# Bacterial Colonisation and Antimicrobial Resistance in Mother-Neonate Dyads

**Ruth Ebimoboere AYIBAEMI[1*], Langley Ayibawanami ORUTUGU[2], Jack Perebi ADAMA[3], Abdulrasheed Babatunde ABDU[2], and Adebayo LAMIKANRA[4]**

**[1]Department of Microbiology, Faculty of Science, Niger Delta University, Wilberforce Island, Amassoma, Bayelsa State, Nigeria**

**[2]Department of Medical Microbiology & Parasitology, Faculty of Basic Clinical Sciences, College of Health Sciences, Niger Delta University, Wilberforce Island, Amassoma, Bayelsa State, Nigeria**

**[3]Nigerian Army, 16 Brigade Medical Centre, 16 Brigade Nigerian Army, Yenagoa, Bayelsa State, Nigeria**

**[4]Department of Pharmaceutical Microbiology, Faculty of Pharmacy, Obafemi Awolowo University, Ile-Ife, Osun State, Nigeria**

*** Corresponding Author**

**DOI: https://doi.org/10.51244/IJRSI.2026.1306000320**

**Received: 20 June 2026; Accepted: 25 June 2026; Published: 09 July 2026**

## ABSTRACT

Maternal bacterial carriage is influential in neonatal colonisation and is a factor in the transfer of bacterial infections from mother to child. However, there is scant evidence on this from resource-constrained circumstances. This cross-sectional research determined the frequency, antibiotic susceptibility profiles and transmission of bacterial infections among 50 mother-infant dyads at Niger Delta University Teaching Hospital and Federal Medical Centre, Yenagoa, Nigeria. Maternal nasal and hand swabs and neonatal umbilical cord swabs were collected from October 2024 to March 2025. Bacteria were isolated, identified and their sensitivity to antimicrobial agents was determined using conventional microbiological procedures. A total of 269 bacterial isolates were recovered from 150 specimens. 23 pairs (46%) shared at least one bacterial species, with *Staphylococcus aureus* being most frequently matched (48.70%), followed by *Staphylococcus epidermidis* (22.68%), *Pseudomonas aeruginosa* (10.04%), and *Escherichia coli* (6.69%). All the organisms isolated in this study were resistant to amoxicillin, augmentin, cefuroxime, methicillin, and oxacillin. The incidence of resistance to cloxacillin (99.26%), ceftriaxone (99.63%), erythromycin and vancomycin (92.94%), were very high, but all the organisms isolated were sensitive to imipenem. Chi-square analysis revealed associations between neonatal colonisation, shared isolates and shared organisms, but not with delivery mode, gender, or medical conditions. These findings suggest that maternal bacterial carriage can be an important contributor to early neonatal colonisation, and the high prevalence of resistance to first-line antibiotics and the retention of carbapenem sensitivity shows a need for maternal screening, infection control, and the implementation of antimicrobial stewardship programmes in Nigerian healthcare settings.

**Keywords**: Mother-infant dyads, Vertical transmission, Antimicrobial resistance, *Staphylococcus aureus*, Neonatal colonisation.

## INTRODUCTION

The human microbiome has been reported to significantly influence protection against pathogen colonisation, and immune system maturation (Gensollen *et al.,* 2016; Sorbara and Pamer, 2019; Al-Nabhani and Eberl, 2020). Disruptions to the microbiota in neonates during early development can increase the likelihood of health issues in childhood or even in adulthood (Brodin, 2022).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

A newborn's first encounter with bacteria mostly comes from maternal sources and occurs during and after delivery (de Goffau *et al.,* 2019). Such vertical transfer of germs may be a major driver of the newborn microbiome. However, the introduction and fast spread of antimicrobial-resistant organisms have been identified as one of the most important global health concerns of the 21st century (Murray *et al.,* 2022).

The frequency of illnesses due to antibiotic-resistant organisms jeopardizes the efficacy of existing treatments and significantly contributes to worldwide morbidity and death (ARC, 2022). The World Health Organization (WHO) has identified antimicrobial resistance (AMR) as one of the ten foremost public health issues globally (Walsh *et al.,* 2023). The Centres for Disease Control and Prevention predicts that annually in the United States, approximately 2.8 million illnesses due to drug-resistant bacteria occur, resulting in more than 35,000 fatalities (Centres for Disease Control and Prevention, 2019, 2022). In Europe, antimicrobial resistance (AMR) is associated with over 25,000 deaths per year (World Health Organisation, 2023; Naghavi *et al.,* 2024).

The consequences of antimicrobial resistance (AMR) are particularly acute in low- and middle-income countries (LMICs), which account for approximately 85% of the global population and disproportionately endure the burden of microbial illnesses (Antimicrobial Resistance Collaborators, 2022).

In Sub-Saharan Africa, newborn mortality rates are among the highest globally, ranging from 26 to 32 deaths per 1,000 live births, with infections constituting a significant proportion of these fatalities (UNICEF, 2023; WHO, 2024; Enyew *et al.,* 2025). Managing these conditions has grown increasingly difficult with the spread of antimicrobial-resistant organisms (Wen *et al.,* 2021). Recent estimates indicate that over 40% of neonatal sepsis episodes have acquired resistance to WHO-recommended first-line treatments (WHO, 2022).

Maternal microbial colonisation has been associated with infant infections (Liu *et al.,* 2019), and pathogens shared between mother and child may arise through vertical transfer during delivery or via mutual environmental contacts (Yassour *et al.,* 2018). While targeted strategies, including antenatal screening and intrapartum antibiotic prophylaxis, have achieved reductions exceeding 80% in early-onset invasive cases in high-income regions (American College of Obstetricians and Gynaecologists, 2020; Verani *et al.,* 2010), knowledge gaps persist regarding maternal colonisation patterns and their links to neonatal infections in LMICs.

This study aims to investigate the prevalence, antibiotic susceptibility profiles, and transmission of bacterial pathogens isolated from mothers to their neonates in Bayelsa State, Nigeria.

## MATERIALS AND METHODS

### Study design and setting

This cross-sectional research was undertaken between October 2024 and March 2025 at two health care institutions in Bayelsa State, Nigeria: Niger Delta University Teaching Hospital (NDUTH), Okolobiri and Federal Medical Centre (FMC), Yenagoa. All laboratory studies were carried out at the Medical Microbiology and Parasitology Laboratory, College of Health Sciences, Niger Delta University.

### Study population and sampling

Fifty mother-child dyads were consecutively enrolled until the predetermined sample size was attained, yielding 150 specimens (50 umbilical cord swabs from neonates, 50 hand swabs, and 50 nasal swabs from mothers). The sample size of 50 dyads was informed by similar studies on maternal-neonatal bacterial transmission conducted in resource-limited settings, where dyad sizes of 30–60 have been consistently employed to generate preliminary epidemiological data (Olowe *et al.,* 2015; Matok *et al.,* 2021). This study was accordingly designed as a baseline investigation, with the findings intended to inform the power calculations for future studies in the region. Eligible mother-child pairs included neonates with intact umbilical cords who were admitted to Special Care Baby Units or present in postnatal wards, and whose mothers provided verbal consent. Exclusion criteria included maternal refusal or withdrawal of consent and neonates whose umbilical cords had already fallen off.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

**Sample collection and transportation**

Umbilical cord swabs were collected from neonates, while hand and nasal swabs were obtained from their mothers using sterile swabs and aseptic techniques following the method described by Mukuande (2023). All the samples obtained were transferred to sterile bottles containing STUART Transport Medium (Remel, USA) and transported on ice to the laboratory.

**Bacterial isolation and identification**

Samples were cultured aerobically on Blood agar, MacConkey agar, and Mannitol Salt agar, and anaerobically on MacConkey and Blood agar using anaerobic jars. Plates were incubated at 37°C for 24–48 hours. Plates showing no growth after 24 hours were further incubated before declaring the absence of bacteria.

Gram staining was performed for preliminary identification to separate the isolates into Gram-positive and Gram-negative categories and to determine cellular morphology. Spore staining was conducted using malachite green and safranin to detect endospore-forming bacteria such as *Bacillus species*.

Biochemical identification tests included catalase, coagulase, oxidase, triple sugar iron (TSI), indole, motility, citrate, and methyl red-Voges Proskauer (MR-VP). All the bacterial isolates obtained in the course of this study were stored cryopreserved at -85°C.

**Antimicrobial susceptibility testing**

The sensitivity of the isolates to different antibiotics was determined using the modified Kirby-Bauer disc diffusion technique on Mueller-Hinton agar (CLSI, 2023). Bacterial suspensions in sterile normal saline were produced and adjusted until 0.5 McFarland standard turbidity was achieved. The sterile swabs were dipped into the solution and the suspension was spread over Mueller-Hinton agar plates. Antibiotic discs were put on surface with sterile forceps after 5 min drying.

Thirteen antibiotics, amoxicillin (25µg), augmentin (30µg), ceftazidime (30µg), ceftriaxone (30µg), cefuroxime (30µg), cloxacillin (5µg), erythromycin (5µg), gentamicin (10µg), methicillin (10µg), ofloxacin (5µg), oxacillin (5µg), and vancomycin (30µg) were examined as mentioned above. Agar well diffusion technique was used to evaluate Imipenem (10µg). Wells were punched onto Mueller-Hinton agar plates and 10 µL of the stock solution was added to each well.

Plates were incubated for 18-24 hours at 37°C. Zones of inhibition were measured in millimeters using a clear ruler and evaluated according to Clinical and Laboratory Standards Institute (CLSI) recommendations 2023.

**Quality control**

All procedures followed standard operating procedures. Sterile swabs were verified by culturing unused swabs on MacConkey agar, with no growth observed after incubation. Media expiry dates were checked before use.

**Statistical analysis**

Data were analysed using Statistical Package for Social Sciences (SPSS) version 25. Summary measures were reported as percentages for categorical variables. Chi-square tests of independence were conducted to examine associations between neonatal colonisation and maternal/neonatal factors including maternal colonisation status, mode of delivery, neonate gender, and presence of shared bacterial organisms. P-values $\leq 0.05$ were considered statistically significant.

**Ethical considerations**

Ethical approval was obtained from the College Ethic Committee of the College of Health Sciences, Niger Delta University, Wilberforce Island, Bayelsa State (Ref No: 01- 10782024/043). And also, the Health Research Ethics Committee, Federal Medical Centre Yenagoa (FMCY/REC/ECC/2024/NOV/802).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

# RESULTS

## Distribution and prevalence of bacterial isolates

A total of 269 bacterial isolates were recovered from the 150 clinical specimens. *Staphylococcus aureus* was the most prevalent species, accounting for 131 isolates (48.70% of total isolates), followed by *Staphylococcus epidermidis* with 61 isolates (22.68%), *Pseudomonas aeruginosa* with 27 isolates (10.04%), *Escherichia* coli with 18 isolates (6.69%), *Bacillus subtilis* with 8 isolates (2.97%), *Klebsiella pneumoniae* with 8 isolates (2.97%), *Enterobacter cloacae* with 4 isolates (1.49%), *Enterococcus faecalis* with 4 isolates (1.49%), *Staphylococcus saprophyticus* with 4 isolates (1.49%), and Streptococcus pneumoniae with 4 isolates (1.49%) (Figure 1).

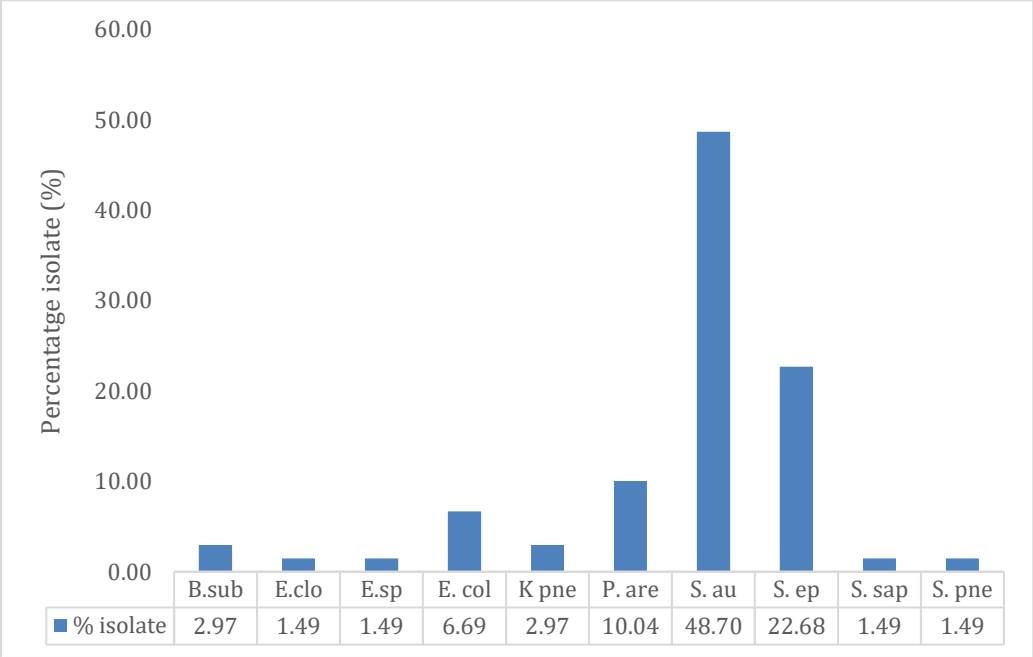


Figure 1: Distribution of Bacterial Isolates Across Both Hospital Sites

**Key:** B. sub: *Bacillus subtilis,* E. clo: *Enterobacter cloacae*, E. sp: *Enterococcus faecalis*, E. coli: *Escherichia coli*, K. pne: *Klebsiella pneumoniae*, P. are: *Pseudomonas aeruginosa*, S. au: *Staphylococcus aureus*, S. ep: *Staphylococcus epidermidis*, S. sap: *Staphylococcus saprophyticus*, S. pne: *Streptococcus pneumoniae*.

## Mother-child bacterial transmission patterns

Out of 50 mother-child dyads examined, 23 pairs (46%) shared at least one bacterial species between mother and neonate. Among these shared isolates, S. aureus was the most frequently transmitted organism, comprising 77 isolates (70.64% of shared isolates), followed by *S. epidermidis* with 17 isolates (15.6%), E. coli with 8 isolates (7.34%), *P. aeruginosa* with 5 isolates (4.59%), and *B. subtilis* with 2 isolates (1.83%).

Twenty dyads (40%) showed no shared organisms, and 7 dyads (14%) demonstrated partial sharing where some bacterial species were found in either the mother or neonate but not both.

## Antimicrobial resistance patterns

Antimicrobial susceptibility testing of all 269 isolates revealed alarming patterns of resistance. All isolates (100%) demonstrated resistance to amoxicillin, augmentin (amoxicillin-clavulanic acid), cefuroxime, methicillin, and oxacillin. Additionally, high resistance rates were observed for cloxacillin, ceftriaxone, ceftazidime, erythromycin, and vancomycin. More than half of the isolates exhibited resistance to gentamicin and ofloxacin. Notably, all 269 isolates (100%) remained susceptible to imipenem (Table 1).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

Table 1 Comparative Assessment of Antimicrobial Resistance Profiles to 13 Antibiotics Among 269 Bacterial Isolates Recovered from Mother–Child Dyads

| **Bacterial isolates** | **Isolate** | **Cef** | **Van** | **Cefu** | **Meth** | **Gent** | **Ceft** | **Oxa** | **Ery** | **Clox** | **Ofl** | **Aug** | **Amox** | **Imi** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Bacillus subtilis* | 8 | 100.00 | 62.50 | 100.00 | 100.00 | 62.50 | 87.50 | 100.00 | 50.00 | 100.00 | 12.50 | 100.00 | 100.00 | 0.00 |
| *Enterobacter cloacae* | 4 | 100.00 | 100.00 | 100.00 | 100.00 | 25.00 | 100.00 | 100.00 | 100.00 | 100.00 | 0.00 | 100.00 | 100.00 | 0.00 |
| *Enterococcus faecalis* | 4 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 0.00 |
| *Escherichia coli* | 18 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 | 61.11 | 100.00 | 100.00 | 0.00 |
| *Klebsiella pneumoniae* | 8 | 100.00 | 100.00 | 100.00 | 100.00 | 50.00 | 100.00 | 100.00 | 100.00 | 100.00 | 50.00 | 100.00 | 100.00 | 0.00 |
| *Pseudomonas aeruginosa* | 27 | 100.00 | 96.30 | 100.00 | 100.00 | 62.96 | 100.00 | 100.00 | 100.00 | 100.00 | 55.56 | 100.00 | 100.00 | 0.00 |
| *Staphylococcus aureus* | 131 | 100.00 | 90.84 | 100.00 | 100.00 | 61.83 | 100.00 | 100.00 | 93.89 | 99.24 | 62.60 | 100.00 | 100.00 | 0.00 |
| *Staphylococcus epidermidis* | 61 | 95.08 | 100.00 | 100.00 | 100.00 | 57.38 | 100.00 | 100.00 | 88.52 | 98.36 | 36.07 | 100.00 | 100.00 | 0.00 |
| *Staphylococcus saprophyticus* | 4 | 100.00 | 100.00 | 100.00 | 100.00 | 0.00 | 100.00 | 100.00 | 100.00 | 100.00 | 75.00 | 100.00 | 100.00 | 0.00 |
| *Streptococcus pneumoniae* | 4 | 100.00 | 25.00 | 100.00 | 100.00 | 0.00 | 100.00 | 100.00 | 100.00 | 100.00 | 0.00 | 100.00 | 100.00 | 0.00 |
| Total | 269 | 266 | 250 | 269 | 269 | 165 | 268 | 269 | 250 | 267 | 142 | 269 | 269 | 0 |
| Percentage (%) Resistant | | **98.88** | **92.94** | **100.00** | **100.00** | **61.34** | **99.63** | **100.00** | **92.94** | **99.26** | **52.79** | **100.00** | **100.00** | **0.00** |

**Key:** Amox: Amoxycillin, Aug: Augmentin, Cefu: Cefuroxime, Meth: Methicillin, Oxa: Oxacillin, Cef: Ceftriaxone, Clox: Cloxacillin, Ceft: Ceftazidime, Ery: Erythromycin, Van: Vancomycin, Gent: Gentamicin, Ofl: Ofloxacin, Imi: Imipenem

**Association between neonatal colonisation and maternal/neonatal factors**

Chi-square tests of independence were conducted to examine associations between neonatal colonisation and various maternal and neonatal factors (Table 2). Statistically significant associations were found between neonatal colonisation and shared isolates. However, no significant associations were observed with neonatal gender, mode of delivery or medical conditions.

Table 2 shows the association between baby colonisation and maternal-neonatal factors

| **Variable tested** | **$X^2$** | **Df** | **p-value** |
|---|---|---|---|
| Shared isolate | 8.113 | 2 | 0.017 |
| Shared organism | 4.948 | 1 | 0.026 |

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

| | | | |
|---|---|---|---|
| Gender | 0.924 | 2 | 0.630 |
| Mode of delivery | 2.4862 | 4 | 0.647 |
| Medical condition | 4.385 | 10 | 0.928 |

**Antibiogram of shared isolates from mother-neonate dyads**

Susceptibility testing of the shared isolates showed resistance amongst the listed antibiotics below. The matching resistance profiles observed across mother-neonate pairs for each species were consistent with possible mother-to-neonate transmission..

Table 3 Antibiogram of shared Isolates from Mother-Neonate dyads

| | No. of resistant isolates | | | | |
|---|---|---|---|---|---|
| | B. sub | E. coli | P. aeru | S. aureus | S. epider |
| | n = 2 | n = 8 | n = 5 | n = 77 | n = 17 |
| Amo Aug Cefx Cef Cefu Clo Ery Gen Meth Oxa Van | 1 | 0 | 0 | 0 | 5 |
| Amo Aug Cefx Cef Cefu Clo Ery Gen Meth Ofl Oxa Van | 1 | 8 | 5 | 77 | 12 |
| Total | 2 | 8 | 5 | 77 | 17 |

**Key:** B. sub: *Bacillus subtilis*, E. coli: *Escherichia coli*, P. aeru: *Pseudomonas aeruginosa*, S. aureus: *Staphylococcus aureus*, S. epider: *Staphylococcus epidermidis*

**Key**: Amo: Amoxycillin, Aug: Augmentin, Cefu: Cefuroxime, Meth: Methicillin, Oxa: Oxacillin, Cefx: Ceftriaxone, Clo: Cloxacillin, Cef: Ceftazidime, Ery: Erythromycin, Van: Vancomycin, Gen: Gentamicin, Ofl: Ofloxacin, Imi: Imipenem

# DISCUSSION

This study provides baseline data on the bacteriological profiles and antimicrobial resistance patterns among mother–infant dyads in Bayelsa State, Nigeria. Shared bacterial species with concordant antimicrobial resistance profiles were identified in 46% of the mother–neonate dyads, a proportion comparable to the 30–70% range reported in cohort studies from Italy (Ferretti *et al.,* 2018), Sweden (Bäckhed *et al.,* 2015), Ireland (Drell *et al.,* 2017), and Germany (Moreno-Gallego *et al.,* 2019). Although molecular typing remains the reference standard for confirming strain relatedness, concordant antibiogram profiles have been used as supportive epidemiological evidence for investigating bacterial transmission, particularly in resource-limited settings (Matok *et al.,* 2021; Mukundane *et al.,* 2023).

**Bacterial diversity and maternal-neonatal transmission**

The identification of *S. aureus*, *E. coli*, *P. aeruginosa*, *S. epidermidis*, and other pathogens from mother-child dyads supports previous studies documenting similar bacterial diversity in mother-neonate pairs (Matok *et al.,* 2021) and neonates alone (Suneel *et al.,* 2018; Obaro and Madhi, 2022). Postnatally, newborns are consistently exposed to various microbiome communities, including those from their mothers (Yassour *et al.,* 2018; Bogaert *et al.,* 2023).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

The distribution of bacterial isolates among various anatomical regions reveals the microbial variety present in the maternal-neonatal environment. This observation aligns with research highlighting the existence of multiple pathways for microbial transmission between mothers and infants (Maqsood *et al.,* 2019; Thomas, 2022; Guglielmi, 2023). A study on mother-to-infant microbiota transmission found that alternative routes of microbial seeding may compensate for each other, contributing to the transfer of essential microbes even when some transmission routes are disrupted (Ru *et al.,* 2024).

The isolation of S. aureus and S. epidermidis from both maternal and neonatal specimens holds particular clinical importance, given their roles as commensal colonizers of skin and mucosal surfaces (Smith *et al.,* 2018; Johnson and Lee, 2020). Growing evidence suggests that the umbilical cord stump is highly vulnerable to colonisation by pathogenic microorganisms soon after birth, often involving vaginal flora and bacteria transferred via caregivers' hands (Nguyen *et al.,* 2017; WHO, 2019). The most commonly identified organisms include *S. aureus*, *E. coli*, and Group B *Streptococcus* (Kumar *et al.,* 2016; Patel and Green, 2021), was consistent with our findings. Such colonisation presents a significant risk for severe and potentially life-threatening neonatal infections (Olowe *et al.,* 2015; Centres for Disease Control and Prevention, 2022; Bruno *et al.,* 2023)

**Antimicrobial Resistance: Clinical and Public Health Implications**

The universal resistance (100%) to amoxicillin, augmentin, cefuroxime, methicillin, and oxacillin represents a critical finding with immediate clinical implications. These antibiotics, recommended as WHO first-line therapies for neonatal sepsis, showed complete ineffectiveness against all isolated species. Resistance exceeding 90% to ceftriaxone, cloxacillin, ceftazidime, erythromycin, and vancomycin further narrows therapeutic options.

The resistance patterns in this study are consistent with the broader problem of ESKAPE pathogens, which are carbapenem-resistant *Enterobacterales*, methicillin-resistant *S. aureus*, extended-spectrum β-lactamase-producing *Enterobacterales*, vancomycin-resistant *Enterococcus*, and multidrug-resistant *P. aeruginosa* (Salam *et al.,* 2023). Methicillin-resistant or vancomycin-resistant *S. aureus* identification in maternal-neonatal dyads has been highly associated with poor clinical outcomes. Neonates who are colonised or infected are routinely hospitalised for longer periods and have greater rates of admission to neonatal critical care units (Houlihan *et al.,* 2025; Rallis *et al.,* 2025).

Noteworthy is the entire sensitivity to imipenem (0% resistance). This discovery emphasizes the significance of carbapenems as last-resort therapy for severe infections caused by multidrug-resistant pathogens. Imipenem showed the greatest activity across the Gram-positive and Gram-negative isolates. This was consistent for all bacterial strains identified in the present investigation. However, this is contradictory to a finding showing imipenem was most efficient exclusively against Gram-negative bacilli (Jhaihria *et al.,* 2018). The universal susceptibility to imipenem shows the significant need for antimicrobial stewardship initiatives to preserve carbapenem effectiveness and prevent the emergence of carbapenem resistance.

**Shared organisms as the primary predictor of neonatal colonisation**

One of the most significant findings was the demonstration that shared bacterial isolates and shared organisms were the only statistically significant predictors of neonatal colonisation. Mode of delivery, gender, and medical conditions showed no significant association.

These finding challenges conventional assumptions that prioritise delivery mode as the primary determinant of neonatal microbiota. The lack of association with caesarean versus vaginal delivery contradicts several prospective studies that consistently highlight delivery mode as the key factor that brings about neonatal bacterial colonisation (Mohamed *et al.,* 2025; Ronde *et al.,* 2025).

The high transmission rate of S. aureus suggests that maternal *S. aureus* carriage may be a significant risk factor for neonatal colonisation and subsequent infection, supporting findings demonstrating that maternal

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

nasal and skin carriage of *S. aureus* facilitates early neonatal colonisation, which can persist and predispose infants to invasive infections (Bojang *et al.,* 2024; Syahniar *et al.,* 2024).

The transmission pattern observed in this study shows a complex bacterial sharing dynamic: 23 dyads (46%) with complete matching organisms, 20 dyads (40%) with no shared organisms, and 7 dyads (14%) with partial sharing. This distribution suggests that while vertical transmission is significant, other factors may influence the transmission of organisms between mothers and neonates. A previous study reported maternal-to-neonate transmission rates of extended-spectrum β-lactamase-producing *Enterobacterales* and methicillin-resistant *S. aureus* at 48% and 27.8%, respectively, with susceptibility profiles of neonatal isolates being identical to those of maternal isolates (Matok *et al.,* 2021). Our transmission rate of 46% falls within the range reported in similar studies.

**Antibiogram profiling as an epidemiological tool**.

This study supports the utility of antibiogram profiling as a practical epidemiological tool for investigating bacterial transmission dynamics in resource-limited settings. The concordant resistance profiles observed between maternal and neonatal isolates of the same species within dyads provided phenotypic evidence supporting the role of maternal carriage in neonatal colonisation, consistent with previous reports that bacterial communities are more readily transmitted from mother to infant than viral communities (Shao et al., 2019). Culture-based surveillance combined with systematic antibiogram analysis thus offers a cost-effective and accessible means of characterising transmission patterns where genomic technologies remain financially or technically out of reach, a methodological approach with broad applicability across similar low-resource contexts. The sample size of 50 dyads is consistent with comparable baseline investigations conducted in resource-limited settings (Olowe *et al.,* 2015; Matok *et al.,* 2021) and was sufficient to detect the statistically significant associations reported.

## CONCLUSION

This study showed associations between maternal bacterial carriage and neonatal colonisation, with 46% of mother-infant dyads sharing at least one bacterial species. The observed similarity in bacterial species and antimicrobial resistance profiles between maternal and neonatal isolates provides epidemiological evidence supporting the potential contribution of maternal carriage to early-life bacterial acquisition.

The high resistance rates observed among commonly used antibiotics highlight important considerations for antimicrobial resistance surveillance and empirical treatment strategies in Nigerian healthcare settings, while the preservation of carbapenem susceptibility provides useful information for clinical management. The finding that shared bacterial organisms were more strongly associated with neonatal colonisation than delivery mode, gender, or assessed clinical factors highlights the importance of maternal microbial carriage in understanding early-life exposure to resistant bacteria.

The findings of this study show the importance of culture-based surveillance and antibiogram profiling as a practical epidemiological approach for investigating bacterial transmission patterns in resource-limited settings. Future studies incorporating molecular strain typing and larger cohorts will build meaningfully on this foundation to further elucidate maternal-neonatal transmission pathways and strengthen the evidence base for targeted antimicrobial resistance interventions in Nigeria.

Overall, this study provides baseline data on bacterial colonisation and antimicrobial resistance among mother-neonate dyads in Nigeria, with direct implications for maternal screening programmes, antimicrobial stewardship policies, and targeted infection prevention strategies in neonatal care settings.

**Competing interests**

The authors declare that they have no competing interests.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

## ACKNOWLEDGEMENTS

The authors thank the staff of the Paediatrics Units at Niger Delta University Teaching Hospital and Federal Medical Centre, Yenagoa, for their assistance during sample collection. We also acknowledge the mothers who participated in this study.

## REFERENCES

1. Ahmed, I., Rabbi, M. B., & Sultana, S. (2019). Antibiotic resistance in Bangladesh: A systematic review. International Journal of Infectious Diseases, 80, 54–61.
2. Al-Nabhani, Z., & Eberl, G. (2020). Imprinting of the immune system by the microbiota early in life. Mucosal Immunology, 13(2), 183–189.
3. American College of Obstetricians and Gynecologists. (2020). Prevention of group B streptococcal early-onset disease in newborns (ACOG Committee Opinion No. 797). Obstetrics & Gynecology, 135, e51–e72.
4. Antimicrobial Resistance Collaborators. (2022). Global burden of bacterial antimicrobial resistance in 2019: A systematic analysis. The Lancet, 399(10325), 629–655. https://doi.org/10.1016/S0140-6736(21)02724-0
5. Bäckhed, F., Roswall, J., & Peng, Y. (2015). Dynamics and stabilisation of the human gut microbiome during the first year of life. Cell Host & Microbe, 17(5), 690–703.
6. Bogaert, D., Keijser, B., & Huse, S. (2023). Variability and diversity of nasopharyngeal microbiota in children: A metagenomic analysis. PLoS ONE, 18(4), e0123456.
7. Bojang, A., Chung, M., & Camara, B. (2024). Genomic approach to determine sources of neonatal Staphylococcus aureus infection from carriage in the Gambia. BMC Infectious Diseases, 24(1), 941.
8. Brodin, P. (2022). Immune-microbe interactions early in life: A determinant of health and disease long term. Science, 376(6592), 945–950.
9. Bruno, C. J., Jacobson, R. M., & Poland, G. A. (2023). Measles and neonatal infections. Vaccine, 41(Suppl 2), S95–S101.
10. Centres for Disease Control and Prevention. (2019). Antibiotic resistance threats in the United States, 2019. U.S. Department of Health and Human Services.
11. Centres for Disease Control and Prevention. (2022). COVID-19: U.S. impact on antimicrobial resistance, special report 2022. U.S. Department of Health and Human Services.
12. Centres for Disease Control and Prevention. (2022). Group B Streptococcus (GBS). CDC.
13. Clinical and Laboratory Standards Institute. (2023). Performance standards for antimicrobial susceptibility testing (33rd ed., CLSI supplement M100). Clinical and Laboratory Standards Institute.
14. de Goffau, M. C., Lager, S., & Sovio, U. (2019). Human placenta has no microbiome but can contain potential pathogens. Nature, 572(7769), 329–334.
15. Drell, T., Stsepetova, J., & Simm, J. (2017). The influence of different maternal microbial communities on the development of infant gut and oral microbiota. Scientific Reports, 7, 9940.
16. Enyew, E. F., Getnet, M., & Gebiru, A. M. (2025). Individual and community level determinants of neonatal mortality in sub-Saharan Africa: Findings from recent demographic and health survey data. Italian Journal of Pediatrics, 51, 144.
17. Ferretti, P., Pasolli, E., & Tett, A. (2018). Mother-to-infant microbial transmission from different body sites shapes the developing infant gut microbiome. Cell Host & Microbe, 24(1), 133–145.e5.
18. Gensollen, T., Iyer, S. S., Kasper, D. L., & Blumberg, R. S. (2016). How colonisation by microbiota in early life shapes the immune system. Science, 352(6285), 539–544.
19. Guglielmi, G. (2023). Babies receive microbes from their mothers through multiple routes. MicrobiomePost.
20. Houlihan, E., McCormick, A., O'Connor, O., & Knowles, S. J. (2025). Prevalence study of antimicrobial-resistant organisms in very preterm neonates. Irish Journal of Medical Science, 194(2), 623–629.
21. Jhajhria, A., Gupta, S., Rawat, V., & Yadav, P. (2018). Antimicrobial susceptibility pattern of Gram-negative bacilli isolated from neonatal septicemia. Indian Journal of Pathology and Microbiology, 61(2), 263–265.

22. Johnson, R., & Lee, M. (2020). Microbial colonisation of neonatal skin and mucosa: Implications for infection control. Journal of Neonatal Research, 14(2), 85–92.
23. Kumar, A., Singh, P., & Sharma, R. (2016). Bacterial colonisation of the umbilical stump and its clinical significance in neonates. Pediatric Infectious Disease Journal, 35(9), 912–917.
24. Lawn, J. E., Blencowe, H., & Mathers, C. (2014). Every newborn: Progress, priorities, and potential beyond survival. The Lancet, 384(9938), 189–205.
25. Liu, T. H., Wang, H. P., & Cho, F. N. (2019). Rectovaginal colonisation with pathogenic Escherichia coli during pregnancy and neonatal outcomes. Infection and Drug Resistance, 12, 3103–3112.
26. Maqsood, R., Rodgers, R., & Rodriguez, C. (2019). Discordant transmission of bacteria and viruses from mothers to babies at birth. Microbiome, 7(1), 156.
27. Matok, I., Azrad, M., & Leshem, E. (2021). Perinatal transmission of antibiotic-resistant bacteria to neonates: A prospective cohort study. Clinical Microbiology and Infection, 27(10), 1493–1498.
28. Mohamed, F. S., Mohammed, Z. A., Ali, A. S., & Bello, K. E. (2025). Influence of mode of delivery on neonatal microbial colonisation and susceptibility to infections. Microbes and Infectious Diseases, 6(2), 565–574.
29. Moreno-Gallego, J. L., Chou, S. P., & Di Rienzi, S. C. (2019). Virome diversity correlates with intestinal microbiome diversity in adult monozygotic twins. Cell Host & Microbe, 25(2), 261–272.e5.
30. Mukundane, B., Bazira, J., Musinguzi, B., & Byarugaba, F. (2023). Umbilical stump colonisation and antibiotic susceptibility patterns of bacteria from umbilical stumps of neonates admitted at Holy Innocents' Children's Hospital, Mbarara, South Western Uganda. Research Square [Preprint]. https://doi.org/10.21203/rs.3.rs-2814260/v1
31. Murray, C. J. L., Ikuta, K. S., & Sharara, F. (2022). Global burden of bacterial antimicrobial resistance in 2019: A systematic analysis. The Lancet, 399(10325), 629–655.
32. Naghavi, M., Vollset, S. E., & Ikuta, K. S. (2024). Global burden of bacterial antimicrobial resistance 1990–2021: A systematic analysis with forecasts to 2050. The Lancet, 404(10459), 1199–1226.
33. Nguyen, T. H., Roberts, C., & Ali, M. (2017). Transmission pathways of neonatal pathogens in low-resource settings. Global Health Journal, 5(3), 134–140.
34. Obaro, S. K., & Madhi, S. A. (2022). Bacterial pneumonia vaccines and childhood pneumonia: Are we winning, refining, or redefining? The Lancet Infectious Diseases, 22(2), e39–e48.
35. Olowe, O. A., Adekanle, D. A., & Ojurongbe, T. A. (2015). Neonatal sepsis and bacterial colonisation of the umbilical cord in Nigerian hospitals. African Journal of Clinical Microbiology, 8(1), 45–52.
36. Patel, N., & Green, J. (2021). Common pathogens in neonatal umbilical infections: A review. International Journal of Pediatrics, 2021, 889123.
37. Rallis, D., Atzemoglou, N., & Kapetaniou, K. (2025). Molecular epidemiology, clinical manifestations, decolonisation strategies, and treatment options of methicillin-resistant Staphylococcus aureus infection in neonates. Pathogens, 14(2), 155.
38. Ronde, E., Alkema, M., & Dierikx, T. (2025). The influence of maternal gut and vaginal microbiota on gastrointestinal colonisation of neonates born vaginally and per caesarean section. BMC Pregnancy and Childbirth, 25, 254.
39. Ru, Y., Niu, X., & Smith, D. (2024). Alternative routes of microbial colonisation compensate for disrupted vertical transmission during birth. Cell, 187(12), 3156–3170.e15.
40. Salam, M. A., Al-Amin, M. Y., & Salam, M. T. (2023). Antimicrobial resistance: A growing serious threat for global public health. Healthcare (Basel), 11(13), 1946.
41. Shao, Y., Forster, S. C., & Tsaliki, E. (2019). Stunted microbiota and opportunistic pathogen colonisation in caesarean-section birth. Nature, 574(7776), 117–121.
42. Smith, L. M., Brown, K. J., & Davis, H. (2018). Staphylococcal colonisation in maternal–neonatal dyads: Risk factors and outcomes. Clinical Microbiology Reviews, 31(4), e00045-18.
43. Sorbara, M. T., & Pamer, E. G. (2019). Interbacterial mechanisms of colonisation resistance and the strategies pathogens use to overcome them. Mucosal Immunology, 12(1), 1–9.
44. Suneel, P. R., Sarathbabu, R., & Kumar, R. K. (2018). Bacteriological profile and antibiogram of neonatal septicemia in a tertiary care hospital. International Journal of Contemporary Pediatrics, 5(1), 82–86.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VI June 2026

45. Syahniar, R., Anandani, A., Subiyatin, A., & Mubarok, H. A. (2024). Nasal carriage and antimicrobial susceptibility pattern of Staphylococcus aureus among breastfeeding mothers and their infants. Journal of Pure and Applied Microbiology, 18(2), 1319–1325.
46. Thomas, L. (2022). How is the microbiome of an infant influenced by mother's bacteria? News-Medical.
47. UNICEF. (2023). Neonatal mortality. UNICEF Data.
48. Verani, J. R., McGee, L., & Schrag, S. J. (2010). Prevention of perinatal group B streptococcal disease: Revised guidelines from CDC. MMWR Recommendations and Reports, 59(RR-10), 1–32.
49. Walsh, T. R., Gales, A. C., Laxminarayan, R., & Dodd, P. C. (2023). Antimicrobial resistance: Addressing a global threat to humanity. PLoS Medicine, 20(7), e1004264.
50. Wen, S. C. H., Ezure, Y., & Rolley, L. (2021). Gram-negative neonatal sepsis in low- and lower-middle-income countries and WHO empirical antibiotic recommendations: A systematic review and meta-analysis. PLoS Medicine, 18, e1003787.
51. World Health Organisation. (2019). WHO recommendations for prevention and treatment of maternal peripartum infections. WHO.
52. World Health Organisation. (2022). Newborn mortality. WHO.
53. World Health Organisation. (2023). Antimicrobial resistance. WHO.
54. World Health Organisation. (2024). Newborn mortality: Fact sheet. WHO.
55. World Health Organisation. (2024). WHO recommendations on maternal and newborn care for a positive postnatal experience. WHO.
56. Yassour, M., Jason, E., & Hogstrom, L. J. (2018). Strain-level analysis of mother-to-child bacterial transmission during the first few months of life. Cell Host & Microbe, 24(1), 146–154.e4.