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Research Article

Antimicrobial Resistance and Virulence Gene Profiles of Escherichia Coli Isolated from Stool Samples of Patients with Gastrointestinal Disorders in Buea, Cameroon

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Abstract

The rising antibiotic resistance in *Escherichia coli* (*E. coli*), coupled with the presence of virulence factors, poses significant challenges for infection management, amplifying pathogenicity and therapeutic failure. This study investigated the antimicrobial resistance and virulence gene profiles of *E. coli* isolates from stool samples of patients with gastrointestinal disorders and explored potential associations between antibiotic resistance and virulence factors. A total of 244 *E. coli* isolates were recovered from 373 stool samples, identified through biochemical methods and confirmed by polymerase chain reaction (PCR). Singleplex and multiplex PCR assays were employed to screen for 22 virulence genes, categorized into adhesins (*fimH*, *afa*, *papE*, *papC*, *nfaE*, *bmaE*), invasins (*ibeA*, *colV*), toxins (*stx1*, *stx2*, *cdtB*, *est*, *elt*), capsule synthesis genes (*kpsII*, *rfc*), siderophores (*iutA*, *chuA*, *ire*), and others, including *pai*, *hlyA*, and *eaeA*. Antibiotic susceptibility testing (AST) was performed on 50 randomly selected isolates harboring at least one virulence gene using the Kirby-Bauer disc diffusion method. Among the 244 *E. coli* isolates, 178 (73.0%) harbored at least one virulence gene. The detected genes were categorized into adhesins (33.2%), toxins (13.5%), siderophores (10.7%), and capsule synthesis and invasins (5.3%), with *fimH* (18.0%) and *papC* (6.1%) being the most prevalent. Notably, two virulence genes, *rfc* and *eaeA*, were not detected in any isolates. Antibiotic susceptibility testing (AST) revealed alarmingly high resistance rates to beta-lactams (90% for Amoxicillin and Ampicillin), Tetracycline (80%), Doxycycline (74%), and Sulfamethoxazole (88%), with all isolates exhibiting complete resistance to Metronidazole. In contrast, Ceftriaxone demonstrated moderate efficacy (48% sensitivity), while Ciprofloxacin (74%) and Gentamicin (76%) were the most effective antibiotics. A significant positive correlation ($r = 0.635$, $P = 0.000$) was observed between the presence of virulence genes and antibiotic resistance, underscoring the interplay between these factors. These findings highlight the need for further research to unravel the molecular mechanisms linking virulence and resistance in *E. coli*. Targeted interventions addressing both virulence and resistance factors are essential to improving therapeutic outcomes and combating multidrug-resistant *E. coli.*

Keywords

E.coli., *E. coli* Isolates, Virulence Genes, Antibiotic Resistance

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1. Introduction

Escherichia coli (*E. coli*) is a versatile bacterium that naturally inhabits the gastrointestinal tracts of humans and animals, where many strains play beneficial roles in metabolism, immune system development, and protection against pathogenic microbes [1]. However, some strains have acquired virulence factors, transforming them into pathogenic variants capable of causing a range of intestinal and extra-intestinal diseases [2, 3]. These virulence factors, encoded by specific genes, include adhesion molecules, toxins, biofilm formation factors, and siderophores, which facilitate tissue attachment, cell damage, bacterial growth, and iron scavenging [4].

The pathogenicity of *E. coli* is driven by a complex mechanism involving numerous virulence factors that vary across pathotypes. These factors are encoded by distinct gene clusters that enhance the bacterium's ability to adapt to diverse environments, colonize the gastrointestinal tract, and cause disease [5]. Furthermore, the expression of these virulence genes is often activated under favorable conditions, underscoring their role in disease manifestation [6]. Supplementary table S1 [4, 6, 7-14] highlights some of the virulence genes elaborated by E.coli and the role they play in infection. Understanding the genetic basis of *E. coli* virulence provides valuable insights into its pathogenesis and highlights targets for preventive strategies.

Antimicrobial resistance (AMR) is recognized as one of the most pressing global threats to public health and development, as highlighted by the Antimicrobial Resistance Collaborators [15]. Their report estimates that bacterial AMR was directly responsible for 1.27 million deaths worldwide in 2019 and contributed to an additional 4.95 million deaths. Multidrug-resistant (*MDR*) *Escherichia coli* strains have become increasingly prevalent, posing significant challenges in clinical settings by rendering previously treatable infections more difficult to manage [16, 17]. Studies globally have reported high levels of antibiotic resistance in *E. coli* isolates associated with gastrointestinal infections, urinary tract infections, and bloodstream infections [18, 19]. The situation in Cameroon is particularly concerning, with multiple studies documenting high resistance levels in *E. coli* isolates to commonly used antibiotics, including beta-lactams, aminoglycosides, fluoroquinolones, and sulfonamides [20-22]. Aggravating this challenge is the coexistence of virulence and antibiotic resistance genes in *E. coli*. Recent studies have shown that these genes can reside on the same mobile genetic elements, such as plasmids and transposons, facilitating their co-transfer and enhancing both pathogenicity and resistance [23]. This dual functionality is further complicated by regulatory networks that coordinate virulence gene expression and antibiotic resistance mechanisms, as well as biological processes like biofilm formation, quorum sensing, and adhesion to host cells [24, 25]. Efflux pumps, for instance, that facilitate antibiotic resistance by expelling out of the bacterial cell antibiotics also play important roles in biological processes such as biofilm formation, quorum sensing, bacterial adhesion to host cells, and evasiveness [25, 26].

In this study, *E. coli* strains were isolated from stool samples of patients with gastrointestinal disorders to investigate their antimicrobial resistance patterns and virulence gene profiles. These bacteria were studied as potential causative agents of gastrointestinal infections rather than as components of the normal flora, given the clinical presentation of the patients. The study aimed to assess the association between virulence genes and antibiotic resistance, providing insights into the pathogenicity and treatment challenges posed by *E. coli* infections in this region. Understanding these associations is critical for effective patient management and combating the rising threat of AMR in pathogenic *E. coli*.

2. Materials and Methods

2.1. Study Design and Study Sites

This cross-sectional study was conducted at the Laboratory for Emerging Infectious Diseases, Faculty of Science, University of Buea, involving two health facilities in Buea: Solidarity Health Foundation in Molyko and Buea Regional Hospital Annex. Buea, the capital of the Southwest Region, is situated on the eastern slope of Mount Cameroon. According to the 2013 census, the population was estimated at about 300,000 inhabitants [27]. However, the population has markedly increased in recent years due to an influx of internally displaced people from other parts of the Southwest and Northwest regions as a result of the Anglophone crisis in Cameroon [28]. The Buea Regional Hospital Annex (coordinates: 4.148856, 9.237076) is a main referral hospital in the Southwest Region, serving patients from across the region. Solidarity Health Foundation (coordinates: 4.158374, 9.281743) is located in Molyko, one of the densely populated areas in Buea.

2.2. Ethical Considerations

Ethical endorsement for this work was obtained from the Faculty of Health Sciences Institutional Review Board, University of Buea (no 2022/1537-12/UB/SG/IRB/FHS), administrative authorization was obtained from the South West Regional Delegation of Public Health (No R11/MINSANTE/SWR/RDPH/PS/536/716). Information sheets presenting the objectives and procedures of the study were explained to each participant. Answers were also given to their questions concerning the study. Written informed consent was obtained from each participant before his/her discretionary participation.

2.3. Sample Collection and Transportation

Stool samples were collected from participants at both Solidarity Health Foundation and Buea Regional Hospital Annex after obtaining informed consent. The samples were taken from patients with gastrointestinal disorders who were advised to undergo stool testing. Sociodemographic information and clinical indications for each participant were recorded using a simple structured questionnaire. A total of 372 stool samples were collected between March 2022 and July 2022 using sterile containers. To prevent the multiplication of endogenous microbes, all samples were maintained at a temperature of 4 ℃ in a cool box with ice packs and transported to the laboratory within two hours of collection for analysis. Samples were processed immediately upon arrival at the laboratory.

2.4. Isolation of Presumptive E. coli

Each sample was inoculated, by streaking, on MacConkey agar (L: S-BIOTECH, USA) and incubated for 24 h at 37 ℃. Pink-coloured colonies were purified on nutrient agar ((L: S-BIOTECH, USA) and transferred to eosin methylene blue (EMB) agar (CONDALAB, Laboratories Conda S.A, Spain) and methyl red Voges–Proskauer (MRVP) broth (BD Life Sciences, New Jersey, United States) to check for the presence of E. coli characteristics. Isolates that produced green metallic sheen on EMB agar, catalase positive, oxidase negative, methyl red-positive, VP-negative and appearance as gram-negative rods were considered presumptive E. coli.

2.5. DNA Extraction

All isolates identified as presumptive E. coli were inoculated on nutrient broth and incubated overnight at 37 ℃. Genomic DNA was extracted from each isolate using the QIAamp DNA Mini Kit (QIAGEN, Germany) following the manufacturer's instructions. The extracted DNA was stored at -20 ℃ until used.

2.6. Confirmation of E. coli and Amplification of Virulence Genes by PCR

To confirm the isolates, the extracted DNA from each isolate was subjected to molecular screening by PCR using the 16S rRNA species-specific primers (ECO1 and ECO2) (Supplementary table S2). All isolates confirmed for the presence of 16S rRNA were further analysed for 22 virulence genes (pai, fimH, papE, eaeA, ibeA, fyuA, bmaE, sfa, iutA, chua, papC, hlyA, rfc, nfaE, kpsMT II, papC, cvaC, cdtB, stx1, stx2, afa, cnf1) (supplementary table 2) [3, 29-37].

PCR (for16S rRNA and the analysis of the virulence genes) was performed using Multigene optimax thermocycler (Labnet international, Inc. USA) as follows; reaction mix of 25 µL was done in 2 ml PCR tubes containing 12.5 µL master mix (One Taq 2x Master Mix with Standard Buffer, New England BioLabs), 0.5 µL each of 20 µmol forward and reverse primers (Inqaba biotech, South Africa), 6.5 µL nuclease-free water and 5 µL DNA template. PCR cycling was performed using the conditions summarized in (supplementary Table 2). All PCR products were analysed electrophoretically in 1.5% agarose gel prepared in 1X Tris Borate EDTA (TBE) buffer, SYBR safe DNA gel staining dye (1 µL in 10 mL of agarose) was added to the molten agarose before pouring in the gel trough for solidification. One microlitre of the loading dye (blue/orange 6X loading dye, PROMEGA-USA) was mixed in 5 µL of each PCR product, loaded into each well and electrophoresed at 80 V for 60 min in 1X TBE. A 100 bp DNA ladder (Invitrogen, Life technologies, USA) was used as molecular weight marker. The PCR products were visualized under ultraviolet light and gel pictures taken using a Gel Documentation-XR (BIORAD, Hercules, CA USA).

2.7. Antibiotic Susceptibility Testing Procedure

2.7.1. Selection of Isolates and Antibiotics for Antibiotic Susceptibility Testing

To achieve the objective of relating antibiotic resistance to virulence, fifty randomly selected E. coli isolates containing at least one virulence gene were used for antibiotic susceptibility testing. Fourteen antibiotics were chosen to ensure representation from different antibiotic classes, including Beta-Lactams (Ampicillin (AMP, 10 µg), Amoxicillin (AMX, 30 µg), Ceftriaxone (CRO, 30 µg)), Chloramphenicol (C, 30 µg), Fluoroquinolones (Ciprofloxacin (CIP, 5 µg), Norfloxacin (NOR, 30 µg)), Tetracyclines (Doxycycline (DO, 30 µg), Tetracycline (TE, 30 µg)), Aminoglycosides (Gentamicin (CN, 30 µg), Kanamycin (K, 5 µg), Spectinomycin (SPT, 25 µg), Streptomycin (S, 25 µg)), Sulfonamides (Sulfamethoxazole (STX, 25 µg)), and Nitroimidazoles (Metronidazole (MET, 30 µg)). It is important to note that metronidazole is not effective against Gram-negative bacteria like E. coli; however, it was included in this study because many participants reported using this antibiotic at home when experiencing symptoms of diarrhoea or gastrointestinal disorders. Although primarily effective against protozoa and anaerobic bacteria, metronidazole's inclusion provides valuable insight into the inappropriate use of antibiotics in self-medication practices and the potential implications for antibiotic resistance in E. coli. This selection ensured coverage of commonly used antibiotic classes while reflecting real-world usage patterns among the study participants.

2.7.2. Antibiotic Susceptibility Testing

Using the Kirby-Bauer disc diffusion method antimicrobial susceptibility testing was performed on the 50 E. coli isolates that were randomly selected against fourteen antibiotics as elaborated above. A bacterial suspension was prepared by

transferring 4 - 5 colonies of the pure culture into a tube containing 2.5 mL sterile normal saline using a sterile inoculating loop. The suspension was vortexed and its turbidity adjusted to that of barium chloride (0.5 McFarland turbidity standard; 1.0 x 108 CFU / µL). One hundred microlitres of the inoculum were spread on Mueller-Hinton agar plate. Plates were allowed to dry at room temperature in the biological safety Cabinet Class II. Disks containing predetermined amounts of antibiotics were dispensed unto the bacterial lawn using a pair of sterile forceps and pressed gently to ensure complete contact with the agar. The disks were placed 15 mm away from the edge of the plate and 25 mm away from each other. Within 15 minutes after the disks were dispensed, the plates were inverted and incubated at 37 ℃ for 24 h. After incubation, they were examined and diameters of the inhibition zones were measured using a ruler and interpreted as recommended by the Clinical and Laboratory Standards Institute, 2022 [38]. The expression of the results as sensitive, intermediate and resistant was performed according to Clinical and Laboratory Standards Institute guidelines. Multi drug resistance (MDR) was considered as bacterial resistance to 3 antibiotics belonging to at least 3 classes or families.

2.8. Statistical Analysis

All data findings were transferred into a Microsoft Excel spreadsheet (Microsoft Corporation, Redmond, WA, USA) and analyzed using R software (version 4.3.2; R Foundation for Statistical Computing, Vienna, Austria). Associations of prevalence and other variables was determined by Chi-square (X2) testing at the significance level of $p< 0.05$. The Shapiro-Wilk test was use to test the normality of data distribution. In order to compare the frequencies obtained, virulence genes and antibiotic resistance, chi-square (X2) test was used. The correlation between virulence genes and antibiotic resistance was established using Pearson's correlation coefficient (r2) in bivariate linear correlations ($P< 0.05$). P-value is significant if it is ≤ 0.05.

3. Results

3.1. Prevalence of *E. coli* and Virulence Genes

Out of the 372 stool samples analysed, the overall *E. coli* prevalence was 244(65.7%), (X^2, p=0.07) supplementary figure S1 shows the colonial morphology of *E.coli* on EMB agar. E coli confirmation was done by the presence of 16SrRNA visualised on 1.5% agarose gel using UV trans illuminator (supplementary Figure S2). The prevalence of *E. coli* was higher in Buea Regional Hospital Anex 147(60%) than Solidarity. Health foundation 79(39.7%) and there was a significant difference between the two hospitals (T-test, P=0.02). The 244 confirmed isolates screened for the presence of 22 virulence genes *pai, fimH, afa, Ibe, papE, papC, nfaE, bMaE, eaeA, cfn, stx1, stx2, HlyA, cdt, est, elt, KpsII, and rfc, iutA, colv, chuA and ire* using singleplex and multiplex PCR assays (supplementary table 3) showed that 178(73.0%) possessed at least one virulence gene. Of the 178 isolates, 81(33.2%) possess adhesins, 33(13.5%) had toxins, 26(10.7%) siderophores, 13(5.3%) for both capsules synthesis and invasins and 12(4.9%) for others as detailed in figure 1. Among all genes tested, *fimH* was the gene with the highest frequency 44(18.0%) followed by *papC* 15(6.1%). Supplementary Table S3 reveals detailed profile of each of the virulence genes investigated in this study. Two amongst the virulence genes tested *rfc* (0.0%) and *eaeA* (0.0%) were not detected in any of the isolates. The gel pictures with expected band sizes for some of the virulence genes detected in this study can be seen in supplementary figures S3, S4, S5, S6, S7, and S8).

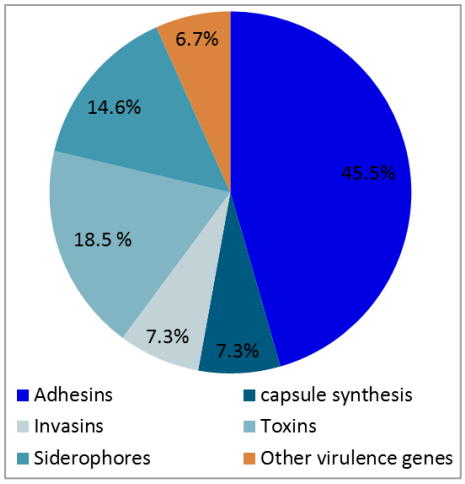

Figure 1. *Prevalence of virulence genes according to different classes.*

3.2. Antimicrobial Susceptibility Patterns

The antibiotic susceptibility profiles of 50 *E. coli* isolates from the stool samples is summarized in the supplementary table S4, with proportions of isolates that were sensitive, intermediate, or resistant to each antibiotic. The statistical significance of resistance patterns was analyzed using Chi-square (X^2) tests. Our findings from the antibiotic susceptibility testing revealed significant resistance patterns across various classes of antibiotics. Among the β-Lactam antibiotics, Amoxicillin (AMX) and Ampicillin (AM) showed alarmingly high resistance rates, with 90% of isolates resistant to both, and minimal sensitivity observed (AMX: 4%, AM: 10%). Ceftriaxone (CRO), a third-generation cephalosporin, presented a more balanced profile, with 48% of isolates being sensitive and 34% resistant. Within the Phenicol and Tetracycline classes, Chloramphenicol (C) exhibited a resistance rate of 64%, while Doxycycline (DO) and Tetracycline (TE) showed even higher resistance rates of 74% and 80%, respectively. Sensitivity to DO and TE was low, with only 20% and 18% of isolates sensitive to these antibiotics. Fluoroquinolones such as Ciprofloxacin (CIP) demonstrated better

efficacy, with 74% of isolates sensitive, though resistance was still present in 24% of cases. Norfloxacin (NOR) was less effective, showing 64% resistance, with only 30% of isolates being sensitive. Among the Aminoglycosides, Gentamicin (CN) stood out as a relatively effective option, with 76% of isolates sensitive and only 20% resistant. However, Kanamycin (K) and other Aminoglycosides like Spectinomycin (SPT) and Streptomycin (S) had high resistance rates (64%, 74%, and 72% respectively), indicating limited efficacy. Of particular concern was the resistance of Metronidazole (MET) to all the 50 clinical isolates, with no intermediate or sensitive responses, reflecting a uniform resistance pattern. Lastly, Sulfamethoxazole (SXT) equally exhibited a high resistance rate of 88%, with only 6% of isolates being sensitive.

4. Discussion

The genome of *E. coli* possesses remarkable plasticity, allowing it to gain or lose virulence genes at a relatively high frequency [10]. Such acquired genetic material may include genes linked to both virulence and antibiotic resistance [39]. Virulence genes and antimicrobial resistance genes can be located on the same mobile genetic elements, such as plasmids, transposons, or integrons, leading to their co-selection when exposed to antibiotics [40]. The transfer of these mobile genetic elements between bacteria can spread both virulence and resistance genes simultaneously, increasing the likelihood of co-occurrence [41]. Also, Antibiotic use exerts selective pressure that not only selects for resistant strains but can also indirectly select for virulent strains carrying resistance genes. This selective pressure enhances the prevalence of multi-resistant and virulent strains [42]. In some cases, virulence genes confer a fitness advantage in hostile environments, such as those created by antibiotic treatment, aiding in the survival and proliferation of resistant strains [24]. This association poses significant clinical challenges, leading to more severe infections and complicating treatment options resulting in higher morbidity and mortality rates.

Various studies have found a positive correlation between the presence of certain virulence genes (e.g., those encoding for toxins, adhesins, or iron acquisition systems) and antimicrobial resistance in clinical and environmental E. coli isolates [43]. Common virulence factors associated with antimicrobial resistance in E. coli include fimbriae, toxins, siderophores, and invasion-associated genes. Understanding this relationship is crucial for developing effective strategies to combat antibiotic-resistant *E. coli* infections [44].

Our study revealed notably that the genes responsible for adhesion otherwise classified as adhesins (*FimH, papC, papE, afa, bmaE and nfaE*) emerged as the most prevalent virulence gene category, as depicted in Figure 1. Adhesins are proteins crucial for bacterial attachment, a pivotal step in bacterial pathogenesis. These proteins not only facilitate initial attachment to host cells but also shield pathogens from potential host defense mechanisms, thereby enhancing the utilization of other virulence attributes [45]. Among all virulence genes examined, *fimH* was the most frequently observed. *FimH*, found on fimbriae, plays a critical role in bacterial attachment by mediating the mannose-sensitive binding of *E. coli* to various cell types [46]. This adherence process amplifies the expression of bacterial toxins and contributes to iron acquisition while evading host defense mechanisms [47]. Toxins were also prevalent, with Shigatoxins (*stx1* and *stx2*) being the most predominant, Shiga toxins are produced by a subset of *E. coli* known as Shiga toxin-producing *E. coli* (STEC), and they pose significant public health concerns, causing a spectrum of gastrointestinal diseases ranging from diarrhea to severe conditions such as hemolytic uremic syndrome (HUS) [48]. The presence of *stx2* is particularly of great concern due to its potency, being 1000 times more potent than *stx1*, and its association with hemolytic uremic syndrome in humans [49]. Additionally, other virulence genes coding for siderophores (iutA, ire, andiutA, ire, chuA), capsules (*KSP11)*, and invasins (ibeA and cva), were detected. Siderophore production by pathogenic *E. coli* is a significant virulence factor, facilitating survival and colonization while leading to various clinical manifestations, including diarrhea, urinary tract infections (UTIs), hemolytic uremic syndrome (HUS), hemorrhagic colitis (HC), neonatal meningitis, and bacteremia [50]. Strains capable of over-producing siderophores are considered hyper-virulent, while those unable to produce or secrete siderophores exhibit reduced virulence and fitness during infection and colonization [8]. Although only one of the two tested genes for capsule synthesis (KpsII) was present, capsules emerged as important virulence determinants, enabling pathogenic bacteria like *E. coli* to evade and counteract nonspecific host defenses during the early phases of infection. Capsules hinder the action of complement and phagocytes [51].

In addition to its virulence attributes, *E. coli* also exhibits resistance to antimicrobial agents. The emergence of multidrug-resistant *E. coli* in various countries over recent decades has raised serious concerns, as it leads to infections with heightened virulence potential [52]. The escalating antibiotic resistance, particularly the concurrent increase in multidrug-resistant *E. coli*, poses a substantial challenge to the treatment of *E. coli*-related diseases [16].

Among the 50 clinical *E. coli* isolates, significant resistance patterns emerged, particularly for commonly prescribed antibiotics. High resistance rates were observed for Amoxicillin and Ampicillin (90% each) and Sulfamethoxazole demonstrated a high resistance rate (88%), Tetracycline (80%) and Doxycycline (74%). Metronidazole showed alarming resistance of (100%) to all the clinical isolates, likely due to its frequent use for gastrointestinal and gynecological infections in Cameroon [53, 54]. This widespread resistance to first-line antibiotics, which are crucial for primary healthcare [55], raises concerns about their continued viability in treating *E. coli* infections and generally other bacteria in clinical settings. In contrast, some antibiotics retained higher effectiveness. Ceftriaxone, a third-generation cephalosporin, had a compara-

tively lower resistance rate (34%). Ciprofloxacin displayed moderate efficacy, with 74% sensitivity, though 24% of isolates were resistant, suggesting it may remain viable when susceptibility testing confirms sensitivity. Gentamicin was notably effective, showing a sensitivity rate of 76% and a low resistance rate of 20%, making it a promising treatment option. This aligns with findings by Afum et al. [56] and Wu et al. [16], who suggested that Gentamicin's intramuscular administration and limited over-the-counter availability have helped curb resistance development. Overall, the observed resistance patterns highlight the limited efficacy of commonly used antibiotics, especially β-Lactams, Macrolides, and certain Aminoglycosides, while Ciprofloxacin and Gentamicin appear to offer more promise due to higher sensitivity rates. These findings underscore the importance of regular susceptibility testing to guide antibiotic selection and reduce reliance on ineffective drugs. Furthermore, *E. coli*'s ability to acquire resistance genes through horizontal gene transfer exacerbates the challenge of antimicrobial resistance [57, 58]. Combating this issue will require vigilant stewardship and prudent antimicrobial use to sustain treatment efficacy and manage resistance.

The results of our antibiotic sensitivity tests unequivocally revealed a compelling relationship between the possession of virulence genes and antibiotic resistance. The statistical analysis, employing Pearson's correlation coefficient, underscored the strength and significance of this association ($r = 0.738$, $p < 0.001$). These findings align with prior research, such as the study conducted by Abd El-Baky *et al*. [16], which posited that the acquisition of virulence and resistance genes can augment the pathogenicity of microorganisms, potentially leading to more severe infections and a heightened risk of treatment failure. Building upon this foundation, a recent investigation by Hassan *et al* [31] also lent support to the notion that a correlation exists between the possession of virulence genes and antibiotic resistance. However, it is imperative to acknowledge the complexity of the relationship between virulence factors and antibiotic resistance. This complexity necessitates further in-depth studies, each tailored to specific geographic areas and the microorganisms under examination.

5. Conclusion

In summary, our study revealed that a substantial proportion of our *E. coli* isolates (73.0%) possessed at least one virulence gene. The randomly selected 50 isolates with at list one virulence gene used for antibiotic susceptibility testing exhibited a marked degree of multidrug resistance against the antibiotics assessed. This underscores the existence of a tangible association between virulence and antibiotic resistance. Nonetheless, it is vital to recognize that antibiotic resistance involves a multifaceted interplay of numerous factors and mechanisms. Consequently, the link between the possession of virulence genes and antibiotic resistance strongly suggested by our findings and supported by existing research, remains a hypothesis requiring further in-depth exploration, with a focus on specific geographic regions and microbial species.

Abbreviations

E. coli	Escherichia coli
AST	Antibiotic Susceptibility Testing
AMP	Ampicillin
Amx	Amoxicillin
CRO	Ceftriaxone
30µg	Chloramphenicol
CIP	Ciprofloxaci
NOR	Norfloxacin
DA	Clindamycin
DO	Doxycycline
TE	Tetracycline
E	Erythromycin
SP	Spiramycin
CN	Gentamicin
K	Kanamycin
SPT	Spectinomycin
S	Streptomycin
MET	Metronidazole
STX	Sulfamethoxazole
PFGE	Pulsed-field Gel Electrophoresis
EDTA	Ethylenediaminetetraacetic Acid
TBE	Tris/Borate/EDTA
MDR	Multiple Drug Resistance
USA	United States of America
AMR	Antimicrobial Resistance
EMB	Eosin Methylene Blue
VP	Voges–Proskauer
MR	Methyl Red
rRNA	Ribosomal Ribonucleic Acid
DNA	Deoxyribonucleic Acid
mL	Mililitres
CFU	Colony Forming Unit
µL	Microlitres

Supplementary Material

The supplementary material can be accessed at https://doi.org/10.11648/j.ejcbs.20251101.11

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Ethical Approval and Consent to Participate

Ethical endorsement for this work was obtained from the

Faculty of Health Sciences Institutional Review Board, University of Buea (no 2022/1537-12/UB/SG/IRB/FHS), and administrative authorization from South West Regional Delegation of Public Health (No R11/MINSANTE/SWR/RDPH/PS/536/716). All participants gave a sign concern before they participated in the study.

Consent for Publication

Not applicable.

Author Contributions

Jerome Kfusi Achah: Conceptualization, data collection, Laboratory investigation, data analysis and drafting of manuscript.

Seraphine Nkie Esemu: Conceptualization, **C**ritically edited the manuscript.

Fabrice Tangi Fongwa: Laboratory analysis (Microbiological and Molecular analysis).

Seraphine Nkie Esemu: Were involved in designing and supervising the entire study, critically revising and contributing to the manuscript's scientific content. S.

Lucy Mande Ndip: Were involved in designing and supervising the entire study, critically revising and contributing to the manuscript's scientific content. S.

Lucy Mande Ndip: Read and gave final approval of the version to be published and agreed to account for all the aspects of the work.

Roland Ndip Ndip: Read and gave final approval of the version to be published and agreed to account for all the aspects of the work.

Declaration

This article or work has not been published previously, nor is it under consideration for publication elsewhere. All authors approve the publication, and if accepted, it will not be published elsewhere, in English or any other language, without the copyright holder's written consent.

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Data Availability Statement

The data is available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest.

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Biography

Jerome Kfusi Achah is a skilled laboratory scientist and microbiologist affiliated with the University of Buea, Cameroon. He holds a Bachelor of Medical Laboratory Sciences (B.M.L.S.), a Master of Science in Microbiology, and is currently pursuing a Ph.D. in Microbiology at the University of Buea. With over a decade of experience, he specializes in molecular and cellular microbiology, focusing on research and diagnostics. As a Graduate Teaching Assistant, Research Assistant, and Laboratory Technologist at the Laboratory for Emerging Infectious Diseases (LEID), Mr. Achah has expertise in diagnosing infectious diseases, including COVID-19, and investigating zoonotic pathogens such as Escherichia coli, Brucella, and influenza viruses. His major work encompasses molecular diagnostics (PCR, ELISA), quality control, and mentoring students in scientific research. Renowned for his proficiency in biosafety, biosecurity, antimicrobial resistance, and emerging infectious diseases, he has made significant contributions to public health in Cameroon.

Seraphine Nkie Esemu is a distinguished academic in the Department of Microbiology and Parasitology at the University of Buea, Cameroon, where she holds a PhD, Master's, and Bachelor's degree in Medical Microbiology. Recognized for her academic excellence, she has received multiple accolades, including the Research Excellence Award from the University of Buea. Currently serving as the Head of the Division for Research and Publications, Dr. Esemu has made significant contributions to research in microbiology, focusing on infectious diseases, antimicrobial resistance, and epidemiology, particularly addressing public health challenges in Cameroon and sub-Saharan Africa. She has published numerous peer-reviewed articles and supervised both undergraduate and MSc students. A recipient of prestigious fellowships, including the African Research Excellence Fund (AREF) Fellowship, Dr. Esemu is actively involved in key microbiology and biosecurity associations.

Fabrice Tangih Fongwa is an aspiring biotechnologist with a strong academic foundation and practical expertise in microbiology and infectious disease diagnostics. He holds a Bachelor of Science in Microbiology (Honors) and a Master of Science in Microbiology, both from the University of Buea, Cameroon. Currently, he is pursuing an MSc in Biotechnology at the Technical University of Denmark, expected in 2025. Professionally, Fabrice served as a Laboratory Technician and Research Assistant at the Laboratory for Emerging Infectious Diseases, University of Buea. His major responsibilities included diagnosing COVID-19, RNA and DNA extraction, performing PCR and ELISA assays, and serotyping Vibrio cholerae during cholera outbreaks. He also contributed to the surveillance of transboundary animal diseases and has training in cell and tissue culture. Fabrice's expertise spans molecular diagnostics, data analysis with R programming, and adherence to biosafety protocols, positioning him for innovative contributions in public health and biotechnology.

Roland Ndip Ndip is a Professor of Microbiology and current Dean of the Faculty of Science, University of Buea, Cameroon. Professor NDIP studied at premier institutions in Nigeria (BSc, MSc, PhD in Medical Microbiology) with specializations in pathogenic and molecular microbiology. He extended his training through participation in short courses and laboratory visits abroad at the University of Glasgow, Scotland, and the German Research Centre for Biotechnology, Braunschweig, Germany. Professor NDIP has a long standing interest and active research programme in infectious disease, and in the utility of both western and traditional (herbal) medicines for the treatment of infectious diseases.

Lucy Mande Ndip is a distinguished academic and researcher at the University of Buea, Cameroon. She holds a Bachelor of Science in Microbiology from the University of Uyo, Nigeria, a Master's degree in Medical Microbiology from Edo State University, and a Doctorate in Medical Microbiology from the University of Buea. She is a Professor with an extensive career in microbiology and infectious diseases. Beginning as an Assistant Lecturer in the Department of Life Sciences at the University of Buea, she has held key positions, including Head of Service for Teaching and Research, Head of the Department of Biomedical Sciences, Deputy Vice Chancellor for Internal Control and Evaluation, and Deputy Vice Chancellor for Research and Cooperation. She is currently the Director of HTTC Buea and the Laboratory for Emerging Infectious Diseases, renowned for its role in public health during the COVID-19 pandemic and cholera outbreaks. Her research focuses on antimicrobial resistance, pathogen virulence, and epidemiology. As a mentor and educator, Professor Ndip has advanced microbiology, nurtured young scientists, and conducted groundbreaking studies addressing global health challenges.