

Molecular Detection of Antimicrobial Resistance Genes in Fecal *Escherichia Coli* from Under-Monitored Regions: A Review

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Abstract

Antimicrobial resistance (AMR) is a growing global threat, undermining the efficacy of antibiotics and contributing to poor clinical outcomes. *Escherichia coli* (*E. Coli*), though typically a harmless gut commensal, plays a pivotal role in the carriage and spread of resistance genes through plasmid-mediated mechanisms. This review investigates three key AMR genes, QnrA, ErmB, and AAC(6')-Ib-cr, in fecal *E. Coli* isolates. These genes confer resistance to fluoroquinolones, macrolides, and aminoglycosides, respectively, and are often embedded within mobile genetic elements, increasing their capacity for horizontal transfer across species and environments. Using molecular detection techniques, including conventional PCR, multiplex PCR, and qPCR, the study explores gene prevalence and public health implications in under-monitored regions, particularly Nigeria. Findings highlight the silent burden of AMR in asymptomatic carriers and emphasize the importance of fecal surveillance, integration of genomic tools, and One Health strategies. The study calls for routine gene-based monitoring and stricter regulation of antibiotic use to curb the spread of resistance determinants in both clinical and community settings.

Keywords: Antimicrobial resistance, *Escherichia coli*, Resistance genes, Fecal surveillance, Molecular detection, Nigeria.

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INTRODUCTION

The molecular surveillance of antimicrobial resistance (AMR) genes in commensal *Escherichia coli* (*E. Coli*) is a crucial yet under-prioritized field in global health. While much focus has been given to clinical isolates, recent attention has turned to fecal carriage of resistance genes in asymptomatic individuals. These silent carriers contribute significantly to the community-wide transmission of resistance traits (Arcilla *et al.*, 2017). The detection of these resistance determinants in healthy individuals reflects a silent and highly mobile reservoir of AMR genes circulating in communities. As such, mapping the global and regional prevalence of QnrA, ErmB, and AAC(6')-Ib-cr is vital for anticipating resistance outbreaks and informing public health interventions (Adel *et al.*, 2021).

E. Coli is a Gram-negative, rod-shaped, facultative anaerobic bacterium belonging to the Enterobacteriaceae family. It colonizes the gastrointestinal tract of humans and animals shortly after birth and generally exists in a state of mutualism with its host. For decades, *E. Coli* has served as a model organism in molecular biology due to its relatively simple genome, fast growth rate, and ease of cultivation. It plays a role in the synthesis of vitamins such as vitamin K, helps exclude pathogenic bacteria through competitive exclusion, and contributes to gut immunity (Belkaid and Hand, 2014). However, not all *E. Coli* strains are benign. The species encompasses a spectrum ranging from non-pathogenic commensals to virulent pathotypes capable of causing diseases such as urinary tract infections (UTIs), neonatal meningitis, enterotoxigenic diarrhea, and sepsis (Figure 1) (Croxen

et al., 2013). The same genomic flexibility that allows *E. Coli* to thrive in diverse environments also enables it to acquire virulence and resistance determinants via mobile genetic elements. This duality, harmless resident

and occasional killer, places *E. Coli* in a unique position within microbial ecology and infectious disease research (Tenaillon *et al.*, 2022).

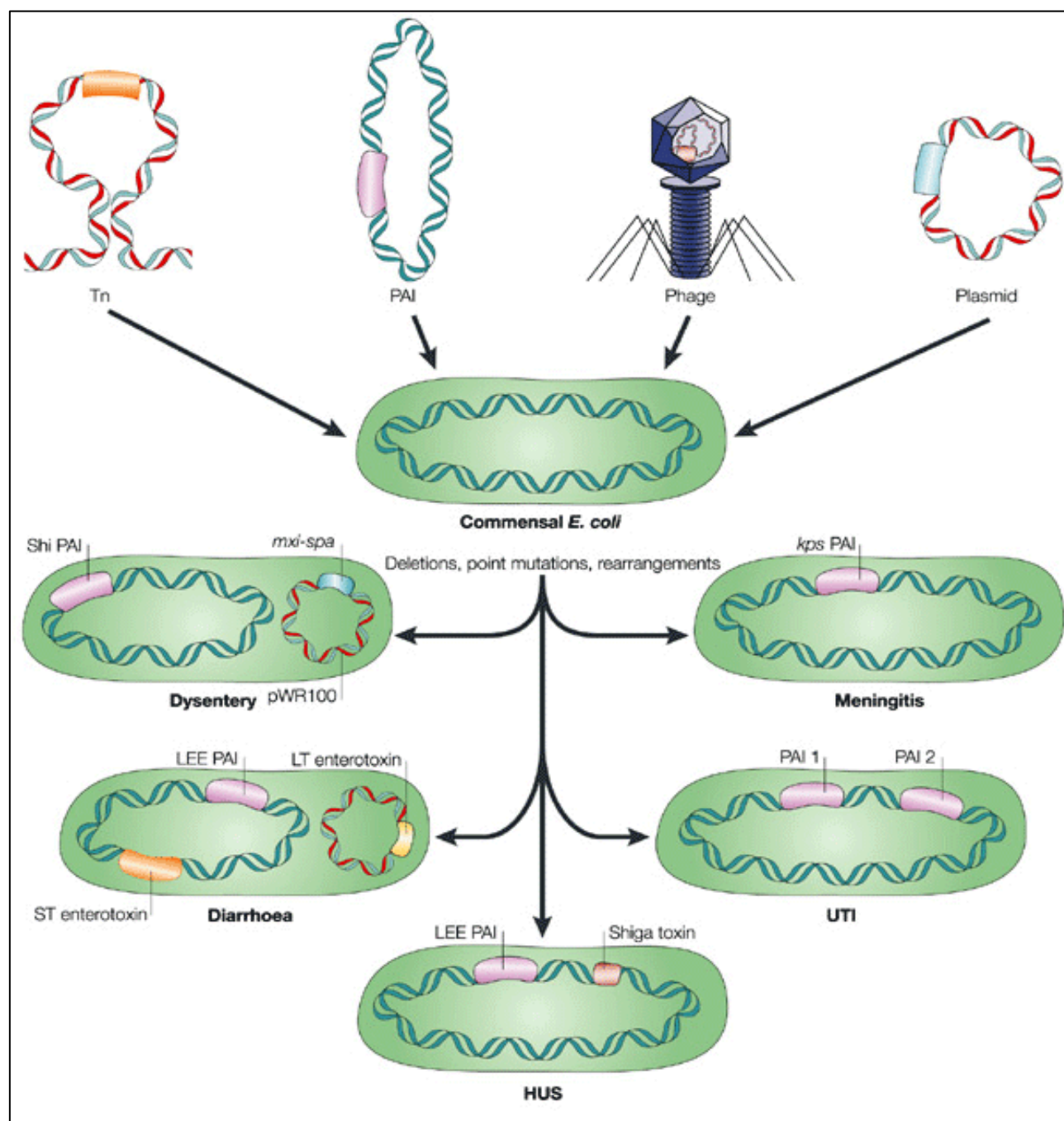


Figure 1: Dual Role of *Escherichia coli* as a Commensal and Pathogen (Source: Gomez-Gomez *et al.*, 2022).

Importantly, even the non-pathogenic strains of *E. Coli* are far from passive. They function as reservoirs and conduits for resistance genes within the gut microbiome (Wang *et al.*, 2017). Through mechanisms such as conjugation and transposition, these strains can exchange genetic material with pathogenic bacteria coexisting in the same microbial community. The gut

thus becomes an incubator not only for microbial diversity but also for resistance evolution and gene transfer. Fecal *E. Coli* in healthy individuals can silently carry genes conferring resistance to critical antibiotics, and these genes can be passed to pathogens during periods of co-infection or stress, such as antibiotic exposure (Bezabih *et al.*, 2021).

Overview of the Global Rise in Antimicrobial Resistance



Figure 2: Spread of Antibiotic Resistance Use in Healthcare, Agriculture, and Community Settings in West Africa
(Source: Adenaya *et al.*, 2025).

Antimicrobial resistance (AMR) has escalated from an emerging clinical challenge to a full-fledged global crisis. The once-transformative impact of antibiotics has diminished in the face of bacterial adaptation, leading to a resurgence of infections once thought to be easily curable. According to the World Health Organization (WHO), AMR causes over 1.27 million direct deaths annually, with millions more affected by complications arising from untreatable infections. This burden is disproportionately borne by low- and middle-income countries (LMICs) particularly in West African region (Figure 2). In these countries factors such as unregulated antibiotic use, poor sanitation, and lack of diagnostic facilities exacerbate the problem (WHO, 2019).

The mechanisms driving the global expansion of AMR are multifactorial. First, the widespread misuse and overuse of antibiotics in both human medicine and animal agriculture create intense selective pressures that favor resistant strain. In many regions, antibiotics can be purchased over-the-counter without prescriptions, leading to inappropriate dosing and duration. In the livestock industry, antibiotics are often used as growth promoters or prophylactics, further accelerating resistance gene selection in zoonotic bacteria (Ventola, 2015). Second, globalization and human mobility facilitate the transcontinental spread of resistant bacteria.

International travel, medical tourism, and global trade in food and animals contribute to the dissemination of resistance determinants across borders. Surveillance studies have revealed the rapid emergence of resistant clones such as extended-spectrum β -lactamase (ESBL)-producing *E. Coli*, carbapenem-resistant Enterobacteriaceae, and fluoroquinolone-resistant *Salmonella* strains in previously unaffected regions (Igbinosa *et al.*, 2022). Third, environmental contamination plays a central role in maintaining AMR gene pools. Pharmaceutical manufacturing waste, hospital effluents, and untreated sewage release antibiotics and resistant bacteria into the environment. These ecological reservoirs, often overlooked in policy discussions, contribute significantly to the persistence and spread of resistance genes in human and animal populations (Aworh *et al.*, 2020).

In this global landscape, fecal *E. Coli* serves as a key surveillance target. Unlike pathogens that may be absent or infrequent in a given population, *E. Coli* is ubiquitous, abundant, and genetically tractable. It provides a reliable snapshot of resistance gene circulation at the community level and offers early warning signs of emerging resistance trends (Igbinosa *et al.*, 2022; Chibuzor *et al.*, 2021). Overuse of Antibiotics in healthcare, livestock, and communities creates selection pressure that fuels AMR (Figure 3).

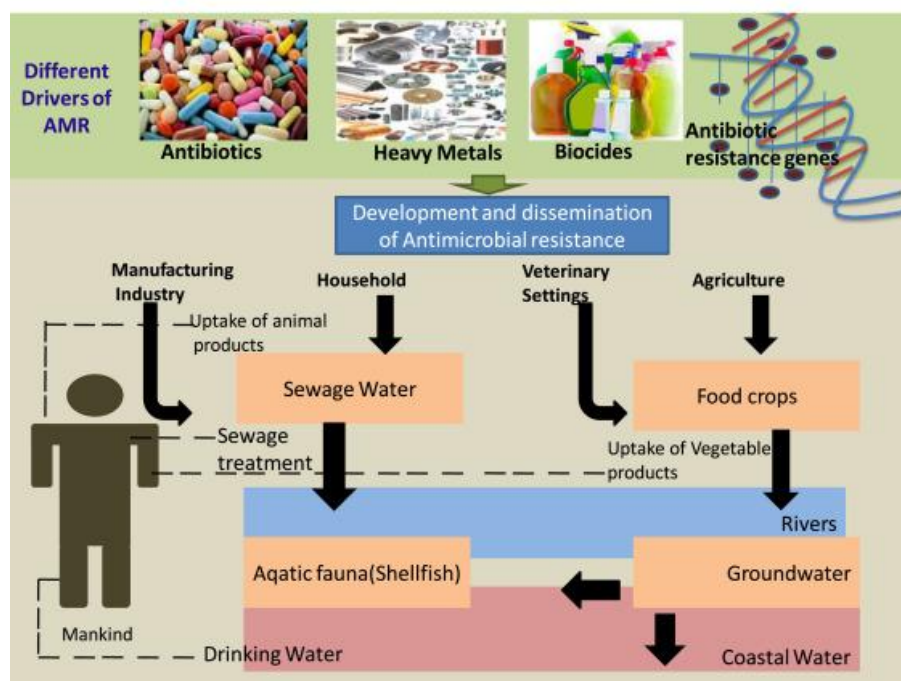


Figure 3: Global Drivers of Antimicrobial Resistance (AMR). (Source: Ahmad *et al.*, 2021)

Global Distribution of Qnr, Erm, and AAC(6')-Ib-cr Genes

The emergence of QnrA was first reported in the United States in the late 1990s, but since then, its prevalence has expanded rapidly across continents (Figure 4). In Europe, multiple surveillance studies conducted between 2015 and 2022 have identified QnrA in both clinical and fecal *E. Coli* isolates, particularly in Spain, France, and Italy, where quinolone use in humans and agriculture remains high (Figure 4). In Germany, a multicenter study conducted in 2019 identified QnrA in

8.4% of fecal *E. Coli* isolates from hospitalized patients, many of whom had not recently been exposed to quinolones, suggesting environmental or community-based acquisition (Figure 4). Similarly, in the UK, large-scale screenings in 2020 revealed QnrA in both hospital-acquired and community-acquired *E. Coli*, with carriage rates as high as 12% in certain urban populations (Figure 4). The gene was frequently associated with IncF plasmids and co-carried other resistance determinants, raising alarms about the potential for co-selection and horizontal gene transfer (Jacoby *et al.*, 2006).



Figure 4: Global Distribution of Qnr, Erm, and AAC(6')-Ib-cr Genes across Human and Environmental *E. Coli* Isolates. (Source: PMC / NCBI)

Across Asia, the prevalence of QnrA is even more pronounced. In China, where fluoroquinolone overuse in livestock and aquaculture is well-documented, QnrA has been detected in over 20% of fecal *E. Coli* isolates from both humans and animals (Figure 4). A national molecular epidemiology study published in 2021 showed that QnrA-positive *E. Coli* strains were genetically diverse, indicating multiple independent acquisition events. India has reported similar trends, with up to 18% QnrA positivity in rural communities, often associated with poor sanitation and over-the-counter antibiotic sales (Figure 4). Bangladesh and Vietnam have also emerged as hotspots for QnrA dissemination, particularly in peri-urban slums and poultry farming sectors (Jacoby *et al.*, 2006).

Sub-Saharan Africa Distribution of Qnr, Erm, and AAC(6')-Ib-cr Genes

In Sub-Saharan Africa, data on the molecular epidemiology of these genes remain fragmented but are increasingly being recognized as essential for building local AMR strategies. Several recent studies have highlighted the growing prevalence of plasmid-mediated resistance genes in fecal *E. Coli* isolates across Nigeria,

Ghana, Kenya, Ethiopia, and South Africa. In Ghana, studies have documented QnrA and AAC(6')-Ib-cr in *E. Coli* from human waste, hospital effluent, and local markets, with genetic evidence pointing to animal-human-environmental cross-transmission. Kenyan investigations into AMR carriage among slum populations in Nairobi have found all three genes co-occurring in fecal samples from children, adults, and animals living in close quarters, highlighting the importance of the One Health framework in understanding regional AMR trends (Figure 5). Despite these concerning findings, Sub-Saharan Africa still faces major gaps in AMR surveillance infrastructure. Molecular diagnostics are often unavailable outside of research institutions, and there is no integrated AMR database that links human, animal, and environmental data. As a result, resistance gene dissemination may go undetected for years, allowing for the establishment of stable reservoirs within human and ecological niches. The rising prevalence of QnrA, ErmB, and AAC(6')-Ib-cr in fecal *E. Coli* in Nigeria and surrounding countries should therefore be considered a warning sign for the emergence of difficult-to-treat infections in the near future. (Nsofor *et al.*, 2021; West African Review, 2024).

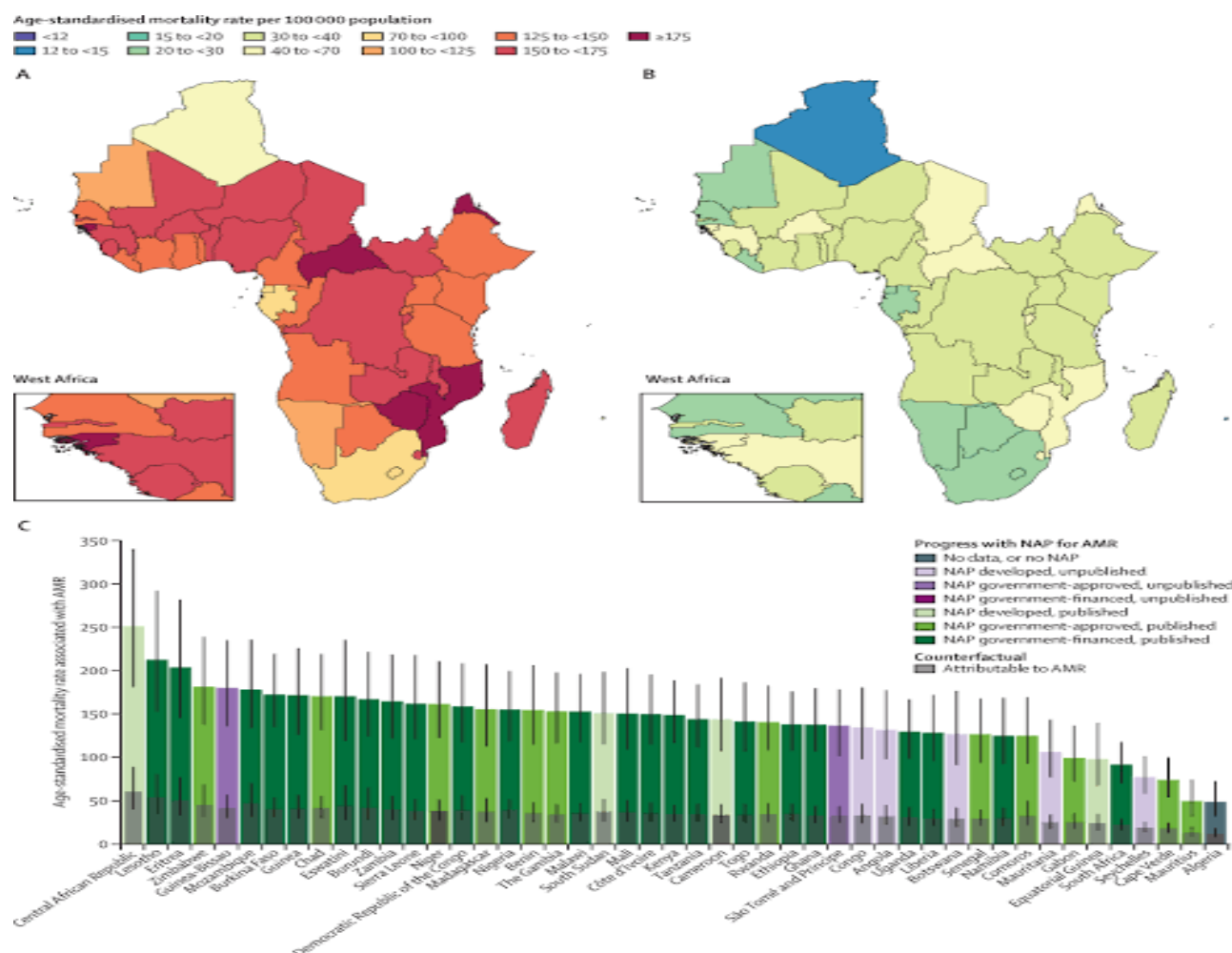


Figure 5: Map of Antimicrobial Resistance Burden in West Africa (Source: WHO, 2021)

Distribution of Qnr, Erm, and AAC(6')-Ib-cr Genes in Nigeria

In Nigeria, recent molecular studies have revealed a disturbingly high rate of fecal carriage of QnrA, ErmB, and AAC(6')-Ib-cr among both healthy individuals and patients without recent antibiotic exposure. Ajayi *et al.*, (2012) first reported the detection of QnrA and QnrB in fecal *E. Coli* from cattle and human handlers in Ado-Ekiti, highlighting the zoonotic interface of resistance gene circulation. A subsequent study in Southern Nigeria by Nsofor *et al.*, (2021) found over 20% of quinolone-resistant *E. Coli* carried QnrA, most of which also harbored the aac(6')-Ib-cr gene. These findings point to the role of both environmental and agricultural exposure routes in resistance

development. (Igbinosa *et al.*, 2022; Ajayi *et al.*, 2012). In 2023, study conducted in Abuja among poultry workers and nearby residents demonstrated high co-carriage of QnrA and AAC(6')-Ib-cr in community fecal samples, with a correlation between resistance gene detection and proximity to poultry farms (Igbinosa *et al.*, 2022). These genes were located on conjugative plasmids capable of transferring into lab strains of *E. Coli* during in vitro conjugation assays, confirming their potential for rapid horizontal spread. Similarly, data from Kano and Lagos show frequent detection of ErmB in *E. Coli* isolated from vegetables irrigated with untreated wastewater, illustrating the environmental dimension of AMR in Nigeria

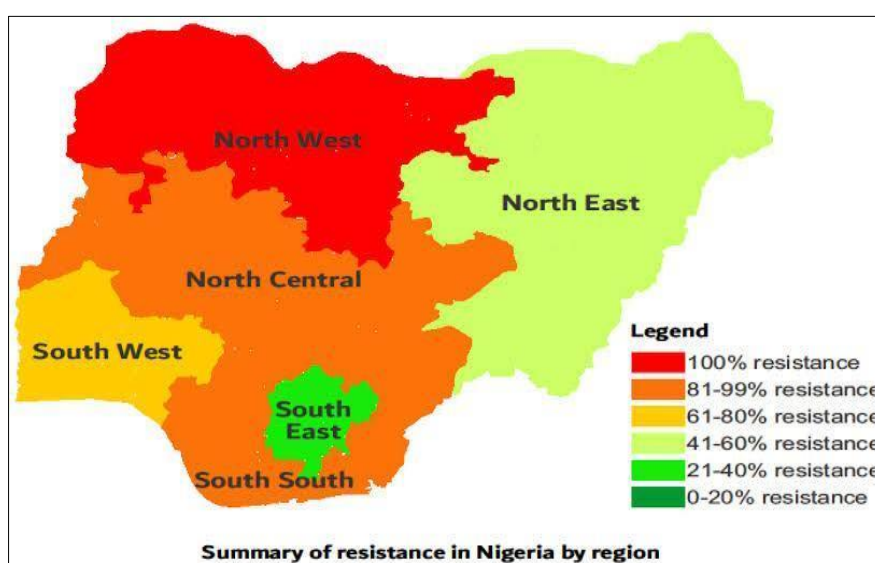


Figure 6: Map of Nigerian Showing Regions Reporting AMR Gene-Positive *E. Coli* in Humans and Animals (Source: WHO, 2020).

Importance of Monitoring Resistance in Fecal *E. Coli* as a Reservoir of Resistance Genes

Fecal *E. Coli* represents one of the most effective indicators of the antimicrobial resistance burden circulating silently in human populations. Its ubiquity in stool samples, adaptability to various environmental conditions, and capacity for horizontal gene transfer make it a strategic target for molecular epidemiological studies. (Aworh *et al.*, 2023). Unlike clinical isolates, which only capture resistance in the context of active infection, fecal *E. Coli* provides insights into the background carriage of resistance genes among healthy individuals, individuals who may later become sources of transmission in households, food chains, or healthcare settings. One of the critical roles of commensal *E. Coli* is that of a genetic reservoir. Through plasmids, integrons, and transposons, fecal *E. Coli* harbors a wide array of resistance determinants that can be passed to other enteric pathogens such as *Klebsiella pneumoniae*, *Shigella*, or *Salmonella*. This gene transfer can occur within the gut, in wastewater systems, or

during food handling. Resistance genes found in fecal bacteria often share homology with those detected in invasive pathogens, suggesting an evolutionary and epidemiological continuum between the gut microbiome and clinical infection.

QnrA, ErmB, and AAC(6')-Ib-cr Genes QnrA gene

QnrA is a plasmid-mediated quinolone resistance (PMQR) gene that encodes a pentapeptide repeat protein capable of protecting DNA gyrase and topoisomerase IV from fluoroquinolone inhibition. While not conferring high-level resistance on its own, QnrA enables bacterial survival in sub-inhibitory concentrations of fluoroquinolones, facilitating the selection of additional chromosomal mutations. It is frequently found on multi-resistance plasmids and often coexists with other resistance determinants, making it a marker for multidrug resistance potential (Nguyen *et al.*, 2023). Quinolone resistance was first considered to be chromosomally mediated, arising through mutations in

the quinolone resistance-determining regions (QRDRs) of *gyrA* and *parC* genes. However, discovery of Qnr proteins encoded on plasmids introduced a paradigm shift. QnrA, originally identified in *Klebsiella pneumoniae* in the early 2000s, functions by physically shielding DNA gyrase and topoisomerase IV from quinolone inhibition (Jacoby *et al.*, 2006). This protein-mediated mechanism confers low-level resistance but facilitates the emergence of high-level resistance when combined with chromosomal mutations (Jacoby, 2005; Rodríguez-Martínez *et al.*, 2016). Recent surveillance in Sub-Saharan Africa and Asia has

confirmed the presence of QnrA genes in fecal *E. Coli* from non-hospitalized individuals (Igbinosa *et al.*, 2022). Their co-existence with extended-spectrum beta-lactamase (ESBL) genes, such as *bla*CTX-M, has been observed, especially in settings with high antibiotic misuse. In these contexts, QnrA serves as both a resistance determinant and a marker of co-resistance and plasmid mobility (Aworh *et al.*, 2023). The model showing how QnrA binds to DNA gyrase and topoisomerase IV, reducing fluoroquinolone activity was established (Figure 7).

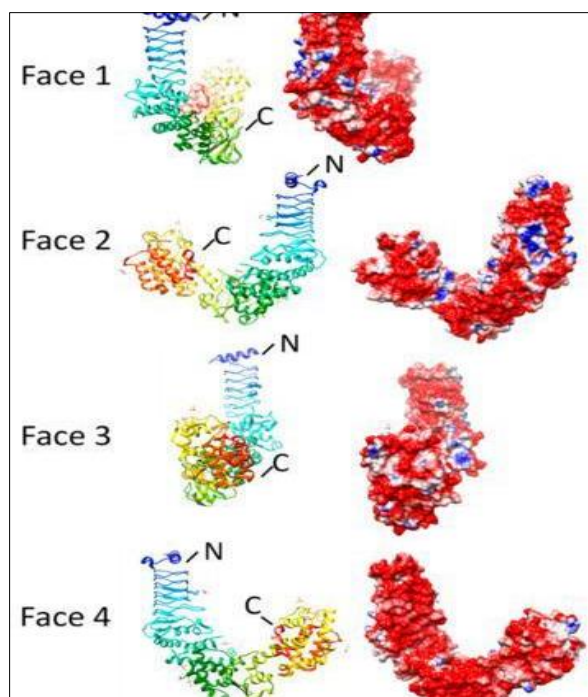


Figure 7: Model for Binding Process of QnrA DNA gyrase and topoisomerase (Source: Ruojing, 2021)

ErmB gene

ErmB is a ribosomal methyltransferase gene that modifies the 23S rRNA of the bacterial ribosome, conferring resistance to macrolides, lincosamides, and streptogramin B (MLSB). Its mobility across Gram-positive and Gram-negative bacteria, including *E. Coli*, is facilitated by conjugative plasmids and transposons. ErmB not only renders macrolide therapy ineffective but also contributes to the co-selection of resistance when other antibiotics are used, making it a pivotal player in the resistance network (Rossolini and Mantengoli, 2014). The ErmB gene, part of the erythromycin ribosomal methylase family, has been extensively studied in Gram-

positive organisms such as *Streptococcus pneumoniae* and *Staphylococcus aureus*. However, it is now increasingly reported in Enterobacteriaceae, particularly in *E. Coli* (Araujo *et al.*, 2019). ErmB acts by methylating the 23S rRNA, preventing macrolide binding and inhibiting protein synthesis (Rossolini *et al.*, 2014). Moreover, ErmB is often co-located with *tetA* (tetracycline resistance) and *sulI* (sulfonamide resistance) genes, raising concern about co-selection through agricultural and environmental exposures (Da Costa *et al.*, 2021). Methylation of the 23S rRNA prevents macrolide binding at the ribosomal A2058 site (Figure 8).

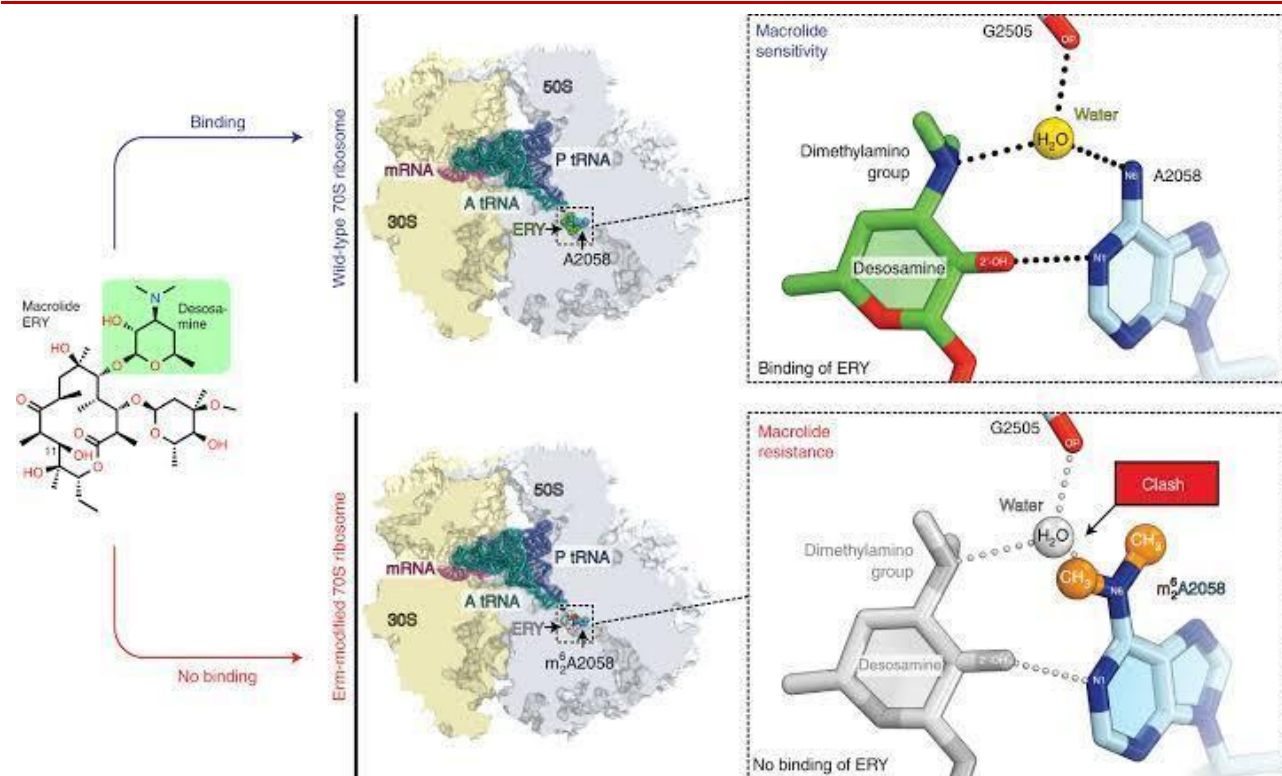


Figure 8: Mechanism of Action of ErmB. (Source: Svetlov *et al.*, 2021)

AAC(6')-Ib-cr gene

The AAC(6')-Ib-cr gene is a variant of the aminoglycoside acetyltransferase gene AAC(6')-Ib, which originally conferred resistance to aminoglycosides such as kanamycin, tobramycin, and amikacin by enzymatically modifying their structure. The -cr variant has gained an additional function, it can acetylate certain fluoroquinolones, particularly ciprofloxacin and norfloxacin, albeit with reduced efficiency. This dual activity makes AAC(6')-Ib-cr one of the few enzymes that mediates resistance to two major antibiotic classes, amplifying its clinical significance (Gomi *et al.*, 2013). AAC(6')-Ib-cr, a variant of the aminoglycoside acetyltransferase gene, has the unique ability to inactivate both aminoglycosides and certain fluoroquinolones through acetylation. Its dual-spectrum resistance is particularly concerning in environments where these drugs are first- or second-line therapies. AAC(6')-Ib-cr is often embedded in integrons and multidrug resistance plasmids, making it highly transmissible across enteric bacteria. Its detection in fecal *E. Coli* from asymptomatic individuals signals not only prior antibiotic exposure but also the potential for onward gene dissemination in clinical and environmental contexts (Wang and Zhao, 2013).

Perhaps the most insidious of the three genes under study, AAC(6')-Ib-cr is a variant of the aminoglycoside acetyltransferase enzyme that has acquired the ability to modify ciprofloxacin and norfloxacin, key members of the fluoroquinolone class. It achieves this through N-acetylation of the piperazinyl amine group on fluoroquinolones, reducing their antibacterial activity (Wang and Zhao, 2013). Its dual activity against aminoglycosides and fluoroquinolones makes it a critical component of multidrug-resistant profiles in both clinical and environmental isolates. AAC(6')-Ib-cr has been reported globally in fecal *E. Coli* from livestock workers, poultry handlers, hospital visitors, and even children in non-clinical settings (Chibuzor *et al.*, 2021; Iroha *et al.*, 2011). In many of these studies, the gene was located on IncFII plasmids, known for their high conjugation efficiency and ability to carry multiple resistance determinants (Nukui *et al.*, 2023). The prevalence of AAC(6')-Ib-cr is increasing globally. In Nigeria, India, and Southeast Asia, the gene has been detected in fecal *E. Coli* from humans, poultry, and environmental sources. Its frequent co-occurrence with ESBL genes such as blaCTX-M further complicates treatment strategies, especially in resource-limited settings where access to carbapenems or new-generation antibiotics is restricted. (Igbinosa *et al.*, 2022).

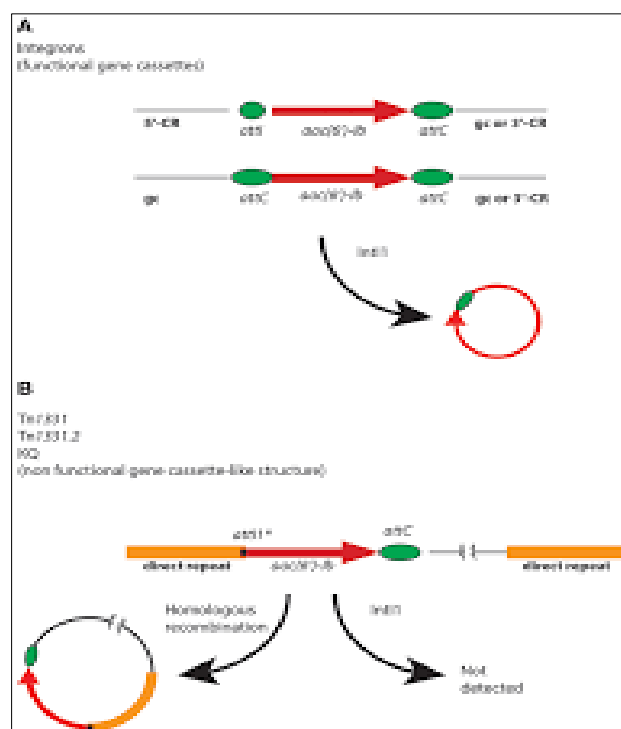


Figure 9: Integration of AAC(6')-Ib-cr within Class 1 Integron Cassette with other AMR genes
(Source: Ramirez *et al.*, 2013)

Molecular Techniques for Detection of Antimicrobial Resistance Genes

The global spread of antimicrobial resistance (AMR), particularly within the gastrointestinal microbiota, has necessitated a shift from purely phenotypic testing to molecular diagnostics capable of detecting resistance genes directly from nucleic acids. These molecular methods are critical in uncovering hidden reservoirs of resistance genes such as QnrA, ErmB, and AAC(6')-Ib-cr in asymptomatic human carriers, especially in fecal *Escherichia coli* (*E. Coli*) (Tramuta and Zucca, 2020; Jacoby *et al.*, 2006). Traditional disk diffusion or broth microdilution methods can identify resistance phenotypes but fail to detect silent or sub-inhibitory resistance genes unless expressed under selective pressure. In contrast, Polymerase Chain Reaction (PCR) and related nucleic acid amplification techniques provide precise identification of resistance genes, regardless of their phenotypic expression.

Conventional PCR

Conventional PCR remains the gold standard for detecting specific genes in bacterial isolates. Conventional PCR is typically performed in a thermal cycler using a master mix composed of DNA template, forward and reverse primers, dNTPs, magnesium chloride, Taq DNA polymerase, and reaction buffer. The cycling program includes an initial denaturation step, usually at 94–95°C, followed by 25–35 cycles of denaturation, annealing (typically 50–60°C), and extension (72°C). The amplified products are then

visualized using agarose gel electrophoresis stained with ethidium bromide or safe dyes like SYBR Green. The expected band size for each gene confirms its presence, while the absence of bands in negative controls confirms the validity of the result (Jacoby *et al.*, 2006). It uses gene-specific primers to amplify target sequences from extracted DNA.

Multiplex PCR

Multiplex PCR allows the simultaneous detection of multiple resistance genes in a single reaction tube by including multiple primer sets. This approach is particularly useful in AMR surveillance where multiple gene targets are under investigation. However, multiplexing requires rigorous optimization of primer concentrations, annealing temperatures, and reaction conditions to avoid non-specific amplification or primer-dimer formation. For QnrA, ErmB, and AAC(6')-Ib-cr, multiplex PCR offers a cost-effective and scalable solution for screening large sample sets, especially in resource-limited settings (Domokos *et al.*, (2016). Multiplex PCR also allows the simultaneous amplification of multiple resistance genes in one reaction, reducing time and cost. For QnrA, ErmB, and AAC(6')-Ib-cr, tri-primer sets can be designed with non-overlapping melting temperatures.

Real-Time PCR (qPCR)

Quantitative or real-time PCR offers the advantage of quantifying the gene copy number in real time using fluorescent dyes (e.g., SYBR Green) or sequence-specific probes (e.g., TaqMan). This method is

particularly useful when estimating the relative abundance of resistance genes in metagenomic samples or evaluating changes in gene expression under different conditions. qPCR is more sensitive than conventional PCR and allows for high-throughput analysis, albeit at a higher cost and requiring advanced instrumentation. In AMR research, qPCR data can be used to correlate gene abundance with antibiotic exposure or resistance phenotypes, providing deeper insights into the ecological dynamics of resistance (Livak and Schmittgen, 2001).

Gel Electrophoresis

Agarose gel electrophoresis is the standard technique used to visualize PCR products. After amplification, a portion of the reaction mixture is loaded into a pre-cast agarose gel stained with a nucleic acid dye. The gel is subjected to an electric current, which causes negatively charged DNA fragments to migrate toward the anode. Separation occurs based on size, with smaller fragments moving faster through the gel matrix. By comparing the migration of PCR products to a DNA ladder (molecular weight marker), the size of the amplified band can be determined (Sambrook and Russell, 2011). The presence of a band at the expected base pair length for each gene (e.g., ~516 bp for QnrA, ~639 bp for ErmB, ~482 bp for AAC(6')-Ib-cr) indicates successful amplification and gene presence. No bands in the negative control validate the absence of contamination, while positive controls confirm assay reliability. For publication or advanced analysis, gel images can be captured using UV transilluminators or gel documentation systems. In some settings, the PCR product can be further validated by restriction digestion or sequencing to confirm specificity (Korlach, 2017). After amplification, products are separated using 1.5–2.0% agarose gel electrophoresis, stained with ethidium bromide or GelRed, and visualized under UV light.

Sequencing Approaches

Sequencing remains the most accurate method. Sanger sequencing is commonly used to verify PCR amplicons, especially when assessing point mutations or differentiating gene variants. The PCR product is first purified using column-based or enzymatic cleanup methods and then subjected to a sequencing reaction using fluorescently labeled dideoxynucleotides. The resulting chromatograms are analyzed for sequence identity using software like Chromas or BioEdit, and aligned with reference sequences from NCBI's GenBank using BLAST. In high-resource laboratories, next-generation sequencing (NGS) technologies provide whole-genome or metagenomic sequencing data, allowing for the detection of all resistance genes in a sample without prior amplification. Platforms such as Illumina, Oxford Nanopore, and PacBio have revolutionized AMR genomics by enabling deep sequencing of microbial communities and the identification of novel or composite resistance elements. However, the cost, complexity, and data management

requirements limit the application of NGS in routine surveillance, particularly in low- and middle-income countries.

Whole Genome Sequencing (WGS)

The Whole Genome Sequencing provides complete resistance gene profiles. The technique detects mutations in quinolone resistance-determining regions (QRDRs). It links plasmid and chromosomal resistance elements. This method is useful for tracing clonal spread of resistant strains. It is used by NCDC and WHO GLASS partners in academic centers across Nigeria and South Africa (Weldhagen and Burns, 2019).

One Health Perspective: Bridging Human, Animal, and Environmental Health

The One Health framework recognizes that the health of humans is interconnected with the health of animals and the environment. Fecal *E. Coli* carrying QnrA, ErmB, and AAC(6')-Ib-cr exemplify this intersection. Carry resistant *E. Coli* in the gut from contaminated food, water, or previous antibiotic exposure. Livestock and poultry are often fed antibiotics as growth promoters or prophylactics, leading to resistance gene accumulation in their gut microbiota. Antibiotic residues and resistant bacteria enter the environment via fecal waste, contributing to environmental gene pools (Adenaya *et al.*, 2025). For example, a poultry farm using fluoroquinolones may select for *E. Coli* carrying both QnrA and AAC(6')-Ib-cr. These strains can contaminate poultry products or surfaces spread through handling, slaughter, or retail and finally ingested by humans, leading to intestinal colonization. Without coordinated One Health surveillance, interventions targeting only clinical settings will fail to interrupt this transmission cycle (Weldhagen and Burns 2019).

CONCLUSION

Detecting antibiotic resistance genes, such as QnrA, ErmB, and AAC(6')-Ib-cr, in fecal *E. Coli* requires precise and scalable molecular methods. Researchers use various molecular techniques to identify these resistance genes. Conventional PCR is a reliable method, but newer techniques like multiplex PCR and qPCR offer faster and more accurate results. Whole genome sequencing and metagenomics are the future of antimicrobial resistance research, providing valuable insights into how resistance spreads. Despite these advances, many communities lack access to these technologies. To effectively combat resistance, we need to invest in laboratory infrastructure, supply chains, and workforce development. This includes training skilled personnel and ensuring reliable access to essential reagents. National investments in molecular laboratories, standardized protocols, and skilled personnel are crucial to controlling the spread of resistance genes. By working together, we can protect public health and ensure that antibiotics remain effective for generations to come. As

researchers continue to develop new techniques and strategies, we edge closer to a future where antibiotic resistance is better managed. With persistence, collaboration, and investment, we can mitigate the impact of resistance and safeguard global health.

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