Tackling the Threat: A Review of Fluoroquinolone Resistance in Salmonella and Campylobacter in Food-Producing Animals

Review Article

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Abstract

This paper explores the serious public health threat posed by fluoroquinolone resistance in *Salmonella* and *Campylobacter* bacteria, which are major causes of foodborne infections. The widespread use of fluoroquinolone antibiotics in food-producing animals has led to an increase in antimicrobial resistance (AMR), significantly limiting treatment options for human infections. This review examines the causes, prevalence, and transmission of fluoroquinolone resistance through a comprehensive literature review. Resistance is primarily driven by chromosomal mutations in genes like *gyrA* and *parC*, and the acquisition of plasmid-mediated resistance (PMQR) genes such as *qnr*. The prevalence of resistance is particularly high in poultry from countries like South Korea and China, with documented high rates of resistance to ciprofloxacin and nalidixic acid. Resistant bacteria are transmitted to humans through direct contact with animals and the food chain. The overuse of antibiotics in animal husbandry, horizontal gene transfer, and poor farm hygiene creates a selective pressure for resistance. The review highlights the need for more standardized and integrated surveillance programs to track resistant strains effectively. A "One Health" approach is crucial, involving stronger regulations, improved biosecurity, and the exploration of alternatives to antibiotics, such as vaccines and bacteriophages. This collaborative strategy is essential to safeguard public health against this global threat.

Keywords: Antimicrobial Resistance, Fluoroquinolone Resistance, Salmonella, Campylobacter, Food-producing Animals, One Health.

Introduction

An estimated 600 million illnesses and 420,000 deaths globally are attributed to foodborne infections, making them a major public health concern. The "One Health" approach emphasizes the idea that human well-being and environmental sustainability are inextricably related to the health of animals used to produce food (Signorini *et al.*, 2018). Animals that produce food are important reservoirs for a variety of foodborne infections, and the increasing demand for animal protein has resulted in intensive livestock production methods that often utilize a lot of antibiotics (Almansour *et al.*, 2023). Despite being intended to preserve animal health and production, this technique unintentionally aids in the development and global spread of antimicrobial resistance (AMR), which poses a serious risk to food safety worldwide (Njoga *et al.*, 2023). Salmonella and Campylobacter species are two of the most important bacterial infections linked to foodborne illnesses and are the main causes of gastrointestinal disorders in people worldwide (Hurd *et al.*, 2010). While Campylobacter infections are a major cause of diarrheal cases worldwide and have been the most reported gastrointestinal bacterial pathogen in humans in the European Union since 2005, non-typhoidal Salmonella (NTS) alone causes millions of cases and thousands of deaths annually (Bukari *et al.*, 2025). These illnesses are usually acquired by humans by the handling and ingestion of tainted food items, such as ground beef, eggs, and undercooked chicken meat. They can also spread through direct contact with polluted settings or diseased animals. It is commonly acknowledged that poultry is a major reservoir for Campylobacter and a major source of human transmission of the bacteria (Rozman *et al.*, 2018).

One extremely significant family of antimicrobials that is widely utilized in both human and veterinary medicine is fluoroquinolones. These medications are essential for treating bacterial infections, such as severe human Salmonella and Campylobacter infections. Since the 1990s, fluoroquinolones have been authorized for use in food-producing animals in the veterinary field. They are used to treat ailments such bacterial pneumonia, diarrhea, and bovine respiratory disease (BRD) (Abukhattab *et al.*, 2022). Fluoroquinolones are classified as "critically important antibiotics" for human medicine by the World Health Organization (WHO), emphasizing their vital importance in public health. Inhibiting bacterial DNA gyrase and topoisomerase IV, which are necessary enzymes for bacterial reproduction, is the main way they work (Grudlewska *et al.*, 2023).

The WHO has identified the emergence of AMR as one of the top ten hazards that demand immediate attention, making it a global health emergency. One of the main causes of the emergence and spread of resistant pathogens is the careless and frequently improper use of antibiotics in clinical and agricultural settings, including their use for therapeutic, prophylactic, metaphylactic, and growth-promoting purposes in animals raised for food. Because it significantly limits the effective treatment choices for potentially fatal human infections, resistance to vital medications like as fluoroquinolones is especially concerning (Lamichhane *et al.*, 2024). Following the use of fluoroquinolones in veterinary medicine, this resistance has been seen to develop in populations of Salmonella and Campylobacter, frequently concurrently in both people and animals. Numerous investigations have documented increased fluoroquinolone resistance in Salmonella and Campylobacter isolates from cattle, pigs, and poultry in various geographical areas. Importantly, a well-known and important route for the spread of AMR that directly endangers human health is the food chain, which transfers these resistant bacteria and their resistance genes from animals that produce food to people (Rozman *et al.*, 2019).

The goal of this review paper is to thoroughly investigate the current situation of fluoroquinolone resistance in Salmonella and Campylobacter in animals that produce food. It will examine these infections' molecular epidemiology, transmission dynamics, prevalence, and underlying resistance mechanisms. With the goal of improving our knowledge and guiding successful interventions for responsible antimicrobial use in animal agriculture and protecting public health, this paper will also address current and prospective strategies for reducing the emergence and spread of fluoroquinolone resistance. As seen in Figure 1, a fundamental principle of the "One Health" concept is the interdependence of environmental, animal, and human health in addressing global health issues.

Figure 1

*This figure illustrates the **"One Health"** approach, showing the interconnectedness of human, animal, and environmental health. It highlights that addressing health challenges requires a collaborative effort across these three areas.*

Literature Review

Chromosome mutations and the acquisition of plasmid-mediated resistance (PMQR) determinants are the two main processes causing the complicated and diverse phenomena of fluoroquinolone resistance in bacteria. Different levels of resistance can result from these processes acting alone or in combination (Elbehiry *et al*., 2025).

Chromosomal Mutations

A key mechanism of fluoroquinolone resistance is chromosomal changes, which mostly impact on the bacterial enzymes that the medication targets or change the cell's capacity to store the drug. Mutations at several chromosomal locations are frequently acquired by bacteria that exhibit high levels of fluoroquinolone resistance.

Target Gene Mutations (GyrA, GyrB, ParC, ParE)

By directly preventing DNA synthesis, fluoroquinolones kill bacteria. They target DNA gyrase (topoisomerase II) and topoisomerase IV, two vital bacterial enzymes. According to Adeboye *et al*. (2020), these enzymes are essential for DNA replication, repair, and segregation. GyrA and GyrB subunits make up DNA gyrase, whereas ParC and ParE subunits make up topoisomerase IV. GyrA and ParC and GyrB and ParE have structural similarities. During the topoisomerization step, fluoroquinolones trap these enzymes on DNA, forming a physical barrier that stops DNA replication and eventually results in cell death (Costa *et al*., 2022). The main cause of resistance is spontaneous mutations in the genes that code for these enzyme subunits, which are more often found in areas referred to as the quinolone resistance-determining regions (QRDRs). According to Silva *et al*. (2015), these mutations decrease the enzymes' affinity for fluoroquinolones, which lowers the drug's efficacy. Point mutations in the gyrA gene, namely in its QRDR, mediate the primary fluoroquinolone resistance pathway in Campylobacter species. The Thr-86-Ile (T86I) substitution in GyrA is a frequent mutation. Reduced susceptibility and clinically significant levels of resistance result from this single point mutation in gyrA, which stops quinolones from binding (Doyle *et al*., 2015). Fluoroquinolone action and resistance in Campylobacter have not been linked to mutations in the gyrB, parC, and parE subunits, while these changes are known to confer resistance in other bacteria.

Mutations in gyrA and parC are commonly associated with fluoroquinolone resistance in Escherichia coli. Codon 83 alterations (like Ser83Leu) and codon 87 substitutions (like Asp87Asn, Asp87Tyr, Asp87Gly, and Asp87His) are

common gyrA mutations. According to Yavari *et al.* (2012), mutations in parC frequently happen at glutamate 84 (Glu84Lys) or serine 80 (Ser80Ile or Ser80Arg). Less often than gyrA and parC, mutations in gyrB and parE have also been linked to E. coli resistance. The most resistant E. coli isolates usually have at least three mutations in their target genes, demonstrating the multifactorial nature of high-level resistance (Torres *et al*., 2021).

Point mutations in the DNA gyrase gene are also frequently the cause of quinolone resistance in Salmonella species, with a single point mutation in gyrA frequently being associated with nalidixic acid resistance. This increases the possibility of a second mutation leading to fluoroquinolone resistance. Topoisomerase IV is the main target in Staphylococcus aureus, as evidenced by the fact that mutations in the grlA gene, which codes for the topoisomerase IV A subunit, are frequently detected before gyrA mutations (Kaur *et al.,* 2024). Higher levels of resistance are usually the result of a gradual process of resistance development, whereby initial changes in the primary enzyme target are followed by further mutations in the secondary target. As shown in Figure 2, fluoroquinolones function by blocking vital bacterial enzymes required for DNA replication, such as DNA gyrase and topoisomerase IV.

Figure 2

This diagram explains how fluoroquinolones work. The drugs inhibit essential bacterial enzymes, specifically DNA gyrase and topoisomerase IV, which are necessary for DNA replication. This inhibition ultimately leads to bacterium cell death.
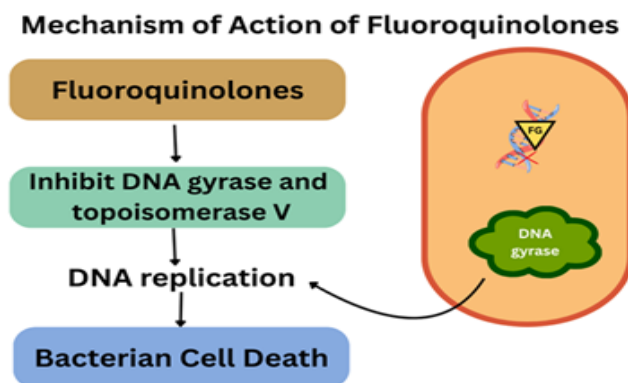
Methods and Materials

Plasmid-Mediated Resistance (PMQR)

Plasmid-mediated quinolone resistance (PMQR) is a process in which bacteria develop fluoroquinolone resistance not only through chromosomal changes but also through mobile genetic elements (MGEs) like plasmids. PMQR mechanisms are important because they contribute to multidrug resistance by being easily spread by horizontal gene transfer (HGT) across many bacterial strains and throughout various environments, including the food chain (Wiśniewski, *et al*., 2024).

Transmembrane protein systems called efflux pumps aggressively move antimicrobial substances, such as fluoroquinolones, out of bacterial cells to lower intracellular concentrations and impart resistance. These systems are commonly associated with multidrug resistance (MDR) characteristics because they may extrude a large spectrum of structurally varied substances. A functionally described multidrug efflux mechanism that contributes to antibiotic resistance in Campylobacter is the CmeABC efflux pump (Founou *et al*., 2018). Insertion sequences (IS) can stimulate its expression and provide resistance to fluoroquinolones and macrolides. One variation that has been identified as a "super efflux pump" that contributes to MDR in Campylobacter is RE-CmeABC. Multidrug resistance in Salmonella and Escherichia coli is largely caused by efflux pump systems such as AcrAB-TolC (Habib *et al*., 2021). It has been demonstrated that deletions in genes encoding elements of these efflux pumps, such as acrAB and tolC, greatly enhance sensitivity to fluoroquinolones. Increased efflux and resistance result from the overexpression of these chromosomally encoded pumps, which are frequently controlled by chromosomal alterations (e.g., in marR).

The significance of other efflux pumps in fluoroquinolone resistance, such as NorA in Staphylococcus aureus, is also well-established. Overexpression of these pumps can be a one-step mechanism of resistance since they can transport fluoroquinolones and other unrelated chemicals out of the cell. Mobile genetic elements often contain genes for efflux pumps, which promote their horizontal transfer and broader distribution (Anderson *et al.*, 2001). Plasmid-mediated quinolone resistance (PMQR) determinants, known as Qnr proteins, shield the bacterial DNA gyrase and topoisomerase IV enzymes from fluoroquinolone damage. Qnr proteins raise the minimum inhibitory concentration (MIC) of fluoroquinolones and nalidixic acid by binding to these enzymes and preventing fluoroquinolones from interfering with DNA replication (Pal *et al.*, 2024). Numerous Qnr variations, such as qnrA, qnrB, qnrS, qnrC, and qnrD, have been discovered. Resistance to several antimicrobial classes, including β-lactams, can be co-transmitted when these genes, which are usually found on transferable plasmids, coexist with other resistance genes.

Salmonella spp. and E. coli isolates from animals and food items have been shown to include the qnrB (e.g., qnrB2, qnrB19) and qnrS (e.g., qnrS1) genes (Dekker et al., 2019). For instance, it was discovered that the qnrB2 and qnrB19 genes were present in Salmonella Kentucky-ST314 and Poona-ST308 isolates from chicken in Ghana, respectively. The effectiveness of fluoroquinolone therapies in both people and animals is seriously threatened by the spread of these Qnr-harbouring plasmids. The common aminoglycoside acetyltransferase AAC (6')-Ib that imparts PMQR has a variation called the AAC (6')-Ib-cr enzyme. This enzyme is special because, in addition to its main role of inactivating aminoglycosides, it may acetylate and so decrease the action of certain fluoroquinolones that include a piperazinyl group, such as ciprofloxacin and norfloxacin (Abreu *et al.*, 2023). Usually found on plasmids, the gene encoding AAC (6')-Ib-cr permits horizontal transmission between bacterial species. In places like Singapore, its presence has been found in a variety of Enterobacteriaceae, such as E. coli and Klebsiella spp. This enzyme's discovery emphasizes several molecular pathways that lead to the development of fluoroquinolone resistance in harmful bacteria in animals that produce food and the environment at large (Teklemariam *et al.*, 2023).

Epidemiology and Prevalence of Resistance

The World Health Organization (WHO) has identified the rising incidence of antimicrobial resistance (AMR), especially to fluoroquinolones (FQs), in Salmonella and Campylobacter as one of the top ten dangers that demand immediate attention (Lekshmi *et al.*, 2023). This is a serious global health problem. These organisms are one of the main causes of foodborne illnesses globally, and the effective treatment choices for serious human infections are severely limited by their resistance to FQs. Since FQs were first used in veterinary medicine, it has frequently been noted that Campylobacter and Salmonella populations have developed FQ resistance simultaneously in both people and animals. In the fight against AMR, this emphasizes the vital necessity of a "One Health" strategy that recognizes the interdependence of environmental, animal, and human health (Espunyes *et al.*, 2021). One of the main causes of its appearance and dissemination is the careless and improper use of antibiotics for medicinal, preventive, metaphylactic, and growth-promoting purposes in animals raised for food.

Results and Findings

Poultry

Poultry, including chickens and ducks, are widely recognized as major reservoirs for *Salmonella* species and a significant source of their transmission to humans. Studies in various regions highlight high prevalence and concerning resistance patterns (Qamar, *et al.*, 2023). In South Korea, national monitoring data from 2023 show *Salmonella jejuni* isolates from duck feces (81%), chicken carcasses (78.3%), and duck carcasses (65.5%) have the highest resistance rates to (fluoro)quinolones, specifically ciprofloxacin and nalidixic acid (Bedekelabou, *et al.*, 2021). Another study found high ciprofloxacin resistance (79.2%) and tetracycline resistance (68.5%) in *Campylobacter* isolates from retail chicken meat, with 45.3% exhibiting multidrug resistance (MDR). From ducks in South Korea, ciprofloxacin resistance was 91.4% in *C. jejuni* and 100% in *C. coli*, with 91.4% of *C. jejuni* and all *C. coli* strains showing MDR (Ortega-Sanz, *et al.*, 2025).

In China, a study in Shandong province in 2009 and 2012 found that *Salmonella* isolates from chickens, ducks, and pigs were overwhelmingly resistant to at least one antibiotic (>99%). In 2012, resistance to nalidixic acid was

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particularly high (95.9%), followed by sulphafurazole (78.2%) and ampicillin (72.3%) (Kurcubic, *et al.,* 2025). Ciprofloxacin resistance was detected in 41.5% of isolates. *Salmonella Enteritidis* was the most common serotype in chickens, exhibiting 100% resistance to nalidixic acid in 2009 and 2012. While no *S. Enteritidis* was resistant to ciprofloxacin in 2009, this increased to 16.9% by 2012. *Salmonella Indiana* isolates, commonly found in 7-week-old chickens and 6-week-old ducks, showed severe MDR, with all 58 isolates resistant to at least 10 antibiotics, including ciprofloxacin (Smith, *et al.*, 2017). In Ghana, *Salmonella* was found in 9% of poultry meat samples. Fluoroquinolone resistance was high, with 63% of *Salmonella* isolates resistant to ciprofloxacin and 100% resistant to tetracycline. Specific serovars like *Salmonella Kentucky* (ST198) showed a high ciprofloxacin MIC (16 µg/mL) with double mutations in *gyrA* and *parC*, while *Salmonella Poona* (ST308) carried transferable *qnrB2* PMQR genes (Kunadu, *et al.*, 2020). Across Southeast Asian countries, resistance to traditional antibiotics like ampicillin, tetracycline, and sulfonamides was high in *Salmonella* isolates from poultry and other food animals. Nalidixic acid resistance ranged from 9% to 36% in *Salmonella* from food animals, indicating its extensive use. Ciprofloxacin resistance was generally low (1–7%) in most Southeast Asian countries for food animal isolates (Garvey, *et al.*, 2022).

Swine

Pigs are also significant reservoirs for *Salmonella*. In Shandong, China, *Salmonella Derby* (58%) and *Typhimurium* (18%) were the most common serovars in pig samples. *Salmonella* from pigs in 2009 showed common resistance to tetracycline (78.7%), sulphonamides (76.6%), streptomycin (42.6%), and chloramphenicol (36.2%) (Rabello *et al.*, 2020). All nine *Salmonella Typhimurium* isolates from pigs were resistant to tetracycline, ampicillin, amoxicillin/clavulanic acid, florfenicol, kanamycin, sulphafurazole, and trimethoprim/sulphamethoxazole, with seven also resistant to chloramphenicol, streptomycin, and gentamicin (Barata *et al.*, 2024). In a study of pigs exposed to enrofloxacin, fluoroquinolone resistance emerged in the native *Campylobacter coli* population. In Ethiopia, the prevalence of *Salmonella* in pigs was reported at 43.81%. The widespread use of antibiotics in the swine industry, with 9,000 kg of fluoroquinolones used annually in Korea, contributes to the emergence of resistant bacteria (Divsalar *et al.*, 2019).

Cattle

Salmonella species are frequently isolated from cattle feces, hides, and lymph nodes. However, studies on FQ resistance in *Salmonella* from cattle, especially beef cattle, have often shown remarkably low prevalence, even in herds where FQs like enrofloxacin are used (Kunadu, *et al.*, 2020). For instance, a study of *Salmonella* in feedlot cattle in the US found that nearly all isolates (379 out of 380) were susceptible to both nalidixic acid and ciprofloxacin, with only one isolate resistant to nalidixic acid. This was consistent with previous research demonstrating extremely low prevalence of quinolone-resistant *Salmonella* in feedlot cattle (Black, *et al.*, 2018). Furthermore, the number of enrofloxacin treatments for bovine respiratory disease (BRD) was not significantly associated with the prevalence or quinolone susceptibility of *Salmonella*. The 2014 NARMS Integrated Report also indicated that ciprofloxacin resistance remained low (<10%) for all *Salmonella* isolates tested from cattle. Despite this, some MDR *Salmonella* serotypes, such as *S. Dublin*, *S. Reading*, and *S. Typhimurium*, have been isolated from cattle and ground beef. *Salmonella Cerro* has also been associated with human salmonellosis from beef and dairy cattle, though its resistance is mainly attributed to non-fluoroquinolone drugs (Ohta *et al.*, 2018). Overall, *Salmonella* fecal prevalence in pre-harvest cattle can be highly variable among feedlots. In contrast to *Salmonella*, *Campylobacter* isolates from cattle often show a high prevalence of quinolone resistance. The reason for this difference in resistance between the two pathogens in cattle is the subject of ongoing investigation.

Discussion

Fluoroquinolone resistance in *Salmonella* and *Campylobacter* in food-producing animals presents a significant public health challenge, largely due to their complex transmission dynamics and severe public health implications. These pathogens, which are leading causes of foodborne diseases globally, are primarily transmitted to humans through the farm-to-fork pathway (Dargatz *et al.*, 2016).

On the farm, food-producing animals such as poultry, cattle, and swine serve as significant reservoirs for *Campylobacter* and *Salmonella*, often asymptomatically. The widespread and sometimes inappropriate use of

antimicrobials, including fluoroquinolones, in animal production for therapy, prophylaxis, or as growth promoters exert selective pressure that fosters the emergence and proliferation of resistant strains. These resistant bacteria and their antimicrobial resistance genes (ARGs) can then move from farm animals to humans through several routes (Benedict *et al*., 2011). Direct contact with animals or their biological substances, such as feces, by veterinarians, farmers, abattoir workers, and food handlers, constitutes one pathway. More broadly, indirect transmission occurs via the food chain when humans consume contaminated food products like meat, eggs, or milk. The environment also plays a crucial role, as ARGs and resistant bacteria can be excreted in animal waste, contaminating soil, manure, and water, which can then further spread to crops, other animals, and eventually to humans. For instance, fluoroquinolone-resistant *Campylobacter jejuni* from poultry meat has been directly correlated with human clinical isolates (Webb *et al.*, 2016).

The human health burden from fluoroquinolone-resistant *Salmonella* and *Campylobacter* infections is considerable. While most infections are self-limiting, severe cases, especially in vulnerable populations such as the young, elderly, or immunocompromised, necessitate antimicrobial therapy. Resistance to critically important antibiotics like fluoroquinolones can lead to treatment failure, requiring the use of more expensive, potentially toxic, or "last resort" drugs, and in some instances, making infections untreatable (Sproston *et al*., 2018). This is compounded by the fact that resistance to first-generation quinolones like nalidixic acid, observed at high rates in regions like Shandong, China (up to 95.9%) and Southeast Asia (19–36%), increases the risk of developing resistance to more advanced fluoroquinolones. The emergence of extended-spectrum cephalosporinase-producing isolates resistant to both fluoroquinolones and third-generation cephalosporins is particularly alarming, directly leading to failed salmonellosis treatments (Abraham *et al*., 2020). Quantifying this impact, one study estimated the mean annual increased risk in the US population (adults over 18) for persistent symptoms due to compromised fluoroquinolone treatment to be 1 in 61 billion for *Salmonella*, 1 in 33 billion for MDR *Salmonella*, and 1 in 2.8 billion for *Campylobacter*. Globally, AMR infections are projected to cause 10 million deaths annually by 2050, with trillions in economic costs. High levels of fluoroquinolone resistance, for example, 75% in *Campylobacter* from Ghanaian poultry, highlight a significant reservoir for human infections (Isada *et al*., 2022). Furthermore, international trade plays a significant role in the global spread of resistance. The extensive commercial exchange of animal-derived food products means that resistant strains emerging in one country can rapidly disseminate across continents. Developing nations, often major exporters of food animals and products, contribute to this spread, as do international travelers who can acquire resistant bacteria and become vectors upon returning to their home countries. This global movement of animals and animal products, therefore, bypasses geographical borders, challenging local and national control efforts and emphasizing the critical need for a coordinated "One Health" approach to surveillance and intervention (Ruiz-Palacios *et al*., 2007). Pathogens like *Salmonella* are transmitted to humans primarily through the food chain, direct contact with animals, and contaminated water, as depicted in Figure 3.

Figure 3
This graphic depicts the primary ways that pathogens like Salmonella are transmitted to humans. The pathways include the food chain (contaminated food and water), direct contact with infected animals, and the consumption of contaminated food.
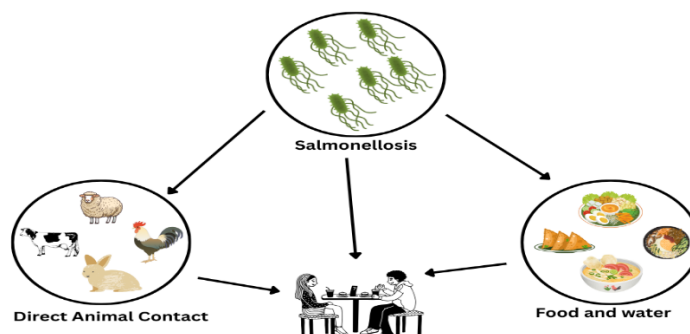
Factors Contributing to Resistance Development

The development of fluoroquinolone resistance in *Salmonella* and *Campylobacter* is a complex issue influenced by several interconnected factors, primarily rooted in the overuse and misuse of fluoroquinolones in veterinary medicine, facilitated by horizontal gene transfer, and exacerbated by biosecurity and management practices on farms. The overuse and misuse of fluoroquinolones in veterinary medicine exert significant selective pressure for the emergence of resistant strains. Fluoroquinolones, such as ciprofloxacin and enrofloxacin, are broad-spectrum antimicrobials used to treat bacterial diseases in food-producing animals like poultry, cattle, and pigs (Angulo *et al*., 2005). They are employed for various purposes, including therapy, prophylaxis (disease prevention), and as growth promoters. The widespread and sometimes inappropriate application of these critically important antimicrobials in food production has been directly linked to the rise of resistance. For example, the reduced susceptibility to ciprofloxacin detected in *Salmonella* can be associated with its overuse or the use of enrofloxacin in food-producing animals. In some regions, such as intensive farms in China, enrofloxacin has been widely used. Globally, up to 80% of antibiotics produced are used in food-producing animals, with a projected 67% increase in antibiotic use in animals by 2030, particularly in countries like China, Brazil, and India (Fernández *et al*., 2016). China, for instance, is the largest consumer of antibiotics in animal husbandry, with 52% administered for growth promotion. This widespread use, often in sub-therapeutic doses as growth promoters or in prophylactic mass treatments, creates an environment where resistant bacteria are selected and thrive. Furthermore, in many developing nations, the use of antimicrobials in animal farming is largely unregulated, and there is a lack of control over the distribution of veterinary pharmaceuticals, contributing to misuse and the perpetuation of high resistance levels. The indiscriminate use, including off-label drug use and a failure to adhere to withdrawal periods, significantly exacerbates the problem.

Horizontal gene transfer (HGT) plays a critical role in the rapid dissemination of antimicrobial resistance genes (ARGs) among *Campylobacter* and *Salmonella* species (Price *et al*., 2007). This process allows bacteria to acquire and spread resistance determinants through various mobile genetic elements (MGEs), such as plasmids, integrons, transposons, and insertion sequences. Plasmids are extrachromosomal DNA molecules that can replicate independently and carry ARGs, readily transferable between different bacterial species and even across diverse settings. For instance, plasmid-mediated quinolone resistance (PMQR) has emerged as a significant public health risk in *Salmonella enterica*, as these plasmid-borne resistance genes can be horizontally transferred among strains and are often co-transmitted with other resistance markers, leading to multidrug resistance (Xia *et al*., 2019). Examples include the *qnr* genes (e.g., *qnrA, qnrB, qnrS*) that increase resistance to nalidixic acid and fluoroquinolones. The *cfr(C)* gene, which confers cross-resistance to five antimicrobial classes classified as 'Critically Important Antimicrobials' by the WHO, can also be spread through horizontal transfer, often located on plasmids or associated with multidrug-resistance genomic islands. Additionally, the VirB-Type IV Secretion System (VirB-T4SS) plasmid in *Campylobacter* can carry and spread co-harbored multidrug resistance genes. Integrons are genetic elements capable of capturing and expressing gene cassettes that often contain ARGs, contributing to lateral genetic transfer via transposons and conjugative plasmids (Vinayamohan *et al*., 2022). Class 1 integrons are widely distributed among *Salmonella* strains, with identical AMR mobile elements frequently found in *Salmonella* isolates from animals and humans, suggesting a role for raw foods in disseminating resistant bacteria and genes through the food chain. Transformation (uptake of naked DNA) and transduction (DNA transfer via bacteriophages) are other mechanisms of HGT that contribute to the spread of resistance determinants in *Salmonella* and *Campylobacter*. The constant selective pressure from antibiotic use in animal agriculture significantly boosts these genetic exchange processes (Oladeinde *et al*., 2021).

Poor biosecurity and management practices on farms create ideal conditions for the emergence and widespread dissemination of resistant bacteria. Factors such as the cleanliness of floors and facilities are crucial in controlling the prevalence of *Campylobacter*. Inadequate hygiene and management can exacerbate the problem by facilitating the persistence and spread of pathogens. Effective pre-harvest interventions, including robust biosecurity measures, pest control, and rigorous cleaning and disinfection protocols, are essential to prevent the colonization of poultry and other livestock by pathogens like *Salmonella* and *Campylobacter* (Guernier-Cambert *et al.,* 2021). When animals are housed in proximity, as in intensive livestock systems, horizontal transmission of pathogens occurs rapidly. Moreover, bidirectional transmission between humans and animals, coupled with widespread antimicrobial use, creates continuous antibiotic pressure, facilitating the increase and spread of multidrug-resistant *Campylobacter*. In many

low- and middle-income countries, poor hygienic measures and farming practices are prevalent in intensive production systems. Implementing good agricultural practice (GAP) and good hygiene practice (GHP) is fundamental to reducing the risk of infectious diseases at the farm level and preventing foodborne transmission along the entire farm-to-fork pathway. These measures, along with a comprehensive "One Health" approach encompassing human, animal, and environmental health, are vital for surveillance and control of antimicrobial resistance (Lima *et al.*, 2020).

The complex cycle of antimicrobial resistance, driven by factors like antimicrobial usage in food animals, results in the transmission of resistant bacteria and genes to humans through various pathways, including the food chain and the environment, as shown in Figure 4.

Figure 4

This illustration shows the complex cycle of antimicrobial resistance. It starts with the use of antimicrobials in food-producing animals, which leads to the spread of resistant bacteria and genes to humans through pathways like the food chain, contaminated environments, and manure.
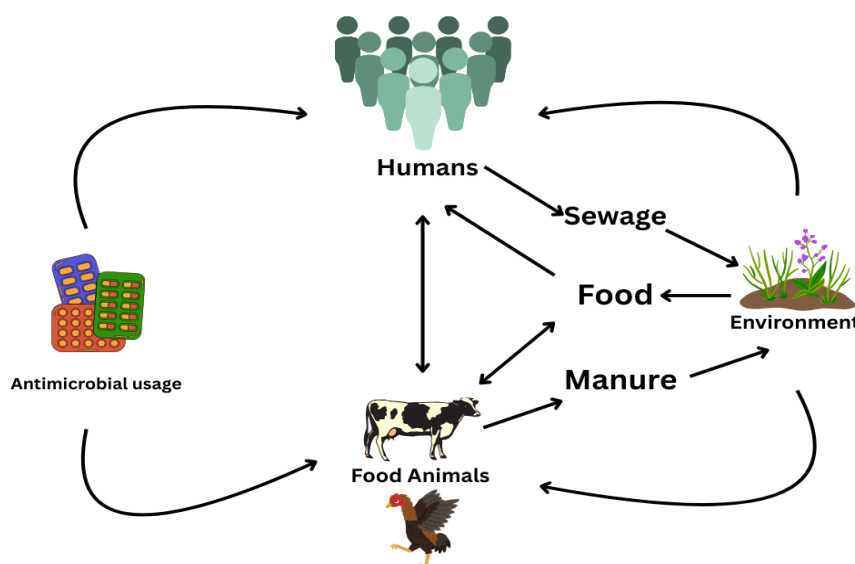
Strategies for Tackling the Threat

Tackling the threat of fluoroquinolone resistance requires a multifaceted approach involving robust regulatory and policy measures, effective farm-level interventions, and widespread public awareness and education. Regulatory and Policy Measures are critical for controlling the emergence and spread of resistance. Restriction of Fluoroquinolone Use is paramount; policies such as making fluoroquinolones available only by veterinary prescription or under veterinary oversight are essential to ensure prudent use (Matheou *et al.*, 2025). The use of "last line" human antibiotics in food production should be avoided to prevent resistance. Some countries, like Australia, have never approved broad-spectrum antimicrobials like fluoroquinolones for food-producing animals, while the EU has forbidden their use as growth promoters since 2006. Stricter antibiotic use policies and enhanced surveillance are urgently needed to mitigate the growing threat of antimicrobial resistance (AMR) in *Campylobacter* (Elbehiry *et al.*, 2025). Governments must enforce the registration and monitoring of veterinary drug use and promote good practices by trained professionals. Surveillance and Monitoring Programs are indispensable, necessitating integrated food chain surveillance systems that conduct long-term epidemiological and molecular studies from farm-to-fork, adhering to a "One Health" approach and international guidelines. National reference laboratories are crucial for providing harmonized, high-quality data on AMR bacteria. Such programs, like the Danish Integrated Antimicrobial Resistance Monitoring and Research

Programme (DANMAP), have demonstrated success in reducing antibiotic consumption and resistance by monitoring the impact of interventions (Founou *et al*., 2016).

Farm-Level Interventions directly address the reservoirs and transmission pathways of resistant bacteria. Improved Biosecurity and Hygiene are fundamental, encompassing practical measures such as better sanitation, including the cleanliness of floors and facilities on farms, which play a crucial role in controlling *Campylobacter* prevalence (Manyi-Loh *et al*., 2018). Effective pre-harvest strategies, like robust biosecurity, pest control (including rodents), and rigorous cleaning and disinfection protocols, are vital to prevent colonization of livestock by pathogens. Implementing "all-in/all-out" management systems significantly reduces *Campylobacter* colonization in poultry flocks. Vaccination programs offer a promising alternative to antibiotics. Preventing infectious diseases through immunization can significantly reduce the need for antibiotic use in food-producing animals (Ye *et al*., 2025). For instance, studies have shown that vaccination against *Salmonella* can induce immunity against pathogenic species in poultry without adverse effects, thereby reducing antibiotic dependence. Alternative Therapies are also being explored, with promising options like bacteriophages, probiotics, and prebiotics. Bacteriophages, for example, specifically infect and kill bacteria, and phage cocktails have successfully reduced *Campylobacter* loads in broiler chickens and *Salmonella* in poultry and pigs. Probiotics can enhance the gastrointestinal microbial environment, helping to out-compete pathogenic microorganisms (Ali *et al*., 2023).

Finally, Public Awareness and Education are vital for fostering responsible antimicrobial use across all stakeholders. Educating farmers, veterinarians, food handlers, and the public on the judicious use of antimicrobials is paramount. Public awareness campaigns should highlight the importance of proper food hygiene practices such as adequate cooking temperatures and food storage to reduce the spread of resistant bacteria (Dhaka *et al*., 2023). Veterinarians need reinforced education on conservative prescribing practices and the One Health approach. Addressing the knowledge gap among farm workers through targeted interventions is also necessary to improve hygiene practices and biosecurity on farms. Ultimately, consistent dissemination of knowledge and behavioral change campaigns are essential to lessen antibiotic consumption and AMR rates.

Conclusion and Future Perspective

Fluoroquinolone resistance in *Salmonella* and *Campylobacter* in food-producing animals is a significant public health crisis driven by the overuse of antimicrobials. This review has highlighted that resistance is widespread, particularly in poultry, and is mediated by both chromosomal mutations, such as those in the *gyrA* gene, and plasmid-mediated resistance genes like *qnr*. This resistance poses a direct threat to human health, as these pathogens are transmitted through the food chain, leading to treatment failures and the need for more costly or toxic "last resort" drugs. Moving forward, addressing this issue requires a concerted effort to fill critical research gaps. There is a need for more standardized and integrated surveillance programs across the food chain, from farm to fork, to better track the emergence and spread of resistant strains. Furthermore, research should focus on developing rapid diagnostic tools to guide judicious antibiotic use and exploring non-antibiotic treatments, such as vaccines, bacteriophages, and probiotics, to reduce reliance on antimicrobials. Ultimately, tackling this global threat requires a coordinated "One Health" approach that recognizes the interconnectedness of human, animal, and environmental health, fostering collaboration between all sectors to implement effective interventions and safeguard public well-being.

Declarations

Ethical Approval and Consent to Participate: This study strictly adhered to the Declaration of Helsinki and relevant national and institutional ethical guidelines. Informed consent was not required, as secondary data available on websites was obtained for analysis. All procedures performed in this study were by the ethical standards of the Helsinki Declaration.

Consent for Publication: The authors give their consent for publication.

Availability of Data and Materials: Data will be made available upon request from the corresponding author.

Competing Interest: The authors confirm that there is no clash of interest/ conflict.

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