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Antimicrobial Resistance in *Escherichia coli*

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A B S T R A C T

Escherichia coli multidrug resistance is becoming an alarming worldwide issue in veterinary well-being in addition to human medicine. Almost all to treat essential antimicrobial drugs can naturally create *Escherichia coli* to become aware. However, this bacterial species can rapidly acquire resistance genes, primarily through the horizontal transfer of genes. The most problematic mechanisms in E. coli correspond to the acquisition of genes coding for extended spectrum β-lactamases, carbapenemases, 16S rRNA methylases, plasmid-mediated quinolone resistance (PMQR) genes, and mobilized colistin resistance (MCR) genes. Although the spread of carbapenemase genes has been mainly recognized in the human sector but poorly recognized in animals, colistin resistance in E. coli seems rather to be related to the use of colistin in veterinary medicine on a global scale. Though genetic studies show that the extended-spectrum β-lactamase producers found in animals are different from those that afflict humans, the cross-transfer of these resistance traits between the human and animal sectors is still a source of contention Resistance genes appeared to spread mostly by plasmids, especially multi - resistance plasmids, but also through other movable elements in gene including gene cassettes and transposons in class 1 and class 2 integrous.

Introduction

Escherichia coli (*E. coli*) are a majorly common aerobic bacterium found in the gastrointestinal microbiota of vertebrates, playing a crucial role in maintaining gut health and function. While *E. coli* is primarily recognized as a communal organism - beneficially coexisting within the host without causing harm - certain pathogenic strains pose serious health risks to both humans and animals (1,2). These pathogenic variants are particularly notorious for causing human gastroenteritis, characterized by severe diarrhea, abdominal pain, and other gastrointestinal symptoms. What makes *E. coli* especially noteworthy in microbiology is its dual role: it contributes significantly to the microbial community that supports host digestion and immune function, while also possessing the capability to induce serious infections under specific conditions (3,4). This duality highlights the complexity of *E. coli* as a microorganism and underscores the need for careful monitoring of its strains.

A pressing concern is the potential transmission of pathogenic or antibiotic-resistant *E. coli* from animals to

humans. This can occur through multiple pathways, including direct contact with contaminated animal waste, consumption of undercooked or contaminated food, and even environmental exposure (5,6). Such transmission dynamics raise alarms about public health, especially given the close interconnection between agricultural practices and human health.

In addition to its pathogenic potential, *E. coli* serves as a significant reservoir for antibiotic resistance genes, which can lead to treatment failures in both veterinary and human medicine (7,8). Over recent decades, researchers have identified an increasing prevalence of resistant gene sequences in *E. coli* isolates, with a substantial proportion acquired through horizontal gene transfer. This process enables *E. coli* to both assimilate resistance genes from other bacterial species and disseminate these genes to neighboring bacteria, amplifying the spread of resistance within the microbial community.

Thus, the issue of antibiotic resistance in *E. coli* is a formidable world health challenge that influences both animals and human populations. The complexity of its interactions with other bacteria, coupled with its ability to adapt and share resistance mechanisms, complicates efforts to manage and control its spread. This chapter aims to provide a comprehensive overview of the current state of antibiotic resistance in *E. coli* isolates derived from animals, focusing on specific classes of antimicrobial agents commonly used in veterinary practice (9,10). By addressing these issues, we can better understand and mitigate the risks associated with *E. coli* in our interconnected ecosystems.

E. coli as a pathogenic and commensal bacterium

The pathogenic bacteria *E. coli*, which typically inhabits the lower intestines in the majority of warm-blooded mammals, is the cause of a disease known widely as "colibacillosis." So, *E. coli* is a multipurpose microbe that may cause both intestinal and extraintestinal infections due to its pathogenic isolates, while the majority of its other isolates are safe to their hosts and are known to exhibit commensalism. Pathotypes, also known as pathovars, are groups of toxic *E. coli* isolates that are associated with distinct diseases (11). *E. coli* pathovars that are intestinally pathogenic cause disorders that range from slight constipation to severe colitis. On the other hand, additional-intestinally transmissible *E. coli* pathovars, mainly asymptomatic intestinal residents that trigger extra-intestinal diseases when they migrate to other regions of the physique, like the bloodstream or urinary system. *E. coli* isolates from the ambient reservoir or from other sick people can also induce animal diseases associated with the coli bacteria. The development or deletion of virulence-associated features linked to *E. coli* pathogenicity distinguishes hazardous and not hazardous *E. coli*. The *E. coli* genome has between 4,000 and 5,000 genes. Of these, about 3,000 genes are shared by the various isolates, whereas the remaining genes primarily relate to factors involved in colonization or virulence (12,13). Whole-genome sequencing has allowed for deeper understanding of the genomic flexibility of *Escherichia coli* and has improved our knowledge of accessory, core genetic makeup of both harmful and mutualistic isolates of the bacteria. Animals can become infected with *E. coli* even as adults; infections are not limited to young animals. Companion animal infections in the lower and higher urinary tract are primarily caused by extraintestinal pathogenic *E. coli*, as was previously mentioned (14, 15). A bird's inhalation of feces can trigger avian-pathogenic *E. coli*, which then spreads throughout the bird's body to produce pericarditis, septicemia, and death from colibacillosis.

Alongside *Staphylococcus aureus, Streptococcus uberis, Streptococcus agalactiae*, and *Streptococcus dysgalactiae*, *E. coli* is one of the main causes of mastitis in cattle livestock, which is a usual swelling response of the mammary gland that dramatically reduces milk manufacturing and causes dramatic profitable loss.

Over 80% of cases of acute mastitis, in which lipopolysaccharide (LPS) is the predominant virulence factor and induces severe clinical symptoms, are specifically caused by *E. coli*. This is followed by a discharge of inflammatory mediators. However, it is generally accepted that *E. coli*-caused mammary in dairy cattle is not linked to particular *E. coli* variants nor does it entail a shared set of virulence indicators across *E. coli* isolates (16,17). Antimicrobial compounds used as growth promoters in animals have been prohibited in Europe since 2006, however most countries continue accept this practice. National measures have also been implemented, primarily in Europe, to limit the use of these essential antimicrobial agents in animals. Multidrug-resistant *Escherichia coli*, including those that produce ESBL/AmpC, has emerged as a primary indicator for estimating the impact of resistance to antibiotics in livestock and other sectors from a One

Health perspective, as antimicrobial drugs have a significant effect on the microbiota of the gut where *Escherichia coli* lies.

Ability to withstand fluoroquinolones and quinolones

A valuable class of antimicrobial agents for the treatment of a variety of infections in humans and animals are quinolones and fluoroquinolones. Their ability to kill almost all bacteria is well established. Genes encoding DNA the gyrase and topoisomerase IV are the drug targets that typically cause rebellion to these antimicrobial agents (18,19). However, other methods, such as decreased outer tissue penetrable, target structure protection, or unregulated deluge pumps, may also be involved. In *Escherichia coli*, the gyrase synthesized from two GyrA and two GyrB subunits is the principal target of (fluoro) quinolones. One of Gram-negative bacteria's secondary targets is topoisomerase IV. Pairs of ParC and ParE subunits make up this enzyme. In the quinolone based resistance-determining domain of GyrA, which is located between Ala67 and Gln107, the majority of mutations were discovered, with codons 83 and 87 seeing the most prevalent mutations. GyrA mutations alone may confer quinolone resistance, but additional gyrA and/or parC mutations are required to achieve fluoroquinolone resistance (20). The use of molecular epidemiologic techniques to these challenges is covered in this review, along with how the resolution of these issues could advance our knowledge of the epidemiology of *E. coli* infections. This piece is a selection from a carefully picked set. Cotrimoxazole, a medication, is widely used in conjunction with trimethoprim and sulfamethoxazole. Since cotrimoxazole is cheap and usually effective, it has been used to treat a variety of *S. pneumoniae* infections, particularly in children. Cotrimoxazole resistance has skyrocketed in many parts of the world; rates have been linked to HIV infection in Africa and Asia, with rates varying from 19% in Europe to over 50% in recent surveillance studies.

Resistance to penicillin in particular is frequently linked to resistance to cotrimoxazole. A significant category of broad-spectrum antibacterial medicines, fluoroquinolones exhibit analogous spectra of activity to structural changes in nalidixic acid, the first quinolone. Regarded as the pioneer of quinolones, nalidixic acid was first used in clinical settings in 1962 to treat Gram-negative urinary tract infections in both humans and animals. In order to enhance the quinolones' antibacterial capabilities and pharmacokinetic characteristics, and molecular structures were subsequently altered. Quinolone medications are categorized into generations mostly based on their antibacterial spectra. Fluoroquinolones, which were produced by fluoridating quinolone protein at position C6, were the precursors of the subsequent generation of quinolones. Norfloxacin, the first fluoroquinolone, was created in 1978 and approved for use in medicine in 1986. One of the most popular fluoroquinolones, ciprofloxacin, entered the pharmaceutical market in 1987. Fluoroquinolone medications exhibit enhanced oral absorption and systemic distribution, and they are effective against a broad spectrum of Gram-negative and Gram-positive bacteria. As a result, these substances' clinical uses have expanded to include the treatment of urinary tract infections, infections of the skin and soft tissues, HIV-related illnesses, and lower respiratory tract infections.

Ability to withstand aminoglycosides

Aminoglycosides are naturally occurring medications whose producers belong to the species Streptomyces and Micromonospora. They are frequently utilized in conjunction with other antimicrobial (usually a β-lactam) to take advantage of their quick germ destroying in aiding complex contamination in humans and animals, including companion and food - providing animals, such as septicemia, pneumonia, meningitis, and urination tract/abdominal infections. In veterinary medicine, streptomycin derivatives and neomycin are the most commonly utilized compounds. Additionally, paromomycin, kanamycin, and gentamicin are utilized. Amikacin is only allowed to be used to treat infections in horses and dogs. Aminoglycosides interfere with translation in a wide range of Gram negative and Gram positive bacterial infections. These significant compounds may have limited therapeutic potential because of two major issues: the first is related to their toxicity (21). However, treatment regimens based on new insights into aminoglycoside pharmacodynamics are utilized to address this problem. The second problem is the widespread spread of bacterial resistance associated with the use of aminoglycosides.

A sketch of the process of resilience to aminoglycosides and its epidemiology in animal-origin *Escherichia coli* is given in the subsequent subsections. Neonatal meningitis, bloodstream infections, urinary tract infections, and sepsis are all brought on by ExPEC.

ExPEC and IPEC have consequently come to be considered too as hazardous varieties of *E. coli* and pathovars. ExPEC is more difficult to separate from ubiquitous *E. coli* due to differences in their virulence factors, which are responsible for the clinical symptoms they overlap. ExPEC may be commensal *E. coli* that break through a sterile barrier to cause extraintestinal infections, although IPEC are likely to have reservoirs outside of the human intestine (12). The materialization of WGS (High-throughput DNA sequencing) has complicated this matter further by posing new queries regarding the taxonomy description of *E. coli* using phylogenetic and conventional clinical microbiological techniques. Aminoglycosides have been utilized as broad-spectrum antibiotics to cure the illnesses brought on by a number of Gram negative and Gram positive bacteria. As suggested by their name, aminoglycosides are composed of amino sugar molecules connected by ligand sugar bonds and an amino alcohol ring. The bacteria's 30S and 50S ribosomal subunit rRNA, for instance, is one of the negatively-charged molecules that those positively-charged amino groupings can interact with to impede protein synthesis and ultimately cause cell death. A new era in aminoglycoside research began in 1943 with the discovery and use of streptomycin, a drug tailored to treating tuberculosis, from Streptomyces griseus.

Trimethoprim and sulfonamides resistance

Synthetic antimicrobial drugs like trimethoprim and sulfonamides block distinct stages in the pathway that produces folic acid. While each of these substances has bacteriostatic properties, combining a sulfuric acid with the antimicrobial agent trim produces synergistic bactericidal effects on vulnerable organisms; this combination is known as “enhanced” sulfonamide. Trimethoprim and sulfonamides have been utilized in both humans and animals for many years. Sulfonamide resistance in *E. coli* from companion and food-producing animals is arbitrated by any one of the three sul genes (sul1, sul2, or sul3). Since the sul1 gene is a component of the class 1 integrons' 3′-conserved section, it is especially widely distributed (8). Because of this, the sul1 gene is frequently found on gene templates in the variable portion of class 1 integrons jointly with other resistant to antimicrobial genes. The following examples show that class 1 integrons are found in *E. coli* from companion animals, wildlife, and healthy and sick food-producing animals worldwide. Class 1 integrons were present in 58 of the 417 *E. coli* isolates from sick dogs, horses, pigs, and cats that were gathered for the BfT-GermVet monitoring research in Germany. Gram-negative bacteria such as Enterobacteriaceae have been shown to possess many dfr genes that provide trimethoprim resistance. They are classified into two main categories, dfrA and dfrB, according to their sizes and structural differences. While the proteins generated by the dfrB gene only have 78 amino acid residues long, the proteins encoded by the dfrA genes range in size from 152 to 189 amino acids each. On gene cassettes introduced into class 1 or class 2 integrons, the majority of dfrB, dfrA genes identified in *E. coli* with invertebrate origins were situated.

From a One Health perspective, resistance to antibiotics in *E. coli* is a critical issue because it affects both the human population and animal sectors. More significantly, multidrug susceptibility in *E. coli* represents a significant and common repository of factors that contribute to resistance to most classes of antimicrobial drugs across a wide range of organisms, including humans. The antibiotics used here have proven essential in the treatment of infectious diseases, extending the lives and enhancing the quality of life of patients. One of the biggest breakthroughs in medicine was their discovery. On the other hand, the abuse and misuse of antibiotics has ended in the development of antibiotic resistance (AMR), a phenomenon in which germs become resistant to the harmful effects of antibiotics. This means that multidrug resistance in animals can result in infections that are challenging to cure. Some evidence could argue for the function of the food chain, even though the various pathways by which immune *E. coli* isolated organisms from livestock to humans remain unclear and their respective significance cannot be determined. This is because the bacteria shown frequently colonize foods at retail in numerous nations and continents (10). Passive transmissions through surroundings or direct interactions with animals are two more possible modes of transmission. Resistance to antibiotics in *E. coli* in livestock has sparked a number of cross- sectoral and collaborative projects, including translational studies, the field of epidemiology and monitoring in both veterinary and human medicine, as *Escherichia coli* is a bacteria which is extensively distributed in both divisions. It is now believed that taking massive action is necessary to defeat the growing prevalence of antibiotic resistance in human-sourced *E. coli*. Antimicrobial stewardship (AMS) programs must be promoted and a One Health strategy must be adopted to address the issue of AMR because it poses a hazard to

public health that affects humans, animals, and the environment. Around 10 million people are predicted to die by 2050 along with at least 28 million will live in hardship as a result of this issue if it is not given priority and remedied. A joint "One Health" approach to combating AMR necessitates increased funding immediately. In light of all the recent research, it is probable that the presence of *E. coli* that produces carbapenemase in livestock doesn't pose a serious risk to human health, despite certain worrying and contemporary rumors to the contrary. On the other hand, researches showed that animals are important repositories of mediated - plasmid the drug colistin resistance genes, which are basically found in isolates of *E. coli* and provide higher risk to humans.

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Author Contributions

Jyotsna Joy: Investigation, formal analysis, writing—original draft. M. Shanmugavadivu: Validation, methodology, writing—reviewing.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

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