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Article

Antibiotic resistance of *Salmonella* species and *Escherichia coli* isolated from poultry farms in Kaduna State

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SUMMARY

Poultry farming provides a major source of animal protein and contributes significantly to economic development in sub-Saharan Africa. However, the intensification of production has created environments that favour the persistence and spread of zoonotic foodborne pathogens, notably *Salmonella* spp. and *Escherichia coli*. The widespread, often non-therapeutic, use of antimicrobials for growth promotion and disease prevention in poultry has accelerated the selection and dissemination of antimicrobial resistance (AMR). This review synthesises current evidence on the prevalence, transmission dynamics, public health implications, and molecular resistance mechanisms of *Salmonella* and *E. coli* in poultry environments, with a focus on findings from Kaduna State, Nigeria. Recent surveillance data indicate high isolation rates from cloacal swabs, litter, and personnel skin samples, with isolates showing marked resistance to critically important antimicrobials, including gentamicin, ciprofloxacin, third-generation cephalosporins (ceftriaxone), and carbapenems (imipenem). The review underscores the urgent need for integrated One Health approaches to curb the spread of AMR in livestock systems.

Keywords: *Salmonella*, *Escherichia coli*, Antimicrobial Resistance, One Health, Poultry Biosecurity

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INTRODUCTION

Over the past decades, global poultry production has grown rapidly, driven by population growth, urbanization, and rising consumer demand for affordable animal protein [1]. This expansion has brought considerable economic benefits but has also generated large volumes of waste, particularly poultry litter, which, while useful as organic fertilizer, can harbour a range of chemical and biological hazards [2,3]. Fresh litter often contains pathogenic bacteria, fungi, viruses, helminths, heavy metals, pesticide residues, and growth-promoting hormones [4].

The use of veterinary pharmaceuticals has transformed poultry disease management. While viral and fungal infections are primarily controlled through vaccination, bacterial diseases are typically managed with antimicrobial agents [5,6]. Yet, despite these interventions, infectious diseases remain a major obstacle to productivity and animal welfare [1].

Among the many bacteria present in poultry environments, *Salmonella* spp. and *E. coli* are particularly significant as indicators of hygiene and biosecurity, and as major causes of foodborne illness [7]. In developing countries, including Nigeria, Bangladesh and Cameroon, inadequate management of these pathogens in farm and processing settings has led to frequent outbreaks of zoonotic infections [7,8,9]. Both organisms are members of the *Enterobacteriaceae* family—Gram-negative, rod-shaped, facultative anaerobes that colonize the gastrointestinal tracts of humans and animals and serve as reservoirs for AMR genes [10,11].

Transmission to humans occurs mainly through the food chain: consumption of undercooked poultry meat, raw eggs, unpasteurized milk, or cross-contamination during handling [12]. The European Food Safety Authority consistently reports poultry products as primary vehicles for human enteric infections [13]. In Nigeria, *Salmonella* infections—ranging from enteric fevers (typhoid and paratyphoid) to gastroenteritis—are endemic across all geopolitical zones [14]. Non-typhoidal *Salmonella* (NTS) strains, which have a wide host range and can be shed asymptomatically by animals, circulate persistently, especially where agricultural sanitation is suboptimal.

Globally, NTS causes an estimated 93.8 million illnesses and 155,000 deaths each year [15]. Meanwhile, pathogenic *E. coli* strains present a dual burden: they threaten human health through food contamination and cause significant economic losses in poultry production through colibacillosis, a disease characterized by pericarditis, perihepatitis, airsacculitis, and septicaemia [16,17,18,19]. The routine use of mass antimicrobial treatments to control colibacillosis has intensified selection pressure, leading to the emergence of multidrug-resistant *E. coli* lineages.

Similarly, *Salmonella* can persist in the avian caecum without causing clinical signs, allowing infected birds to carry high bacterial loads into processing plants [20]. The progressive loss of efficacy of first line and critically important antibiotics is complicating clinical management of human infections [21]. Moreover, the misuse of antimicrobials in livestock leads to drug residues in food and promotes horizontal gene transfer (HGT) in environmental settings [22]. The interconnection between animal, human and environmental health makes AMR in poultry production a clear One Health priority [23].

This review collates and critically appraises the available literature on AMR in *Salmonella* and *E. coli* from poultry farms, emphasizing local data from Kaduna State, and identifies gaps that warrant further research.

Poultry Production: Biological, Economic and Public Health Context

Biology and Sectorial Importance

Domesticated poultry—principally chickens (*Gallus gallus domesticus*), turkeys, ducks and geese are raised globally for meat, eggs, feathers and manure [24]. Modern commercial breeds have been selected for rapid growth and high feed conversion: broilers typically reach market weight within 5–7 weeks, while layers can produce over 300 eggs per year under optimal management [25]. This adaptability to intensive confinement has allowed poultry farming to transition from small scale backyard operations to highly automated industrial systems.

Poultry products now supply over 36% of the world's meat consumption. Chicken is favoured for its low cost, versatility and broad cultural acceptability [26]. Eggs provide highly bio-available protein, vitamins and essential fatty acids, making them crucial for nutrition in developing regions. For smallholder farmers, poultry serves as a liquid asset that can be sold quickly, and because women and children are often involved in flock management, the sector contributes to poverty reduction and gender equity [27].

However, the intensification of production carries public health risks. High-density housing can facilitate the transmission of zoonoses such as highly pathogenic avian influenza, salmonellosis and *campylobacteriosis*, particularly where biosecurity is weak or live-bird markets are unregulated [28]. Sub-therapeutic antimicrobial use for growth promotion and prophylaxis further accelerates AMR development [29]. Surveillance in north-west Nigeria has confirmed that *Salmonella* is highly prevalent; for instance, Jibril *et al.* [30] reported a farm-level prevalence of 47.9% in Sokoto State, with 23 serotypes identified, dominated by *Salmonella Kentucky* (32.9%) and *Salmonella Isangi* (11.0%). These findings underscore the role of poultry as persistent reservoirs for pathogenic *Enterobacteriaceae*.

Poultry housing and production systems

Poultry farms vary from small backyard flocks to large integrated complexes. Housing systems range from climate-controlled broiler sheds and multi-tier layer cages to open side houses with mechanical ventilation, automated feeding and specialized bedding [31]. Production models include conventional intensive, free-range and organic systems, each with different bird densities, outdoor access and veterinary inputs [32]. Diets are formulated to meet specific nutritional requirements across growth stages, typically based on corn, soybean meals, vitamins, minerals and growth-promoting additives [33]. Effective biosecurity perimeter fencing, vehicle sanitation, footbaths, routine diagnostic screening and vaccination is essential to prevent disease introduction [34]. Waste management, including composting or deep-pit storage, is critical to limit environmental pollution and pathogen dispersal [35]. Economic viability depends on balancing regulatory compliance, animal welfare, feed costs and market prices [36].

Biosecurity shortcomings and contamination pathways

Inadequate biosecurity remains a major contributor to bacterial contamination on

poultry farms. Key operational deficiencies include:

- Lax perimeter controls allow vehicles, visitors and wildlife to introduce pathogens [37].
- Overcrowding, which elevates stress hormones, suppresses immunity and facilitates bird-to-bird transmission [38].
- Insufficient cleaning and disinfection of housing, feeders and water lines between cycles, allowing pathogens to persist [37].
- Contaminated feed and water, especially when feed is improperly stored or water is drawn from untreated sources [38].
- Poor waste handling, with uncomposted manure near sheds acting as a continuous source of reinfection [35].
- Inadequate vector control (rodents, flies, wild birds) that enables mechanical and biological transfer of pathogens [37].
- Lack of occupational hygiene—absence of handwashing facilities, dedicated footwear or protective clothing—which can lead to human-animal pathogen exchange [38].
- Insufficient disease surveillance that delays detection and containment of outbreaks [37].
- Improper vaccination protocols that leave flocks vulnerable to preventable infections [34].

Contamination can enter via multiple routes: contaminated feed and water, asymptomatic carriers shedding pathogens in faeces and respiratory secretions, environmental reservoirs (litter, dust, soil), fomites (boots, tools, crates, vehicle tyres), biological vectors, wildlife contact and airborne bioaerosols [37,38].

Health consequences

Bacterial contamination affects animal health, human food safety, occupational safety and the wider environment. Pathogens like *Salmonella*, *Campylobacter*, pathogenic *E. coli* and *Clostridium perfringens* cause enteritis, septicaemia, poor feed conversion and mortality [35,38,39]. Human consumers face gastroenteritis and systemic infections, particularly from undercooked or cross-contaminated poultry products. Farm workers are at risk of skin and gastrointestinal infections through direct contact [39]. Environmental dispersal of resistant bacteria via manure runoff can contaminate soil, surface water and groundwater [35].

Major Enteric Pathogens in Poultry

Escherichia coli

E. coli is a Gram-negative, facultatively anaerobic, rod-shaped bacterium that colonises the gastrointestinal tract of warm-blooded animals [40]. Most strains are commensal, but certain pathotypes carry virulence factors that cause disease in humans and animals [41]. Some lineages, such as Shiga toxin-producing *E. coli* (STEC) including O157:H7, are important foodborne pathogens that can asymptomatically colonise livestock and contaminate meat during processing [31,42].

Pathogenic *E. coli* is categorised by virulence traits and clinical outcomes:

- Enterotoxigenic *E. coli* (ETEC) produces heat-labile (LT) and heat-

stable (ST) enterotoxins that disrupt intestinal fluid balance, causing secretory diarrhoea in humans and young livestock [40].

- Enteropathogenic *E. coli* (EPEC) forms attaching-and-effacing (A/E) lesions on intestinal epithelia and is a major cause of infantile diarrhoea in developing countries [42].
- Enterohemorrhagic *E. coli* (EHEC/STEC) carries phage-encoded Shiga toxins (stx_1, stx_2) that inhibit protein synthesis in host cells, leading to haemorrhagic colitis and haemolytic uraemic syndrome (HUS), characterized by renal failure and microangiopathic anaemia [43].
- Enteroaggregative *E. coli* (EAEC) exhibits a "stacked-brick" adherence pattern and produces enterotoxins and cytotoxins that cause persistent diarrhoea in children and travellers.
- Enteroinvasive *E. coli* (EIEC) invades colonic epithelial cells and multiplies intracellularly, causing inflammatory dysentery, akin to *Shigella*.
- Diffusely adherent *E. coli* (DAEC) adheres uniformly to intestinal cells and is associated with diarrhoea in young children [40].

Transmission is faecal-oral via contaminated water, undercooked meat, unpasteurized dairy, or direct contact with infected animals or environments. Clinical manifestations range from mild, self-limiting diarrhoea to severe systemic disease. Prevention relies on biosecurity, slaughter hygiene, cold-chain maintenance and thorough cooking [41].

Salmonella species

Salmonella is a genus of zoonotic pathogens responsible for substantial foodborne illness worldwide, frequently linked to poultry products [44]. These Gram-negative, facultatively anaerobic rods survive in soil, water, processing equipment and animal intestines [45]. They can colonize the avian caecum without causing overt disease, allowing silent spread through markets and abattoirs [46]. Human salmonellosis is acquired through faecally contaminated food or water, or direct contact with animals [47]. Symptoms include nausea, vomiting, abdominal cramps, fever and diarrhoea. Although usually self-limiting in healthy adults, salmonellosis can progress to bacteraemia, osteomyelitis or meningitis, especially in infants, the elderly and immunocompromised individuals [40]. Control measures include farm sanitation, biosecurity, vaccination of breeders/layers and consumer food hygiene.

The genus is divided into two species: *Salmonella enterica* and *Salmonella bongori* [45]. *S. enterica* is further subdivided into six subspecies, with over 2,500 serovars defined by the Kauffmann-White scheme. Most zoonotic infections are caused by *S. enterica* subsp. *enterica* (subspecies I), including serovars Typhimurium, Enteritidis, Newport and Kentucky [32]. *S. bongori* is mainly associated with cold-blooded animals and rarely causes human disease [48].

Antimicrobial Resistance Mechanisms in Gram-Negative Enteric Pathogens

Antimicrobial resistance (AMR) arises through spontaneous chromosomal

mutations or acquisition of exogenous resistance genes via horizontal gene transfer (conjugation, transformation, transduction) [31]. Bacteria deploy four principal biochemical strategies: target modification, enzymatic inactivation, efflux pumping and reduced permeability [34]. AMR complicates clinical treatment, increasing morbidity, mortality and healthcare costs [49].

General mechanisms

Reduced outer membrane permeability

Gram-negative bacteria possess an asymmetrical outer membrane that restricts hydrophobic compounds. Hydrophilic antimicrobials (e.g., β-lactams, fluoroquinolones) must traverse porin channels. Mutations that down-regulate or alter porins reduce drug accumulation and contribute to resistance [50].

Active efflux

Multidrug efflux pumps (e.g., the RND-family AcrAB-TolC complex in *Enterobacteriaceae*) expel a broad range of antibiotics, including β-lactams, fluoroquinolones, tetracyclines and chloramphenicol, maintaining intracellular concentrations below lethal thresholds [51].

Enzymatic inactivation

Bacteria produce enzymes that modify or destroy antimicrobials. β-lactamases hydrolyze the β-lactam ring of penicillins and cephalosporins. The emergence of extended-spectrum β-lactamases (ESBLs) and carbapenemases can render even advanced therapeutics ineffective [52].

Target modification

Mutations in genes encoding drug targets reduce binding affinity. For instance, point mutations in the quinolone resistance-determining regions (QRDR) of *gyrA*, *gyrB*, *parC* or *parE* alter DNA gyrase and topoisomerase IV, preventing fluoroquinolones binding. Likewise, changes in penicillin-binding proteins (PBPs) reduce β-lactam susceptibility.

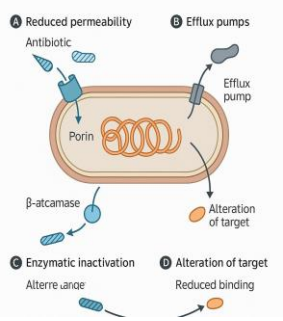

Figure 1: Diagrammatic Summary of Antibiotic Resistance Mechanisms [21].

Specific resistance determinants in *Enterobacteriaceae*

E. coli and *Salmonella* readily acquire mobile genetic elements (plasmids, transposons, integrons) that carry multiple resistance genes. Key mechanisms include:

- **ESBLs:** Plasmid-borne enzymes (CTX-M, TEM, SHV families) that hydrolyze third- and fourth-generation cephalosporins and monobactams but are generally inhibited by clavulanate. ESBL-producing strains often show resistance to ceftriaxone [53].
- **Carbapenemases:** Highly versatile β-lactamases such as KPC, NDM and OXA-48-like enzymes that degrade carbapenems—last-line antibiotics in human medicine. These are often carried on conjugative plasmids alongside

other resistance genes, giving rise to MDR or XDR phenotypes [54].

- **AmpC β-lactamases:** Cephalosporinases that confer resistance to cephamycins (e.g., cefoxitin) and broad-spectrum cephalosporins and are resistant to conventional β-lactamase inhibitors. AmpC can be chromosome- or plasmid-mediated.
- **Plasmid-mediated colistin resistance (*mcr*):** The *mcr* genes encode phosphoethanolamine transferases that modify lipid A, reducing colistin binding. This horizontally transferable resistance threatens colistin, a last-resort drug for carbapenem-resistant infections [55].

Genotypic Architecture of Resistance Determinants

The phenotypic resistance observed in poultry-derived *Salmonella* and *E. coli* isolates is mediated by a variety of established resistance genes, summarized in Table 1

.Table 1: Major Antibiotic Resistance Genes and Characterized Mechanisms

Antimicrobial Class	Primary Genes	Specific Biochemical Mechanism	Molecular/Epidemiological Context
B-lactams / Cephalosporins	blaTEM, blaSHV, blaCTX-M	Hydrolysis of the cyclic amide bond within beta-lactam rings.	Widespread on conjugative plasmids; blaCTX-M is highly prevalent in industrial farming worldwide.
Carbapenems	blaNDM, blaKPC, blaOXA-48	Metallo- and serine-protease cleavage of carbapenem molecules.	Last-line clinical indicators; presence in livestock marks a critical public health escalation.
Fluoroquinolones	GyrA, GyrB, qnrA, qnrB, qnrS	Chromosomal target site modification (QRDR) and plasmid-mediated target protection.	Chromosomal mutations drive high-level resistance; *qnr* factors promote low-level resistance and spread easily.
Tetracyclines	tetA, tetB, tetM, tet O	Active efflux pump synthesis and ribosomal protection protein production.	Highly stable in agricultural soils and litter due to long-term use of tetracyclines in livestock.
Aminoglycosides	aac(3)-II, aac(6')-Ib, aadA, aph}	Chemical modification via acetylation, adenylylation, or phosphorylation.	Frequently located on class 1 integrons alongside sulfonamide resistance markers.
Sulfonamides / Trimethoprim	sul1, sul2, sul3, dfrA	Production of alternative bypass enzymes (DHPS and DHFR) with low drug affinity.	Stable environmental markers; frequently linked to class 1 integron structures in animal-derived E. coli.
Phenicols	catA, cmlA, floR	Enzymatic drug acetylation and specialized inner	floR mediates co-resistance to florfenicol (veterinary) and chloramphenicol (human clinical).

		membrane efflux pumping.	
Polymyxins (Colistin)	mcr-1, mcr-2, mcr-5	Phosphoethanolamine modification of Lipid A to reduce negative surface charge.	Discovered globally across poultry export networks; represents a critical threat to last-resort human therapies.

5. Emerging Carbapenemase Concerns in Agriculture

The detection of imipenem resistance and the carbapenemase gene *bla*$_{NDM}$ in poultry-derived *E. coli* is alarming, as carbapenems are not approved for veterinary use. Their presence in livestock environments likely reflects introduction through human-animal contact or co-selection via other antimicrobials used in agriculture [56]. Since *bla*$_{NDM}$ is often carried on highly conjugative plasmids, it can spread to other enteric bacteria within the animal gut and the environment [54]. This raises the possibility that last-line resistance genes may return to humans through the food chain or environmental exposure. Such findings mirror global trends of *mcr* and carbapenemase genes in agricultural settings, underscoring the challenge of managing AMR across the One Health continuum [34].

Mitigation Strategies and Future Directions

Policy and regulatory actions

To curb AMR in poultry production, a multipronged approach is required:

1. Restrict over-the-counter access to critically important antimicrobials, mandating veterinary prescriptions.
2. Phase out sub-therapeutic use of medically important antibiotics for growth promotion or mass prophylaxis, in line with WHO recommendations [29].
3. Improve farm biosecurity through training and support for vector control, composting, clean water supply and occupational hygiene [43].
4. Establish integrated One Health surveillance that monitors AMR trends in humans, livestock and the environment, enabling early warning and evidence-based interventions.

Alternatives to conventional antibiotics

To reduce reliance on antibiotics without compromising productivity, the industry should explore:

- Vaccination: Targeted vaccines against *Salmonella Enteritidis* and pathogenic *Escherichia coli* can lower disease incidence and antibiotic use [40].
- Probiotics and competitive exclusion: Beneficial bacteria, prebiotics and organic acids support gut health and prevent pathogen colonization [57].
- Bacteriophage therapy: Host-specific phages can selectively eliminate multi-resistant

pathogens without disrupting the normal microbiota [5,58].

CONCLUSIONS

Antimicrobial resistance in *Salmonella* and *Escherichia coli* from poultry farms poses a significant threat to animal health, food safety and human medicine. Local data from Kaduna State reveal high prevalence and resistance to critically important drugs, including carbapenems. Addressing this challenge requires coordinated policy, biosecurity improvements, alternative therapies and robust surveillance within a One Health framework. Future research should focus on the molecular epidemiology of resistance genes, their transmission pathways, and the effectiveness of intervention strategies in the Nigerian context.

Authorship Contributions

Concept: Usman, M.U.; Design: Usman, M.U., Egbe, N.E., Sani, A.M. OaiKhen, E.E.; Data Collection/Processing: Usman, M.U.; Analysis/Interpretation: Usman, M.U.; Literature Search: All authors; Writing: Usman, M.U.; Review & Editing: All authors; Supervision: Egbe, N.E., Sani, A.M, OaiKhen, E.E.

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