Born Resistant? Phylogroup stratification of *Escherichia coli* in healthy mother-neonate dyads

Suchandra Banerjee[1], Amrita Bhattacharjee[2], Rajlakshmi Viswanathan[3], Mallika Lavania[4], Sulagna Basu[2]

[1]*School of Biotechnology, KIIT University, Bhubaneswar 751024, Odisha, India.*
[2]*Division of Bacteriology, ICMR-National Institute for Research in Bacterial Infections, P-33 CIT Road, Scheme XM, Beliaghata, Kolkata-700010, India.*
[3]*Bacteriology Group, ICMR-National Institute of Virology, MCC Campus,130/1, Sus Road, Pashan, Pune- 411021, India.*
[4]*Enteric Viruses Group, ICMR-National Institute of Virology, 20-A Dr. Ambedkar Road, Pune-411001, India*

Email: S.Banerjee25@outlook.com

INTRODUCTION

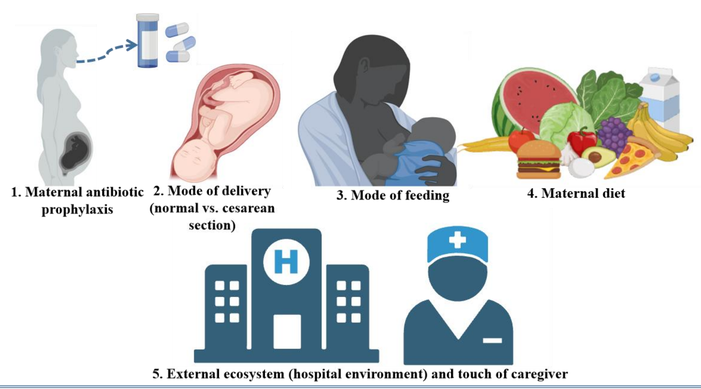
Escherichia coli colonizes the neonatal intestinal mucosa post-birth, functioning as a keystone pioneer taxon during the critical period of immune system maturation. The absence of colonization resistance in this immunologically naïve environment enables early colonizers to exert priority effects on microbial succession and metabolic programming. *E. coli* phylogroups are functionally stratified — phylogroups A and B1 represent commensal lineages, whereas B2, D, E, and F harbor extraintestinal virulence determinants. Its heightened genomic plasticity facilitates rapid antimicrobial resistance gene acquisition, creating One Health surveillance imperatives.

Just after birth, several factors influence which phylogroups dominate the infant gut. Although maternal transmission is presumed to seed initial colonization, it remains unclear whether commensals persist or if pathogenic lineages with higher fitness outcompete them. Also, the selective fitness conferred by antibiotics might enable pathobiont lineages to outcompete the commensals, remodeling the gut microbiome toward a multidrug-resistant architecture.

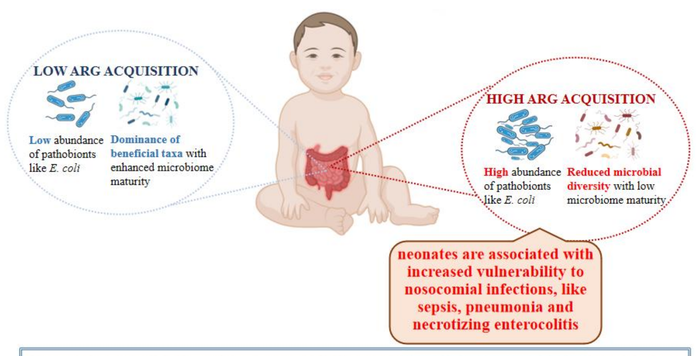
Critically, *E. coli* phylogroup distribution in healthy neonates remains undercharacterized, hindering rational design of microbiome-based interventions.

OBJECTIVES:

- **Characterize the *E. coli* phylogenetic landscape in healthy mother–neonate pairs**
- **Delineate antimicrobial susceptibility patterns across phylogroups**

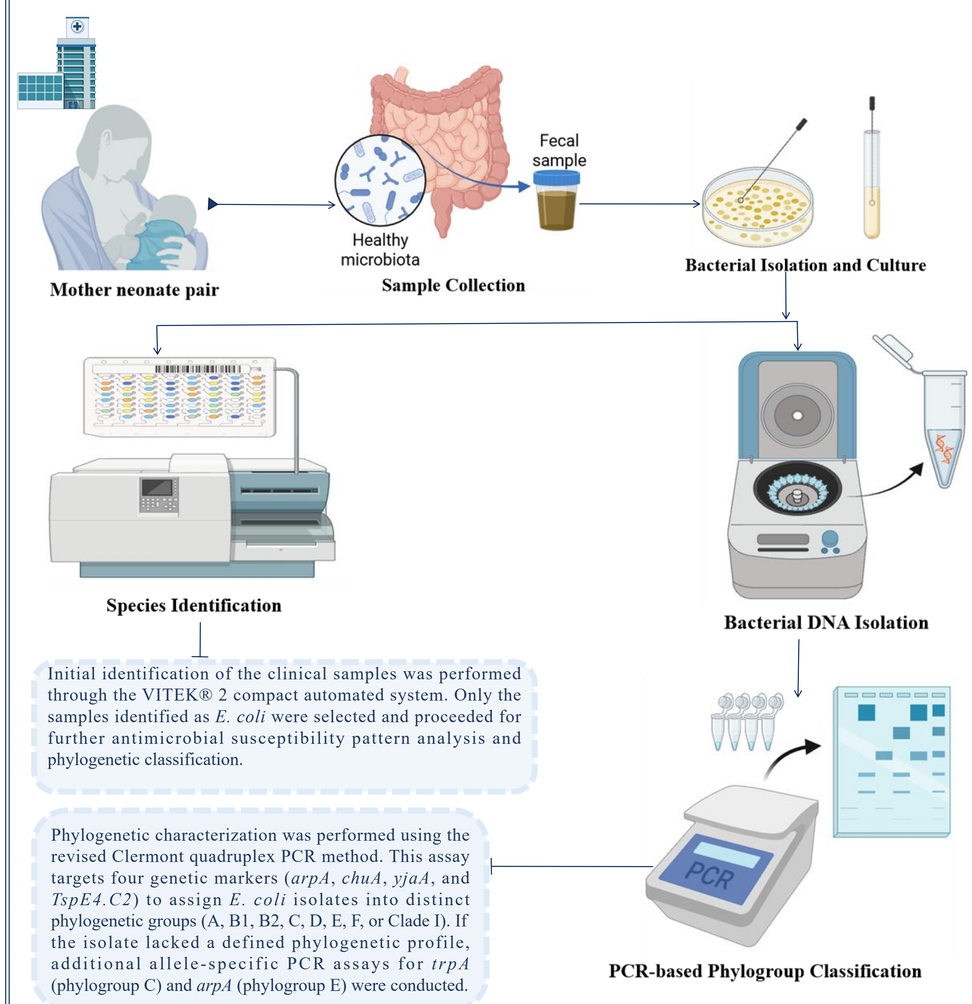


Multifactorial overview of host- and environment-derived variables modulating gut microbial succession in neonates.
Neonates, defined as newborns within the first 28 days of life, represent one of the most immunologically and physiologically vulnerable human populations. This early window is critical, as it marks the foundational period of gut microbiome establishment. Disruptions in microbial colonization during this phase can heighten susceptibility to infections and predispose individuals to immune-mediated conditions— including asthma, allergies, and chronic inflammatory diseases, later in life. The early-life microbiota plays a pivotal role in modulating the nascent immune system, shaping the mucosal barrier integrity, and maintaining intestinal homeostasis.



Neonatal gut antibiotic resistant gene (ARG) acquisition dichotomy and infection risk. Low ARG acquisition correlates with commensal dominance and microbiome stability, while high ARG acquisition, mainly driven by horizontal gene transfer (HGT) is associated with reduced microbial diversity and dysbiosis, significantly elevating vulnerability to a range of nosocomial infections.

METHODS



Initial identification of the clinical samples was performed through the VITEK® 2 compact automated system. Only the samples identified as *E. coli* were selected and proceeded for further antimicrobial susceptibility pattern analysis and phylogenetic classification.

Phylogenetic characterization was performed using the revised Clermont quadruplex PCR method. This assay targets four genetic markers (*arpA*, *chuA*, *yjaA*, and *TspE4.C2*) to assign *E. coli* isolates into distinct phylogenetic groups (A, B1, B2, C, D, E, F, or Clade I). If the isolate lacked a defined phylogenetic profile, additional allele-specific PCR assays for *trpA* (phylogroup C) and *arpA* (phylogroup E) were conducted.

RESULTS

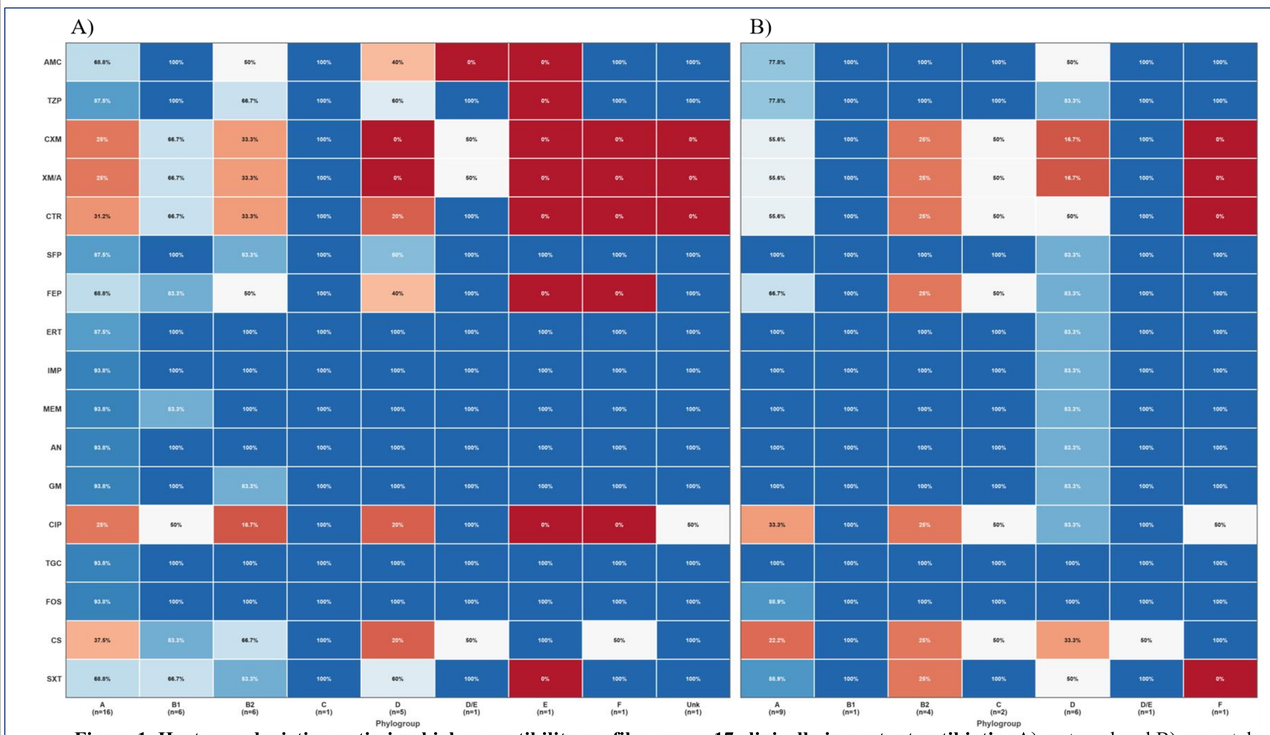


Figure 1. Heat map depicting antimicrobial susceptibility profiles across 17 clinically important antibiotics A) maternal and B) neonatal bacterial isolates. Of study sample size (n=99), only those identified as *E.* coli (n=62) were proceeded for investigation.

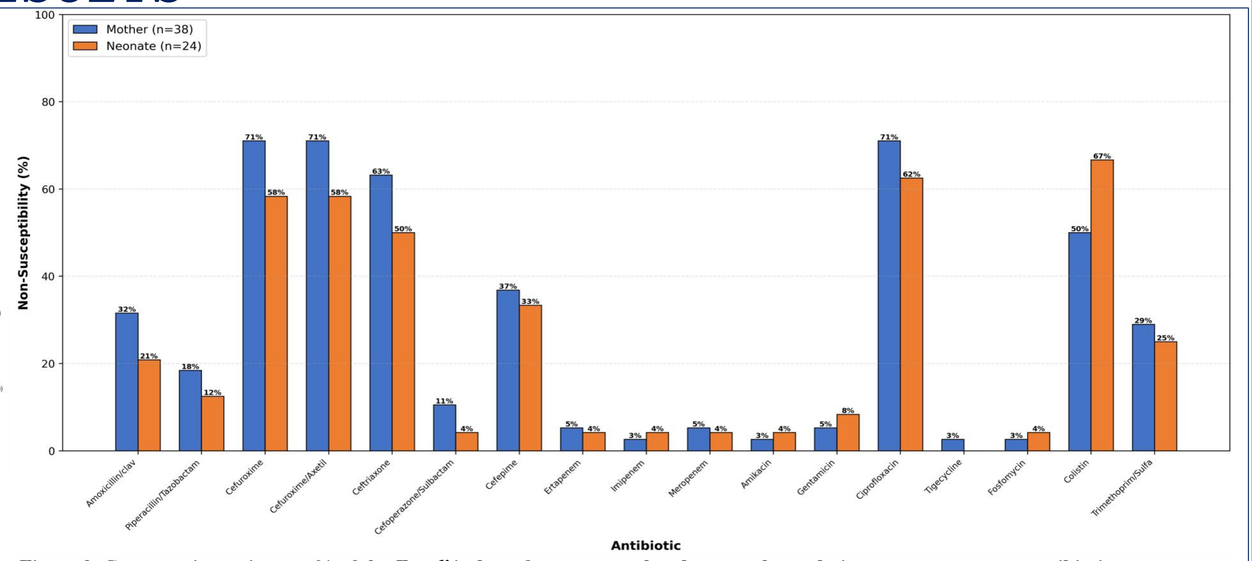


Figure 3. Comparative resistance % of the *E. coli* isolates from maternal and neonatal populations across seventeen antibiotics. Resistance was highest among cephalosporins, with cefuroxime and cefuroxime/axetil recording the greatest rates in both maternal (71% each) and neonatal (58% each) isolates, followed by ceftriaxone (mothers 63%, neonates 50%), collectively indicating a high burden of third-generation cephalosporin resistance in both populations. Ciprofloxacin non-susceptibility was notably elevated in mothers (71%) and neonates (62%), while colistin showed substantially higher non-susceptibility in neonates (67%) compared to mothers (50%), raising concern for last-resort antibiotic failure in the neonatal group. In contrast, carbapenems (ertapenem, imipenem, meropenem) and aminoglycosides (amikacin) demonstrated the lowest non-susceptibility rates across both groups (≤5%), affirming their retained activity against these isolates. Tigecycline and fosfomycin similarly showed low non-susceptibility (≤4%), indicating preserved efficacy.

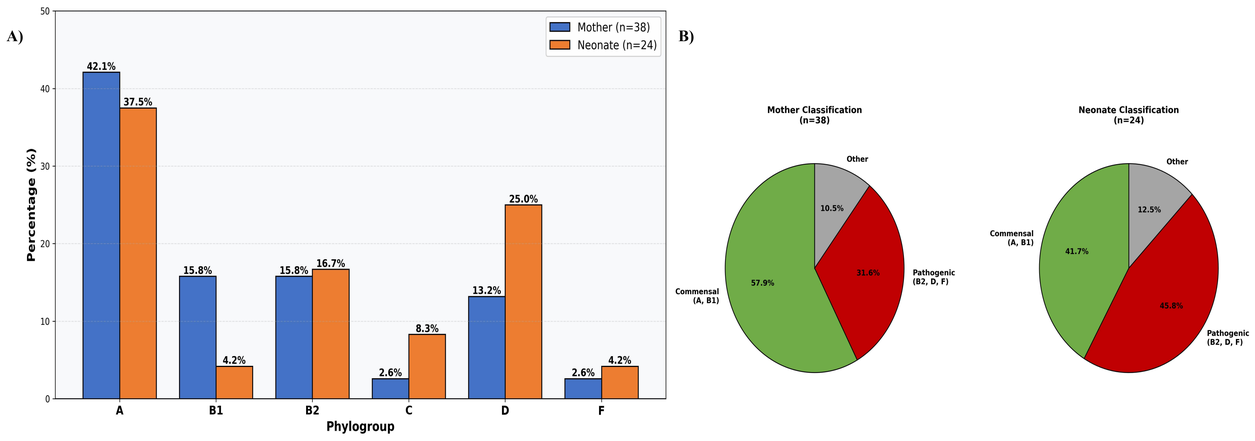


Figure 2. *E. coli* phylogroup distribution demonstrates distinct, non-overlapping maternal-neonatal clustering. A) Percentage distribution of phylogroups A, B1, B2, C, D, and F among maternal (n=38) and neonatal (n=24) isolates. Phylogroup A was the most predominant in both groups, though mothers showed a higher proportion (42.1%) compared to neonates (37.5%). Notably, phylogroup D was markedly more frequent in neonates (25.0%) than in mothers (13.2%), while phylogroup B1 was more prevalent among maternal isolates, suggesting divergent colonisation patterns between the two populations. B) Distribution of commensal versus pathogenic phylogroups in maternal and neonatal isolates. Neonates exhibit higher pathogenic phylogroup prevalence (45.8%) compared to mothers (31.6%).

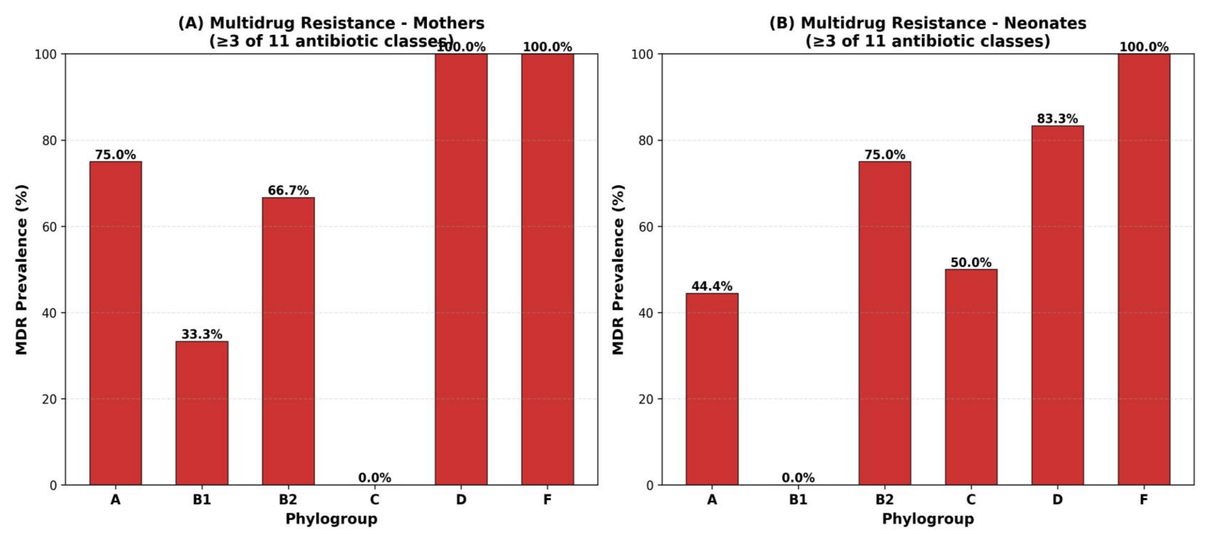


Figure 4. Multidrug resistance prevalence stratified by phylogroup. Comparison of MDR prevalence across phylogroups in (A) maternal versus (B) neonatal isolates. MDR defined as non-susceptibility to ≥3 antimicrobial classes.

CONCLUSIONS

This cross-sectional study serves as the blueprint for microbiome monitoring in vulnerable populations and indicates that early gut colonization is governed by ecological pressures. The presence of *E. coli* phylogroups B2, D, and F, marked by MDR traits in healthy infants is alarming and signifies the silent transmission of high-risk strains. This molecular epidemiological approach positions phylogenetic surveillance as an indispensable tool for comprehensive AMR mitigation strategies.

Phylogenetic studies are essential for combating global antimicrobial resistance.

Microbiome science is the cornerstone for precision medicine, bridging host genetics and environmental exposures aiding targeted therapeutics within a One Health framework.

Investing in mother-neonate research reduces healthcare expenditure and strengthens economic resilience across generations.

Phylogenetic analysis maps evolutionary relationships and genetic diversity, thereby aiding in targeted vaccine development.