maternal colonization, neonatal infection prevention, and environmental controls should instead be implemented as complementary components of prevention strategies [84] [94].

4.2. Neonatal Vulnerability and Clinical Determinants

Neonatal characteristics also influenced susceptibility to AMR acquisition. Prematurity, low birth weight, prolonged hospitalization, invasive procedures, and admission to NICUs were identified as important determinants [11] [95]. Preterm and low-birth-weight neonates may be particularly vulnerable because of immature immune defenses and their greater dependence on prolonged hospital care and invasive interventions, including mechanical ventilation and intravenous catheterization [32] [96].

The relationship between neonatal vulnerability and AMR acquisition is closely connected to the intensity of healthcare exposure. Neonates requiring prolonged hospitalization have greater opportunities for exposure to healthcare workers, equipment, antimicrobial therapy, and contaminated surfaces [97]. Thus, prematurity and low birth weight may function not only as biological determinants of susceptibility but also as markers of increased healthcare exposure [98].

NICUs represent particularly important settings within this transmission pathway because they concentrate highly vulnerable patients who frequently require invasive procedures and antimicrobial treatment [86] [99]. These conditions can facilitate the persistence and circulation of MDR organisms. Repeated or prolonged exposure to broad-spectrum antibiotics may additionally disrupt the developing neonatal microbiota and create selective pressure favouring resistant organisms [100].

The potential consequences of early-life antimicrobial exposure may extend beyond immediate infection outcomes [101]. Early-life antimicrobial exposure has also been linked to effects on infant immune development and to several childhood health conditions [88] [102]. Although these observations do not establish a direct causal relationship between early antibiotic exposure and AMR transmission, they reinforce the importance of antimicrobial stewardship in neonatal populations [103]. Effective AMR control requires a combination of appropriate antimicrobial use, IPC, and continued investigation of alternative therapeutic approaches [90] [104].

4.3. Healthcare-Associated Transmission and NICU Environments

Healthcare facilities emerged as important reservoirs and transmission environments for resistant organisms. The risk associated with neonatal vulnerability was closely interconnected with healthcare practices, environmental contamination, antimicrobial exposure, and the intensity of medical care [105]. NICUs may be particularly susceptible to transmission because of high patient acuity, frequent invasive procedures, prolonged hospitalization, and repeated antimicrobial exposure [106].

Inadequate IPC practices may amplify transmission within these environments. Contaminated surfaces, inadequate hand hygiene, insufficient environmental cleaning, overcrowding, and inappropriate sterilization or equipment management can facilitate the movement of resistant organisms between patients, healthcare work-

ers, equipment, and the surrounding environment [82] [92] [107]. These risks may be particularly pronounced in resource-constrained facilities, where shortages of water, sanitation infrastructure, laboratory capacity, equipment, and trained personnel can limit the consistent implementation of IPC measures.

The evidence therefore suggests that healthcare-associated AMR transmission should not be addressed exclusively through prescribing interventions. Environmental cleaning, hand hygiene, equipment decontamination, appropriate isolation practices, adequate staffing, microbiological surveillance, and antimicrobial stewardship need to operate as complementary components of a comprehensive healthcare-associated AMR prevention strategy [81] [93] [108]. This integrated approach is particularly important in NICUs, where multiple risk factors converge within a confined clinical environment.

4.4. Environmental Determinants, WASH, and AMR Transmission

Environmental determinants were among the most prominent factors identified in the review. Poor water, sanitation and hygiene (WASH) infrastructure, contaminated hospital surfaces, inadequate environmental cleaning, overcrowding, and unsafe waste management were repeatedly associated with conditions conducive to the persistence and dissemination of resistant organisms [18] [94] [109]. These findings broaden the conventional clinical understanding of maternal-neonatal AMR by demonstrating that transmission is influenced by the physical and infrastructural environment in which mothers and neonates receive care. This finding is particularly important in low-resource healthcare settings, where infrastructure limitations may compromise routine IPC practices and increase opportunities for environmental transmission. Evidence from Pakistan further demonstrates the importance of strengthening WASH systems, IPC standards, and antimicrobial stewardship to interrupt potential transmission pathways between community water sources and healthcare facilities [110]. Similar challenges may occur in fragile and conflict-affected settings, including Soudan, where shortages of clean water, sanitation facilities, laboratory services, and infection-control resources may increase dependence on inadequate infrastructure [111]. Under such circumstances, environmental reservoirs may become important sources of neonatal colonization and infection [112].

The environmental pathway is not restricted to healthcare facilities. Arnold *et al.* [113] reported multidrug-resistant bacteria in contaminated household drinking water in Uganda. The association between water contamination, unsafe storage systems, and sociodemographic factors further illustrates how environmental conditions may contribute to the persistence and dissemination of resistant organisms [114]. The prominence of environmental determinants in this review highlights a potential gap in conventional AMR prevention strategies. Although AMR programmes have historically placed substantial emphasis on antimicrobial prescribing and stewardship within healthcare facilities, environmental reservoirs have received comparatively less attention [93] [115]. The findings therefore sup-

port greater integration of environmental surveillance, WASH improvement, sanitation, waste management, and environmental cleaning into AMR prevention programmes [116].

4.5. One Health Dimensions of Maternal-Neonatal AMR Transmission

A major contribution of this review is the demonstration that maternal and neonatal AMR transmission occurs within a broader ecological system involving interconnected human, animal, food, agricultural, and environmental reservoirs. These interconnected pathways provide a strong rationale for applying a One Health framework to AMR prevention and surveillance [6] [16] [117]. Evidence concerning food and agricultural systems illustrates this interconnectedness. Ghimpețeanu *et al.* reported that antibiotic residues are detected in food products in many regions, while differences in regulatory systems and enforcement may influence the extent of exposure. Although regulatory limits for antibiotic residues are established in some settings, gaps in regulation and enforcement may persist elsewhere, potentially contributing to chronic low-level exposure and selection pressure for resistant organisms [118]. Agricultural antimicrobial use is also relevant. In Rwanda, Hirwa *et al.* identified important gaps in farmers' knowledge, attitudes, and practices concerning antimicrobial use and AMR. Appropriate antimicrobial practices were associated with higher educational attainment, suggesting that behavioural interventions and education may contribute to more responsible antimicrobial use in livestock production. These findings reinforce the importance of moving beyond sector-specific approaches and establishing surveillance and interventions that connect human health, veterinary medicine, agriculture, food systems, and environmental health [119].

Similarly, evidence from Kenya identified shared resistance patterns across human, animal, and environmental samples, supporting the value of integrated surveillance for understanding AMR transmission dynamics [120]. Taken together, these findings support a One Health approach in which maternal and neonatal AMR prevention is embedded within broader systems addressing antimicrobial use and resistance across interconnected human, animal, and environmental reservoirs. Importantly, the One Health evidence should be interpreted as contextual evidence regarding potential reservoirs and transmission pathways rather than as proof that each animal, food, agricultural, or environmental reservoir directly caused maternal or neonatal acquisition in the included studies. Establishing such source attribution requires longitudinal sampling and molecular or genomic linkage between reservoirs and maternal or neonatal isolates.

4.6. Implications for Maternal and Neonatal AMR Prevention and Policy

The findings have important implications for maternal and neonatal health policy,

particularly in low-resource settings. The prominent role of environmental and healthcare-associated determinants indicates that reducing neonatal AMR transmission requires investment in IPC, WASH infrastructure, environmental surveillance, sanitation, and healthcare waste management [121]. Healthcare facilities should prioritize reliable access to safe water, functional hand-hygiene facilities, effective environmental cleaning, adequate sterilization capacity, and continuous training of healthcare workers in IPC practices [122].

Rational antimicrobial use is equally important. Policies should seek to reduce unnecessary empirical antibiotic prescribing, improve access to microbiological diagnostics, strengthen prescribing oversight, and regulate informal pharmaceutical markets. Antimicrobial stewardship programmes should incorporate routine AMR surveillance, prescribing audits, microbiological testing where feasible, and continuing education to support appropriate antimicrobial selection, dosage, and duration [123]. However, AMR prevention cannot remain confined to healthcare facilities. Because animal and environmental reservoirs may contribute to the broader transmission network, interventions should involve public health, agriculture, veterinary services, food safety, environmental protection, and water and sanitation sectors. Priority actions should include responsible antimicrobial use in livestock production, monitoring of food and water safety, reduction of environmental antibiotic contamination, and development of multisectoral AMR surveillance systems [124].

For low-resource settings, these interventions should be adapted to local health-system capacity rather than relying exclusively on resource-intensive technologies. Strengthening basic IPC, WASH, diagnostic capacity, antimicrobial stewardship, and surveillance infrastructure may provide a practical foundation for reducing maternal and neonatal AMR transmission [18] [110] [125]. The findings therefore support a layered prevention strategy in which maternal and neonatal interventions are embedded within healthcare-system strengthening and broader One Health surveillance.

4.7. Strengths and Limitations of the Review

This review has several strengths. First, it synthesized evidence from 62 studies across diverse geographical and epidemiological settings, providing a broad assessment of determinants associated with maternal and neonatal AMR transmission. Second, the use of a One Health perspective enabled the review to move beyond conventional clinical determinants and incorporate environmental, agricultural, and zoonotic dimensions of AMR. Third, the synthesis considered maternal, neonatal, healthcare-associated, and environmental determinants together, allowing interconnected transmission pathways to be examined within a common conceptual framework.

Several limitations should nevertheless be considered. Substantial heterogeneity existed across the included studies with respect to populations, study designs, healthcare settings, exposure definitions, microbiological methods, and AMR out-

comes. Such heterogeneity limits direct comparison between studies and makes it difficult to determine the relative magnitude of individual determinants. Differences in methodological quality (Section 3.3) may also have contributed to variation in reported associations, and quality ratings were used to weight, but could not eliminate, this variation. A further limitation concerns the distinction between evidence of association and evidence of transmission. Many studies identified maternal colonization, antimicrobial exposure, environmental contamination, or healthcare exposure as risk factors for neonatal carriage or infection without establishing microbiological or genomic linkage between maternal and neonatal isolates. Consequently, these findings provide evidence of potential or probable transmission pathways rather than definitive proof of maternal-to-neonatal transmission. This limitation is particularly relevant when interpreting evidence from environmental, animal, food, and agricultural reservoirs.

The search strategy carries additional limitations. Google Scholar was used only as a supplementary source and not as a core database. Its results are relevance-ranked, cannot be filtered with controlled vocabulary and cannot be exported in full, and because screening followed a predefined sequential protocol with stopping rules (Section 2.2), not all retrieved records were examined. The search is therefore not exhaustively reproducible, and the ranking favours highly cited and openly accessible publications. This introduces a risk of publication bias: studies with positive or striking findings are more likely to be published, indexed and ranked highly, while null findings and unpublished work may be missed. Grey literature identified through Google Scholar, including theses and preprints, has not necessarily undergone peer review, which is a further source of variation in study quality. Restriction to English-language publications and the absence of a formal assessment of publication bias, such as funnel plots, may compound this risk. The findings should therefore be interpreted with caution.

Primary evidence from some regions, particularly the Democratic Republic of Congo (DRC), was also limited, restricting the extent to which the findings can be directly generalized to settings such as Lubumbashi. Potential publication bias is another consideration, as studies reporting statistically significant or positive findings may be more likely to enter the published literature. Accordingly, the findings should be interpreted primarily as evidence of recurring associations and interconnected transmission pathways rather than definitive evidence of causality. This distinction is important given the heterogeneity of the evidence base and the predominance of observational research.

4.8. Future Research Directions

Future research should prioritize the generation of high-quality primary evidence in low-resource settings where maternal and neonatal AMR transmission remains insufficiently characterized. Prospective and longitudinal cohort studies are particularly needed to establish temporal relationships between maternal colonization, antimicrobial exposure, environmental contamination, neonatal coloniza-

tion, and subsequent infection. Future studies should also prioritize simultaneous and repeated sampling of mothers, neonates, healthcare environments, healthcare workers, water sources, and other relevant reservoirs where feasible. The incorporation of molecular and genomic epidemiological approaches could provide greater resolution for distinguishing true maternal-neonatal transmission from acquisition arising from shared environmental or healthcare exposures. Such approaches would also help clarify whether identical resistant organisms represent direct transmission, common-source exposure, or independent acquisition.

Research should additionally evaluate the effectiveness of interventions targeting environmental contamination, WASH systems, IPC practices, antimicrobial stewardship, and broader One Health determinants. Intervention studies should assess not only microbiological outcomes but also feasibility, cost, sustainability, acceptability, and scalability within routine healthcare systems. There is also a need to strengthen integrated AMR surveillance across human, animal, food, and environmental sectors. Genomic and molecular epidemiological approaches could help distinguish shared transmission pathways from independent emergence of resistance and clarify the contribution of specific reservoirs to maternal and neonatal acquisition. In settings such as the DRC, locally generated evidence should be prioritized to identify context-specific drivers and feasible interventions. Finally, future studies should evaluate integrated surveillance and prevention models under real-world conditions, particularly in resource-constrained maternity units and NICUs. Such evidence will be essential for developing practical, context-specific strategies capable of reducing AMR transmission between mothers and neonates while strengthening broader health-system resilience.

5. Conclusions

This systematic review demonstrates that AMR transmission between mothers and neonates is a multifactorial process involving maternal, neonatal, healthcare-associated, environmental, and broader One Health determinants. Maternal colonization, antimicrobial exposure, neonatal vulnerability, healthcare exposure, WASH deficiencies, and community and environmental antimicrobial pressures were recurrently associated with neonatal AMR acquisition.

Importantly, the evidence for direct maternal-neonatal transmission varied substantially, with the strongest evidence derived from paired sampling combined with molecular or genomic approaches. Therefore, detection of the same resistant organism alone should not be interpreted as definitive proof of transmission. Effective prevention requires an integrated approach combining antimicrobial stewardship, infection prevention and control, maternal-neonatal surveillance, diagnostic and genomic capacity, WASH improvement, and One Health interventions. Future studies should use prospective longitudinal designs with paired maternal-neonatal and environmental sampling to better establish transmission pathways and guide context-specific interventions.

6. Final Statement

AMR transmission between mothers and neonates represents a critical challenge for maternal and child health, driven by interconnected maternal, neonatal, healthcare-associated, environmental, and One Health determinants. This systematic review demonstrates that preventing AMR transmission requires coordinated strategies that extend beyond clinical care to include strengthened antimicrobial stewardship, IPC, surveillance, improved water, sanitation and hygiene (WASH), and multisectoral collaboration. By synthesizing the available evidence, this review provides a comprehensive foundation for informing policy, guiding future research, and supporting the development of integrated interventions to reduce the burden of AMR among mothers and neonates, particularly in low- and middle-income countries.

Author Contributions

Kalala Elisee Kabuya: Conceptualization, Methodology, Investigation, Data curation, Formal Analysis, Writing - original draft; Charline Mukasa Sangany: Supervision, Project administration, Resources, Writing - review & editing.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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