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Original Article

Evaluation of Antibiotic Resistance and Virulence Factors of *Escherichia coli* Strains Isolated from Cancer Patients

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Abstract

Background: Cancer patients are highly susceptible to infections due to immunosuppression. Extraintestinal pathogenic *E. coli* (ExPEC) often exhibit multidrug resistance (MDR) and harbour virulence factors that increase pathogenicity.

Objectives: This study aimed to evaluate the antibiotic resistance patterns and the prevalence of virulence genes (*fim*H, *tra*T, and *hly*A) in *E. coli* isolates from cancer patients.

Methods: The study included *E. coli* isolates (n = 50) collected from various clinical specimens of cancer patients, between September and December 2024 at Hiwa Hematology and Oncology Hospital, in Sulaymaniyah, Iraq. An antibiotic susceptibility test was conducted to assess the bacterial resistance profile, and Polymerase Chain Reaction (PCR) was used to identify virulence genes.

Results: Urinary tract infection due to *E. coli* was commonly observed among patients. MDR was identified in 72% and high levels of resistance were observed against β-lactam antibiotics, particularly ampicillin (88%) and ceftriaxone (72%). Fluoroquinolone resistance among isolates was notably high, especially to Ciprofloxacin and Levofloxacin (80% and 78%, respectively). Amikacin and Gentamicin were identified as suitable antibiotics for treating *E. coli* infection. Among the virulence genes, *fim*H was the most frequently detected (100%), then *tra*T (72%), and *hly*A (30 %). Phenotypically, only one isolate demonstrated hemolytic activity on blood agar, indicating its ability to lyse red blood cells. There was no significant relationship found between antibiotic resistance and the presence of virulence genes.

Conclusion: The results demonstrate that cancer patients are exposed to diverse strains of *E.coli* that possess both antimicrobial resistance and disease-causing potential, underscoring the importance of improving monitoring, prevention, and treatment strategies for *E. coli* infections within this vulnerable group.

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Introduction

Individuals with cancer are more prone to infections, mainly as a result of treatment, though some infections may also stem from the underlying malignancy. [1]. Infection risks for cancer patients also include traditional chemotherapy and radiation treatment. In addition to harming mucosal surfaces, these

treatments raise the risk of microbial colonization and the ensuing mucositis[2]. Feverish neutropenia, the initial and possibly only indication of infection in cancer patients, is a significant and dangerous side effect of cytotoxic chemotherapy [3]. *Pseudomonas aeruginosa*, *Klebsiella* species, and *Escherichia coli* account for the majority of recovered Gram-negative bacterial isolates [4]. A relatively new and diverse pathotype of *E. coli* is known as extraintestinal pathogenic *Escherichia coli* (ExPEC) [5]. Uropathogenic *E. coli* (UPEC), septicemic *E. coli* (SEPEC), and meningitis-related *E. coli* (MNEC) are types of ExPEC that cause infections in the urinary tract (UTI). Blood infections, often known as bacteremia, can affect other sections of the body. The development of antibiotic resistance, particularly since the late 1990s, has made treating infections brought on by ExPEC more difficult [6]. The widespread use of Antibiotics in Iraq has contributed to a significant increase in antimicrobial resistance [7]. Specific virulence genes, along with differences in their prevalence among strains, are responsible for variability in ExPEC disease severity and the multiple types of infections they can generate [8]. The broad range of virulence traits in ExPEC is mainly acquired through mobile genetic elements, including plasmids and pathogenicity islands, which encode functions related to iron uptake, capsule development, lipopolysaccharide production, invasive capacity, host cell attachment, and toxin activity [9, 10]. The process of specific adhesion involves various surface structures [11]. Type 1 fimbriae exhibit high affinity for mannosylated glycoproteins on the UTI epithelium and for the CD48 receptor on brain microvascular endothelial cells. This allows bacteria to invade, colonize, and multiply within host cells, forming biofilm-like intracellular bacterial communities which function as major determinants enabling attachment to and penetration of tissues outside the intestinal tract [12]. Toxin-encoding genes are key examples of virulence factors. Among them, hemolysin proteins (such as *hly*A) are closely associated with the pathogenicity of UPEC, functioning by creating holes in host cell membranes, leading to cell lysis. The synthesis, activation, and secretion of *E. coli* hemolysin (*hly*A) are regulated by the hlyCABD operon [13]. Another class of ExPEC virulence proteins called protectins contributes to serum resistance that enhances bacterial survival in the host [14]. By interfering with the proper assembly and membrane incorporation of the complement membrane attack complex, *tra*T enables bacterial survival in serum [15]. The infection process is significantly influenced by ExPEC's virulence and resistance to antibiotics. Virulence and resistance are closely related to the strain's evolutionary history. Phylogenetic group B2 and, to a lesser extent, group D comprise the majority of ExPEC isolates, while groups A and B1 comprise the majority of commensal isolates. Virulence factors were found at high frequency in isolates from group B2, while they were not found in high prevalence in strains from group A [6, 16]. Thus, we aimed to study the virulence genes (*tra*T, *fim*H, and *hly*A) and antibiotic resistance profiles of the ExPEC strain isolated from cancer patients to enhance clinical outcomes for the susceptible population by identifying potential targets for therapeutic intervention.

2 Materials and Methods

2.1 Sample Collection

A total of 50 pure ExPEC isolates were obtained from urine, blood, wound, and sputum samples of patients with concurrent cancer and infection at Hiwa Hematology/Oncology Hospital, Sulaimaniyah, Iraq, between September and December 2024

2.2 Identification

All samples were initially identified using the BD Phoenix system. For confirmation, additional identification methods were performed as the isolates were cultured on MacConkey and Eosin Methylene Blue agars and kept under incubation conditions of 37 °C for one full day to stimulate bacterial growth. Identification was performed using conventional methods. Afterwards, all samples were preserved and stored in 20% glycerol for further analysis.

2.3 Antimicrobial Susceptibility Testing

The susceptibility of isolates was evaluated using two complementary approaches: the standard Kirby–Bauer disk diffusion technique in accordance with Clinical and Laboratory Standards Institute (CLSI) recommendations, and the BD Phoenix automated system for bacterial identification and susceptibility assessment. For disk diffusion testing, two to three well-separated colonies were selected and emulsified in 5 mL of sterile saline to prepare a homogenous bacterial suspension. The density of the suspension was standardized to a 0.5 McFarland turbidity. This inoculum was uniformly spread across Mueller–Hinton agar plates, followed by placement of thirteen antibiotic disks.

The antibiotics tested comprised cefepime (10 µg), ceftriaxone (10 µg), levofloxacin (5 µg), ciprofloxacin (5 µg), meropenem (10 µg), imipenem (10 µg), amikacin (30 µg), gentamicin (10 µg), ampicillin (25 µg), amoxicillin–clavulanic acid (20/10 µg), trimethoprim–sulfamethoxazole (1.25/23.7 µg), tigecycline (15 µg), and nitrofurantoin (300 µg). After incubation at 37 °C for 16 to 18 hours, inhibition zones were measured and interpreted based on CLSI breakpoints. Susceptibility results were classified as susceptible, intermediate, or resistant. *Escherichia coli* ATCC 8739 was included as a quality control strain.

Isolates showing resistance to at least one agent in three or more antibiotic classes were MDR [17]. The BD Phoenix automated system was additionally employed to confirm resistance profiles and to evaluate susceptibility against an expanded antibiotic panel when required. For consistency between testing methodologies, only antimicrobial agents that were tested using disk diffusion were included in the final data presentation.

2.4 DNA Extraction

Genomic DNA was extracted from *ExPEC* strains using a commercially available purification kit (Invitrogen, Thermo Fisher). The purity and concentration of the isolated DNA were assessed using a Nanodrop spectrophotometer by measuring absorbance at 260 and 280 nm. Primer sequences, anticipated amplicon sizes, and corresponding annealing temperatures are summarized in **Table 1.**

2.5 Polymerase Chain Reaction (PCR)

PCR amplification was carried out on purified genomic DNA using gene-specific primers targeting *fim*H, *tra*T, and *hly*A. Each reaction mixture contained 12.5 µL of master mix (GeneSand Biotech, China), 0.7 µL of each forward and reverse primer, and 0.7 µL of DNA template, with nuclease-free water added to bring the total volume to 25 µL. The cycling conditions included an initial denaturation at

94 °C for 3 minutes, followed by 35 cycles of denaturation at 94 °C for 30 seconds, annealing at 60.2 °C for 30 seconds, and extension at 72 °C for 15 seconds, with a final extension step at 72 °C for 5 minutes. For amplification of the *hly*A gene, the annealing temperature was adjusted to 62.3 °C, and the extension time was increased to 36 seconds due to the larger amplicon size. PCR products were resolved and visualized using 1.5% agarose gel electrophoresis.

2.6 Ethical considerations

The study protocol received approval from the Institutional Review Board (IRB) of Hiwa Haematology/Oncology Hospital and the Ethics Committee of the College of Science, Charmo University, Sulaimaniyah, Iraq (Approval No. 4, dated August 4, 2024). All procedures were conducted in full compliance with the ethical principles outlined in the Declaration of Helsinki (2008).

2.7 Statistical Analysis

Data analysis was performed using GraphPad Prism (version 10). The relationship between virulence gene profiles and antibiotic resistance was assessed using Fisher's exact test, with p-values of ≤ 0.05 considered statistically significant

Table 1: Specific primers for the detection of virulence genes.

Primer Sequence (5' to 3')	Annealing Temp (°C)	Product Size (bp)	Reference
***fim*H**			
(F) CATTCGCCTGTAAAACCGCC	60.2	209	[39]
(R) ATAACACGCCGCCATAAGCC			
***tra*T**			
(F) ATAGTTCACATCTTCCACCATCG	60.2	239	-
(R) TCTCCGGATAAAGCCTACTACTG			
***hly*A**			
(F) AACAAGGATAAGCACTGTTCTGGCT	62	1146	[39]
(R) ACCATATAAGCGGTCATTCCCGTCA			

3 Results

3.1 Clinical specimens across diverse cancer types and microbial identifications.

As shown in Table 2, *E. coli* strains (n = 50) were collected from cancer patients (52% male and 48% female, with an average age of 54 years). Among collected samples, 58% were from urine, blood and wound (20% each), and 2% from sputum Regarding cancer types, hematological malignancies (leukemia, lymphoma, and myeloma) were common (42%), followed by urological cancers (bladder, kidney, prostate) (20%), gastrointestinal cancers (stomach, colon, gall bladder, rectum and liver (16%), breast cancer (6%), and other types of cancer (16%). Gram staining revealed Gram-negative, short rod-shaped bacteria appearing either singly or in pairs. Bacteria grown on MacConkey agar appeared rounded, non-mucoid, and bright pink (lactose fermenters). On the EMB agar, the strains appeared dark purple, and most of

them produced a green metallic sheen with a dark center.

Table 2: Distribution of sample types by gender among cancer patients.

Sample type	Male	Female	Total
	Frequency (Percentages)		
Urine	11 (38%)	18 (62%)	29 (58%)
Blood	10 (100%)	-------	10 (20%)
Wound	5 (50%)	5 (50%)	10 (20%)
Sputum	-------	1 (100%)	1 (2%)
Total	26 (52%)	24 (48%)	50 (100%)

3.2. Antimicrobial Susceptibility Test

Out of all *E. coli* isolates, 72% showed MDR, of which 24% were also carbapenemase producers. 56% were identified as ESBL producers, and Extensively drug-resistant (XDR) strains were 2%, while 26% of isolates were non-MDR. The highest resistance was to Ampicillin (88%), Ciprofloxacin (80%), Levofloxacin (78%), trimethoprim/ sulfamethoxazole (76%), Ceftriaxone (72%), Amoxicillin /Clavulanic acid (62%), and Cefepime (60%). Additionally, 98% of *E. coli* were sensitive to Tigecycline as shown in Figures 1, 2, and 3.

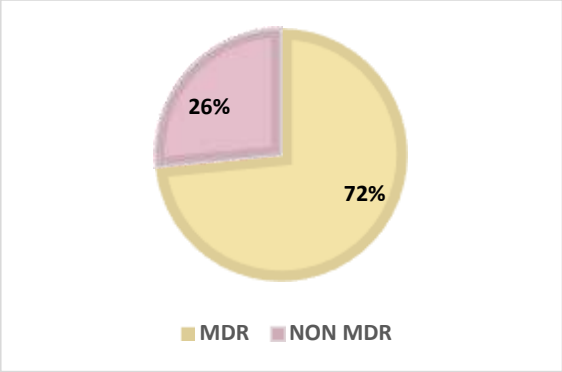

Figure 1: Proportion of MDR and non-MDR isolates.

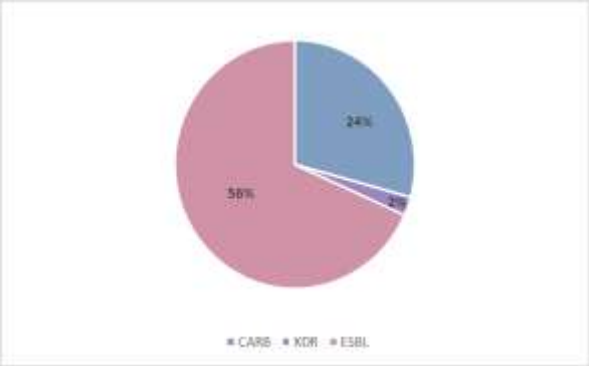

Figure 2: Proportional Distribution of carbapenemase production (CARB), extensively drug-resistant (XDR), and Extended-Spectrum Beta-Lactamase ESBL Within MDR *E. coli* Isolates

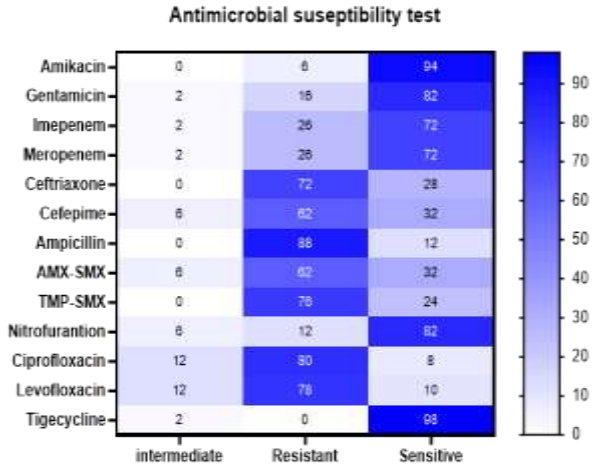

Figure 3: The percentage of susceptibility patterns for *E. coli* isolates against antibiotics.

3.3 Prevalence of Virulence Factor Genes

PCR amplification successfully detected the presence of the target virulence genes(*hly*A, *tra*T, and *fim*H) in varying proportions among the isolates. Adhesion, serum resistance, and toxin generation are often associated with these genes. *hly*A (30%), *tra*T (72%), and *fim*H (100%) were identified, in which 10 isolates carried all these genes concurrently, as shown in Table 3 and Figure 5.

Table 3: Frequency of Co-Occurrence of Virulence Genes in *E. coli* Strains.

Prevalence	Frequency (%)
*fim*H and *tra*T	36 (72)
*fim*H and *hly*A	15 (30)
*tra*T and *hly*A	10 (20)
*fim*H, *tra*T, and *hly*A	10 (20)

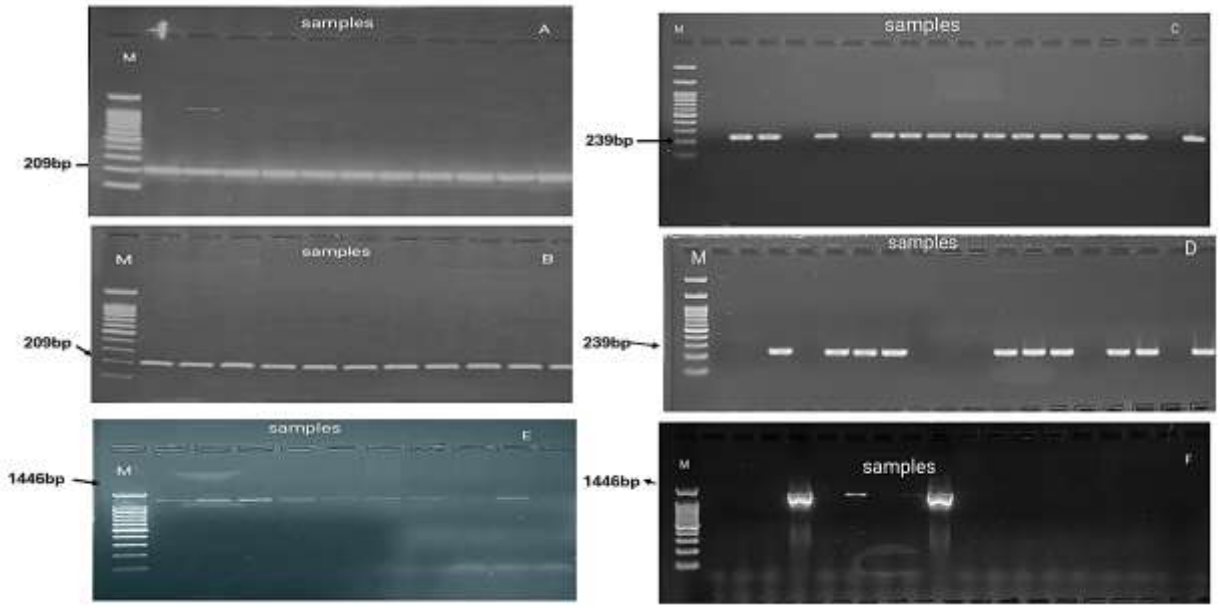

Figure 5: Agarose gel electrophoresis showing PCR amplification of *fim*H (A, B), *tra*T (C, D), and *hly* A(E, F) genes in *E. coli*, using a ladder of 100 bp.

3.4 sequencing

Seven *E. coli* isolates were selected for DNA sequencing. for the *fim*H, *tra*T, and *hly*A genes. The accession numbers obtained from BankIt for the sequenced virulence genes are presented in Table 4. Sequence analysis was performed by aligning the obtained PCR products with *E. coli* reference sequences available in the NCBI database. The *tra*T gene was compared with reference sequence NZ_JBBEZP010000120.1, the *hly*A gene with NZ_JABXCS010000026.1, and the *fim*H gene with NC_000913.3 using the NCBI BLAST alignment tool

Table 4: Summary of sequencing results of *fim*H, *tra*T, and *hly*A genes from *E. coli* isolates.

Isolate ID	gene	Length(bp)	Similarity%	Confirmed as	Accession number
EC.22	*hly*A	790 bp	100%	*hly*A	PX111889
EC.24	*hly*A	797 bp	100%	*hly*A	PX111890
EC.30	*hly*A	510 bp	100%	*hly*A	PX111891
EC.8	*fim*H	177 bp	100%	*fim*H	PX127663
EC.35	*fim*H	177 bp	100%	*fim*H	PX127664
EC.29	*tra*T	196 bp	97%	*tra*T	PX170155
EC.50	*tra*T	193 bp	97%	*tra*T	PX170156

4 Discussion

This study is the first to specifically investigate the antibiotic resistance patterns of *E. coli* isolates from cancer patients in Sulaimani City, Iraq. Due to bone marrow metastases, radiation, chemotherapy, and neutropenia, cancer patients usually have suppressed immune systems. However, the disturbance of mucosal surfaces leaves them open to opportunistic infections. Among cancer patients, UTIs rank among the major contributors to morbidity and mortality [18, 19]. The key findings of this study are as follows: (1)In cancer patients, UTIs were the most common clinical manifestation of *E. coli* infection; (2) the proportion of male and female patients was almost equal. The study cohort comprised individuals with a variety of cancer types, although hematological malignancies were more common. (3) Among the *E. coli* isolates, a considerable degree of antibiotic resistance was noted. Resistance to carbapenems and fluoroquinolones, two essential antibiotics for the treatment of severe infections, was present in a sizable fraction of isolates. (4) The presence of the *fim*H gene in our isolates was notably different from findings in other studies, which was unexpected. (5) The phenotypic and genotypic detection of the *hly*A gene were shown to differ. (6) Nitrofurantoin and aminoglycosides worked well as antibiotics to treat *E. coli* infections. UTIs were the most prevalent infection type (58%), as cancer patients are more susceptible to UTIs because of catheter use, urinary obstruction, and compromised immune systems. The risk of infection is significantly increased by surgery and prolonged catheterization [21]. Having a preponderance of female patients, such results have been documented in India [22] as women are more susceptible to UTIs than men because of anatomical and biological characteristics, including a shorter urethra and its proximity to the anus. Sexual activity also raises the chance of UTI infection, and about 50% of people get fecal contamination [23]. With a prevalence rate of 53.65%, *E. coli* was the most isolated pathogen from UTI in cancer patients at the Basrah Center of Oncology and Al-Sadr Teaching Hospital, Iraq [18]. However, bloodstream infections were the second most common illness among the study sample, specifically in males with hematological

malignancies. This could be due to the tumor itself that weakens the immune system, and the cytotoxic effects of chemotherapy [24]. For the majority of patients, digestive disorders were quite prevalent (25%). *E.coli* may cause bacteremia or sepsis through abdominal translocation in patients with certain intestinal diseases, potentially leading to systemic infections [25]. In the current investigation, 20% of the *E. coli* isolates were clinically significant, especially in immunosuppressed patients, such as those with cancer. Poor wound care, surgical site contamination, or subsequent infections can all lead to this condition [26]. Multidrug resistance (MDR) refers to in vitro susceptibility testing results showing that an isolate is resistant to at least one antibiotic in three or more different classes [8]. *E. coli* MDR has grown to be related to issues in humans and animals globally. Although *E. coli* is inherently susceptible to clinically important antibiotics, it has a strong tendency to acquire resistance genes, mainly via horizontal gene transfer [27]. The current study found that 72% of the isolates were MDR, which is more than that of MDR-resistant *E. coli* (50%) among cancer patients in an Ethiopian study [28]. High resistance to a range of frequently prescribed antibiotics was observed among the *E. coli* isolates. Due to ampicillin's widespread use as a first-line treatment, the highest resistance was found against it (88%), which is in line with worldwide trends. The significant level of fluoroquinolone resistance observed in this study is particularly worrisome as it exceeds rates observed in other regions. These findings are consistent with those of Saeed et al., who reported that *E. coli* isolates from pancreatic cancer patients exhibited 100% resistance to ciprofloxacin and approximately 94.11% resistance to levofloxacin. [29]. Naqid et al. found lower levels of Ciprofloxacin (45.4%) and levofloxacin (23.4%) resistance in Duhok, Iraq [30]. Ciprofloxacin resistance was found in 41% of *E. coli* isolates from leukemia patients in an Iranian study [31]. An alarming trend in antimicrobial resistance in the region was indicated by the *E. coli* strains isolated in Sulaymaniyah, Iraq, which showed a strong resistance profile. Since broad-spectrum drugs are widely and continuously used, the prophylactic use of antibiotics, especially in cancer patients, promotes the development of resistance to antibiotics [32]. Regional variations, the state of community health, and the careless use of antibiotics can all be implicated in the variations documented. Before the collection of diagnostic specimens, a considerable percentage of illnesses were empirically treated, which would have affected the microbiological results. Genetic alterations and resistant strains are encouraged to survive due to the selective pressure resulting from the overuse and misuse of antibiotics. It is frequently difficult to clinically differentiate between bacterial, viral, and non-infectious inflammatory diseases, which results in the improper prescription of antibiotics, especially in cases of respiratory infections, which are frequently viral in origin [33, 34]. *fim*H can be employed as a potential diagnostic marker and/or vaccination candidate because of its high binding ability, which may improve *E. coli*'s pathogenicity [35]. *tra*T is a viable target for therapeutic approaches as it is a prevalent and essential virulence factor [36]. No significant relationship was observed between antibiotic resistance and the presence of any of the investigated genes ($p \geq 0.05$). *fim*H (100%), *tra*T (31.7%), and *hly*A (15.9%) were identified by Hassan et al., who observed a

strong connection between these genes and common antibiotics (CEF, AMP, GEN, SXT, LVO, and NIT) [13]. However, *fim*H was found to be associated with greater levels of quinolone resistance (90.6%) by Guo et al. [12]. At the same time, Roshani et al. discovered 17 virulence genes and concluded that virulence and antibiotic resistance are unrelated [37]. Only one isolate in this study exhibited hemolytic activity on blood agar, despite the hemolysin gene being genotypically detected in 30% of the isolates. The disparity between genotypic and phenotypic data could be explained by the gene being present but not expressed in the designated laboratory circumstances. The lack of phenotypic manifestation despite the gene's existence could be attributed to environmental influences, gene control, or mutations that affect gene expression. Although no study has directly linked the presence of these virulence genes with cancer patients, according to previous studies, virulence factors such as *ibe*A, *iro*N, *sfa*, and *usp* are more commonly present in *E. coli* strains isolated from neoplasia patients than in healthy controls. Additionally, these virulence genes were more common in neoplasia that had progressed [38]. This study is limited by its relatively small sample size, examination of only a limited set of virulence genes, and an antibiotic panel that did not cover all relevant drugs. Future studies should include a larger number of isolates, a broader range of antibiotics, and additional virulence factors to more comprehensively assess the association between *E. coli* resistance and virulence.

5 Conclusion

The high frequency of MDR *E. coli* in cancer patients is highlighted by the significant virulence genes (*fim*H, *tra*T, and *hly*A). The considerable prevalence of antibiotic resistance underscores the urgent need for ongoing surveillance and prudent antibiotic use. The molecular mechanisms of *E. coli* virulence and resistance in cancer Patients should be better understood by conducting additional research with larger sample sizes and more gene panels.

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8. Conflict of interest: No conflicts of interest are declared by the authors in relation to this study.

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