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Original Article

Antibiotics Resistance in *Sul1* Gene of *Escherichia coli*: Physiological and Molecular Study

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Abstract

Background: Antibiotic resistance in *Escherichia coli* is a significant public health concern, particularly in regions with limited healthcare resources. The *sul1* gene, commonly associated with mobile genetic elements, encodes sulfonamide resistance and is prevalent in multidrug-resistant *E. coli* strains linked to diarrheal diseases. This study **aimed** to assess the prevalence and molecular characteristics of the *sul1* gene in *E. coli* strains isolated from patients with diarrhea and to investigate its association with multidrug resistance patterns. **Methods**: A total of 60 fecal samples were collected from diarrheal cases in a clinical setting. *E. coli* strains were isolated and identified through standard microbiological techniques, including selective media culture, Gram staining, colony morphology observation, and API 20E biochemical testing. Genomic DNA was extracted, and PCR amplification of the *sul1* gene was performed. Amplified products were analyzed through agarose gel electrophoresis, sequenced, and aligned with reference sequences. Phylogenetic analysis was conducted to examine genetic relationships among isolates. **Results**: The *sul1* gene was consistently detected across all *E. coli* isolates tested, with an 822 bp amplicon verified by sequencing and BLAST analysis. High sequence similarity (~99%) was observed between the local isolates and reference sequences. Phylogenetic analysis revealed close clustering of the isolates within the *E. coli* clade, indicating genetic homogeneity among local strains. Notable nucleotide substitutions were identified, though they did not result in amino acid changes, suggesting silent mutations. **Conclusions**: The high prevalence of the *sul1* gene in *E. coli* isolates from diarrheal cases emphasizes the role of mobile genetic elements in spreading sulfonamide resistance. **Recommendation**: Regular genetic screening for resistance genes, rational antibiotic use, infection control, and research on alternative treatments for multidrug-resistant bacterial infections are needed.

Keywords: *Antibiotic Resistance; E. Coli; DNA Sequencing; Sul1 Gene*

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Introduction

Escherichia coli (*E. coli*) is a versatile bacterium that exists both as a harmless resident of the human gut and as a significant pathogen responsible for diseases like urinary tract infections, neonatal meningitis, and gastrointestinal disturbances, including severe diarrhea (Lamichhane *et al*., 2024). Diarrheal diseases remain a pressing global health issue, with *E. coli* recognized as a major etiological agent, particularly in children and immunocompromised individuals (Baseri,*et al.*, 2021; Pato and Brown, 1963). This is particularly problematic in low-resource settings, where sanitation challenges amplify the transmission of such enteric pathogens and hinder effective treatment - Antimicrobial resistance (AMR) in *E. coli* has emerged as a critical public health threat, driven largely by the bacterium's capacity to acquire and disseminate resistance genes (Lamichhane *et al*., 2024). One such gene, sul*1*, encodes for sulfonamide resistance and is commonly associated with integrons, which facilitate the horizontal transfer of resistance determinants among bacterial populations. This gene's association with mobile genetic elements amplifies its role in propagating resistance, making infections caused by *sul1* **-** positive *E. coli* challenging to treat with standard antibiotic regimens (Partridge, *et al.* 2018; Moran & Hall, 2019). The clinical isolates are of particular concern because it frequently coexists with genes conferring resistance to other classes of antibiotics, indicating a multidrug-resistant phenotype. Studies suggest that sulfonamide-resistant *E. coli* strains are increasingly prevalent in both clinical and community settings (Algarni, *et al*., 2022; & Hemati *et al*., 2024). This broad resistance profile complicates management strategies for infections caused by these pathogens, especially in cases of paediatric diarrhea where treatment options are limited (Hemati *et al*., 2024). In this study, we aimed to assess the prevalence and molecular characteristics of the *sul1* gene in *E. coli* strains isolated from diarrhea cases. By analysing the genetic makeup and resistance patterns of these isolates, we can enhance our understanding of the role *sul1* plays in antibiotic resistance within diarrheal infections. The findings will contribute valuable data on the epidemiology of sulfonamide resistance in *E. coli* and provide insights for targeted interventions and antibiotic stewardship efforts, helping to curb the spread of multidrug-resistant enteric pathogens.

Material and Methods

Sample Collection and Processing

Sixty faecal samples were collected in sterile containers from patients at Al-Hussein Teaching Hospital, Thi-Qar, Iraq, between February and July 2020. Samples were transported immediately to the laboratory and refrigerated at 4 °C when processing was delayed. For isolation and identification of Escherichia coli, the samples were cultured on MacConkey agar (LAB-UK), blood agar (LAB-UK), and eosin methylene blue agar (LAB-UK), and incubated at 37 °C for 24 hours. Gram staining was performed to assess bacterial morphology, and colony characteristics such as hemolysis on blood agar and lactose fermentation on MacConkey agar were recorded. Species-level identification was confirmed using the API 20E (BioMérieux-Franch) biochemical testing system (Abdullah and Al-Taee, 2024)

Molecular Analysis

Genomic DNA was extracted from confirmed isolates using the Presto™ Mini gDNA Bacteria Kit following the manufacturer's protocol. Polymerase chain reaction (PCR) was performed to detect the sul1 gene using specific primers (Tabel 1), with a final reaction volume of 20 µl containing 5 µl of Master Mix, 1.5 µl of both forward and reverse primers, 3 µl of DNA template, and nuclease-free water. PCR amplicons were analyzed by agarose gel electrophoresis (1%–1.5%), prepared in 1XTBE buffer and supplemented with safety dye after cooling to 45–55 °C; reaction products mixed with loading dye were electrophoresed at 70 V for 45–60 minutes and visualized under ultraviolet illumination. Amplified sul1 gene products were subsequently forwarded to Macrogen (Korea) for DNA sequencing. Nucleotide sequences were analyzed using BLAST, aligned with BioEdit software, and subjected to phylogenetic analysis by the neighbor-joining method, with tree visualization performed using the iTOL platform (Zeadan *et al,.* 2022).

Primers: The primers used for gene amplification, that is give the sequences for *sul1* gene.

Table 1: *Lists The Primer Sequences Used for Gene Amplification*

Gene	Primer Sequences (5´-3´)	Product Size	Reference
sul1	F: TTCGGCATTCTGAATCTCAC, R: ATGATCTAACCCTCGGTCTC	822 bp	Van den Bogaard *et al.,* 2001

Ethical approval

Ethical approval for this research study has been obtained from Ethical Approval Committee, Ministry of Higher Education and Science Resarch, University of Warith Al-Anbiyaa College of Nursing, Iraq.

Results

The consistent presence of the *sul1* gene in all tested isolates highlights a common resistance mechanism in this bacterial population. This finding supports the study's conclusion on the widespread nature of sulfonamide resistance, driven by genetic elements associated with the *sul1* gene. This gel electrophoresis image provides visual confirmation of the molecular evidence supporting the study's findings on antibiotic resistance in *E. coli*.

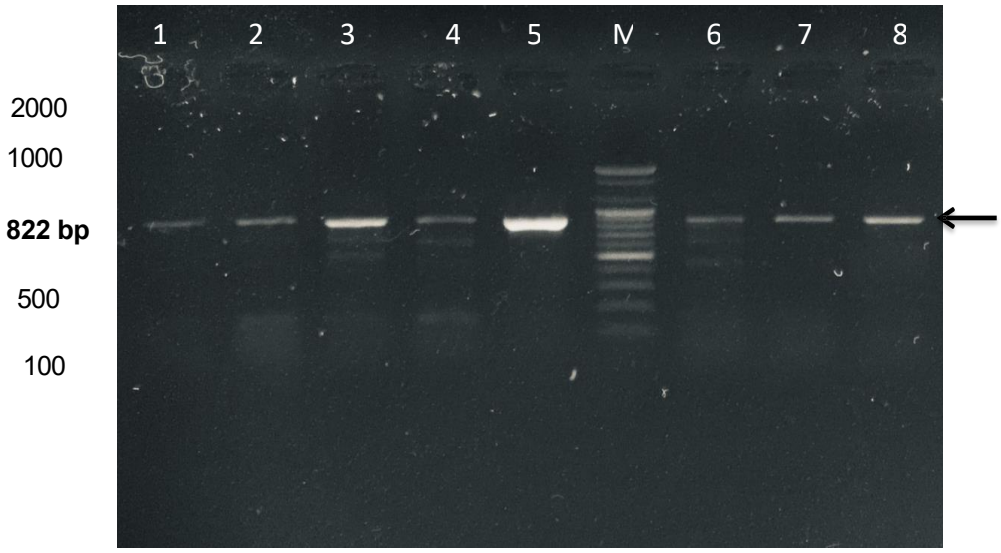

Figure 1: *Agarose Gel Electrophoresis of Sul1 Gene Amplification, Where M: Ladder, 1-8: Positive Results*

Results for sul1 Gene Analysis

Five samples were tested to amplify the sul*1* gene sequence in extrachromosomal plasmids. The *sul1* gene encodes sulfonamide synthase, conferring resistance to sulfonamide in *E. coli* . Sequencing and NCBI BLAST analysis verified high sequence identity, with ~99% similarity between most sequenced samples and the reference target sequences (GenBank accession CP05437.1). This comparison provided precise alignment and positional information for the PCR fragments (Fig. 2).

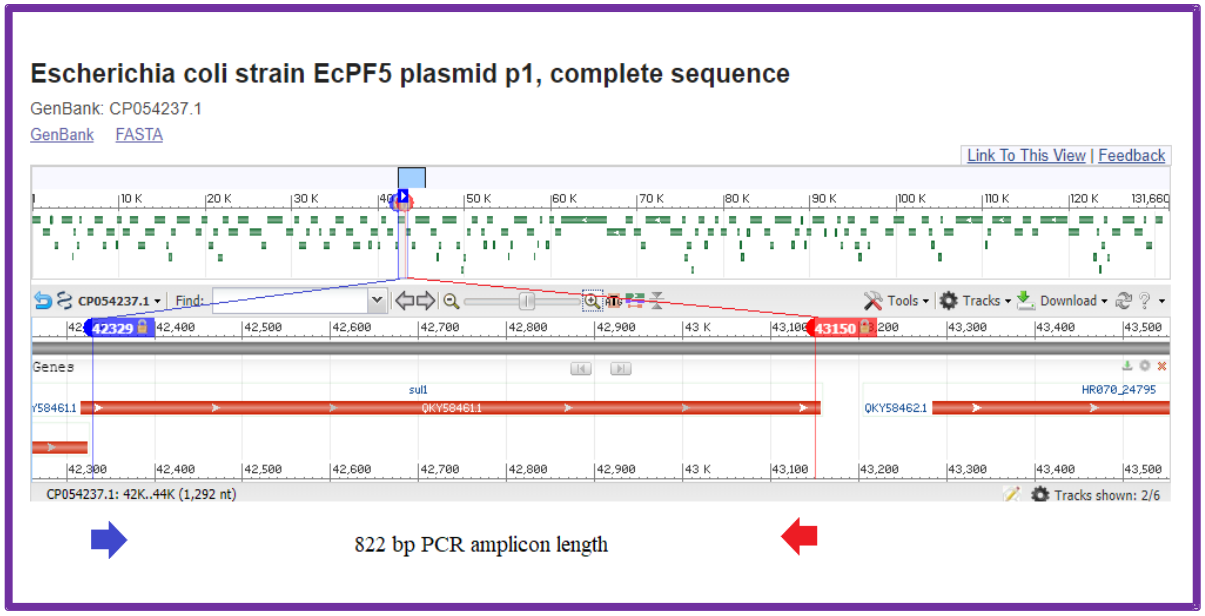

Figure 2: *The exact position of the retrieved 822 bp amplicon that partially covered a portion of the sul1 gene within the plasmid sequences (GenBank acc. no. CP05437.1). The cyan arrow refers to the starting point of this amplicon while the red arrow refers to its endpoint*

The 822 bp amplicons' sequence details were documented, including the locations of both forward and reverse primers (Table 2).

Table 2*: Sequence Positions and Length of the 822 Bp PCR Amplicon Within the Sul1 Gene Region in E. Coli Plasmids (Genbank Acc. CP05437.1), With Reverse and Forward Primer Locations Marked*

Amplicon	Reference locus sequences (5′ - 3′)	length
DNA sequences within the *sul1* genetic locus	CGGCATTCTGAATCTCACCGAGGACTCCTTCTTCGATGAGAGCCGGCGG CTAGACCCCGCCGGCGCTGTCACCGCGGCGATCGAAATGCTGCGAGTCGGAT CAGACGTCGTGGATGTCGGACCGGCCGCCAGCCATCCGGACGCGAGGCCTGT ATCGCCGGCCGATGAGATCAGACGTATTGCGCCGCTCTTAGACGCCCTGTCC GATCAGATGCACCGTGTTTCAATCGACAGCTTCCAACCGGAAACCCAGCGCT ATGCGCTCAAGCGCGGCGTGGGCTACCTGAACGATATCCAAGGATTTCCTGA CCCTGCGCTCTATCCCGATATTGCTGAGGCGGACTGCAGGCTGGTGGTTATG CACTCAGCGCAGCGGGATGGCATCGCCACCCGCACCGGTCACCTTCGACCCG AAGACGCGCTCGACGAGATTGTGCGGTTCTTCGAGGCGCGGGTTTCCGCCTT GCGACGGAGCGGGGTCGCTGCCGACCGGCTCATCCTCGATCCGGGGATGGGA TTTTTCTTGAGCCCCGCACCGGAAACATCGCTGCACGTGCTGTCGAACCTTC AAAAGCTGAAGTCGGCGTTGGGGCTTCCGCTATTGGTCTCGGTGTCGCGGAA ATCCTTCTTGGGCGCCACCGTTGGCCTTCCTGTAAAGGATCTGGGTCCAGCG AGCCTTGCGGCGGAACTTCACGCGATCGGCAATGGCGCTGACTACGTCCGCA CCCACGCGCCTGGAGATCTGCGAAGCGCAATCACCTTCTCGGAAACCCTCGC GAAATTTCGCAGTCGCGACGCCAGAGACCGAGGGTTAGATCAT	822 bp

Refers to the forward primer sequences (placed in a reverse direction)

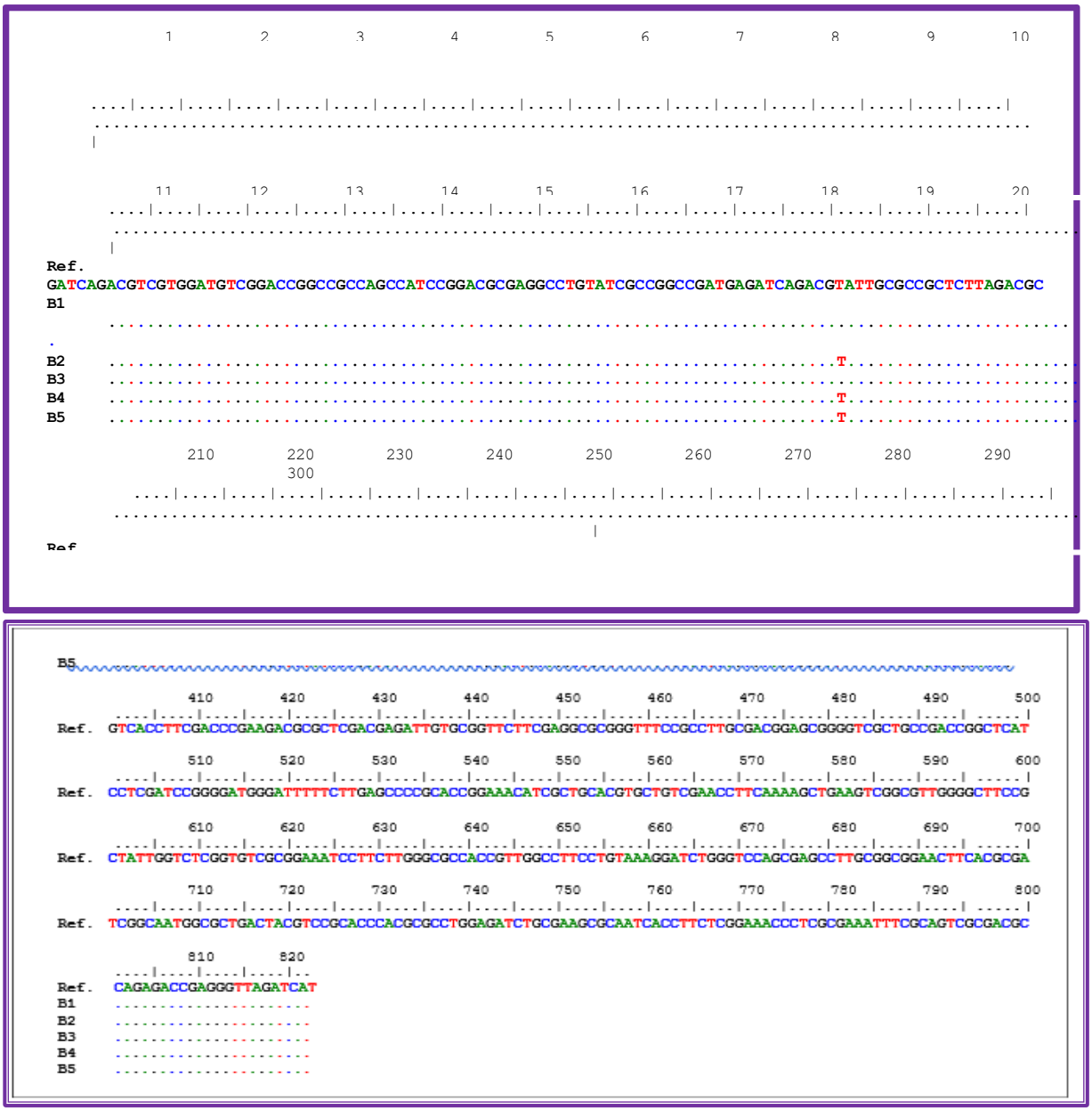

Figure 3 Alignment of DNA sequences for five samples against the reference sequence for the 822 bp sul1 gene fragment. “Ref” indicates the reference sequence, and “B” numbers correspond to sample IDs.

Notably, nucleotide substitutions were identified in samples B2, B4, and B5. These variants were confirmed through sequence chromatograms (Fig. 4).

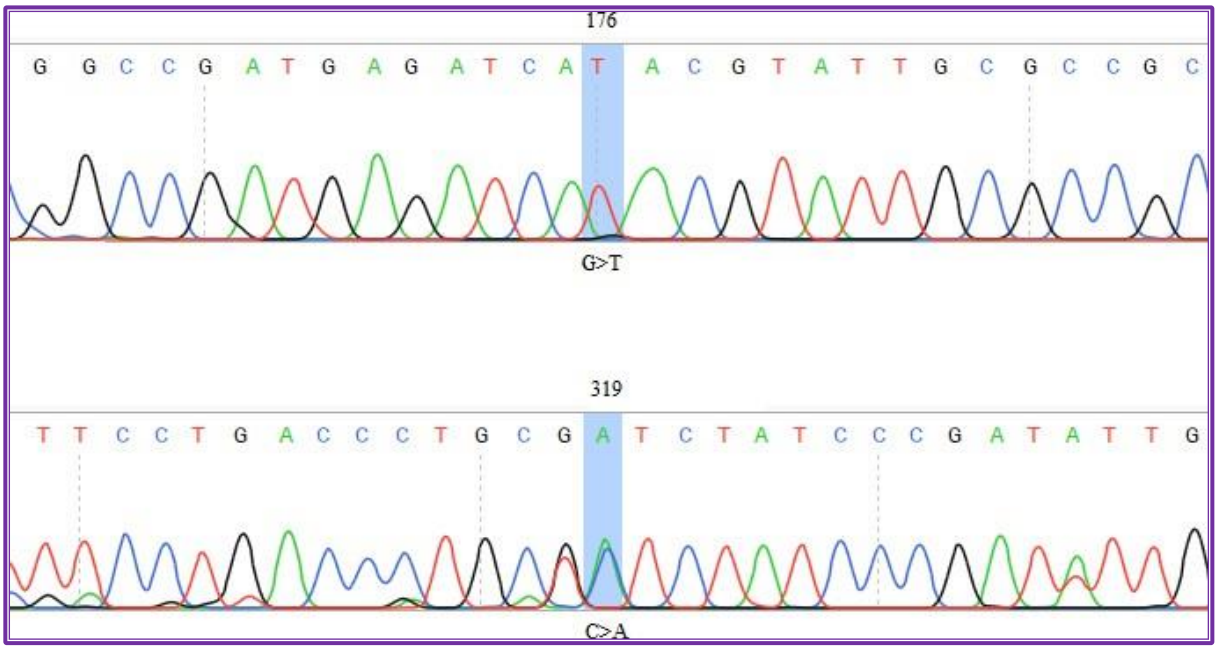

Figure 4: *Chromatogram showing mutations within the 822 bp amplicons for the tetR gene, with substitution mutations marked by ">".*

To determine the impact of these mutations, all nucleotide sequences were translated to amino acids using the Expasy translate tool. Both mutations were identified as silent, with no effect on the resulting protein (Fig. 5). A phylogenetic tree was generated based on these nucleotide variations, comparing B1 to B5 samples with related sequences. The tree included 134 aligned sequences, revealing high similarity among samples and grouping all *E. coli* sequences together (Fig. 5).

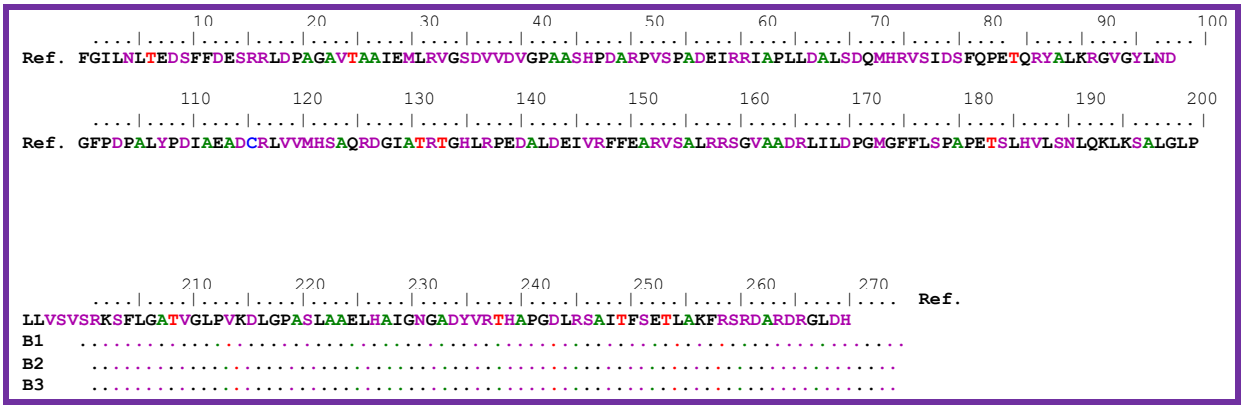

Figure 5: *Alignment of amino acid residues showing the silent mutations in the Sul1 gene*

Table 3: *The Positions of The Amino Acid Substitutions are Highlighted According to Their Corresponding Position Within the Amplified Locus. The Highlighted Colors Refer to the Amino Acid Substitutions*

Sample No.	Native	Allele	Position in the PCR fragment	Position in the reference genome	Amino acid position	Type of mutation
B2, B4, B5	G	T	176	42504	63 Arg	One Silent mutation (63 Arg=)
B1, B2	C	A	319	42647	111 Leu	One Silent mutation (111 Leu=)

V. The symbol "B" followed by a number refers to the investigated bacterial sample numbers

A comprehensive phylogenetic tree has generated in the present study, which is based on the observed nucleic acid variations. This phylogenetic tree contained B1 to B5 samples alongside with other relative DNA sequences. A total number of the aligned nucleic acid sequences in this comprehensive tree is 134 (figure 5). High homology has been observed among the investigated samples with many strains belonging to *E. coli*. Within this tree, addition organism was placed as an outgroup alongside with the main incorporated *E. coli* sequences. All the investigated sequences are obviously positioned in the *E. coli* clades and no apparent deviation from *E. coli* is observed for any one of these clinical isolates.

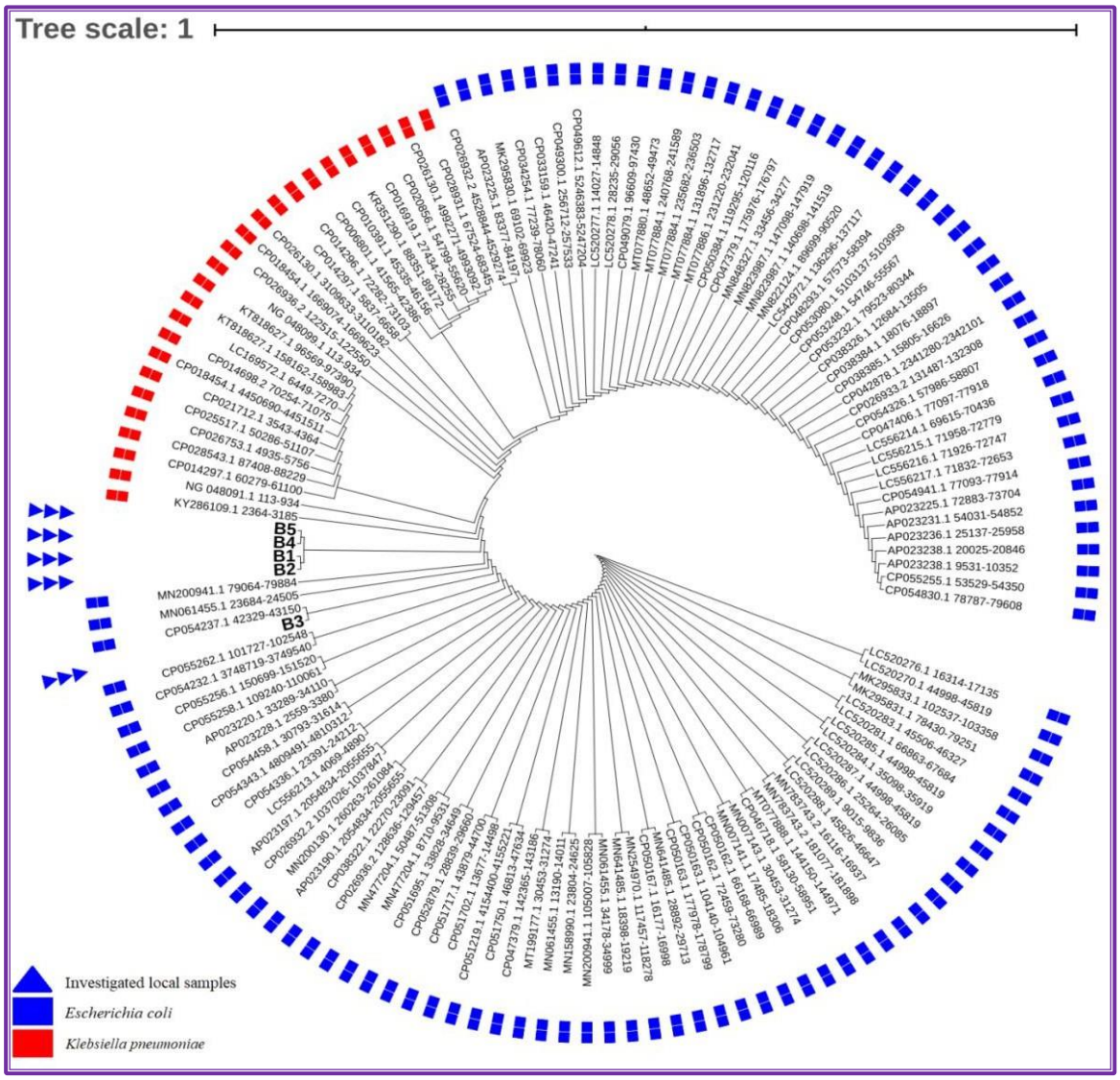

Figure 6 *: Phylogenetic tree showing the relationship of sul1 gene variants in local E. coli samples (cyan triangles) with other sequences from NCBI. Each sample code is labeled as "B#".*

Discussion

This study investigated the prevalence of the *sul1* gene among *Escherichia coli* isolates obtained from diarrheal cases, focusing on the molecular mechanisms of sulfonamide resistance. The findings underscore the significant role of *sul1* as a genetic determinant for antibiotic resistance, revealing high homology with previously documented sequences. The *sul1* gene, found within mobile genetic elements like plasmids, contributes to Multidrug Resistance (MDR) in *E. coli*, making infections harder to manage, particularly in paediatric and immunocompromised populations (Poey *et al*., 2019).

The successful amplification and sequencing of the *sul1* gene in this study align with previous research that emphasizes the widespread distribution of sulfonamide resistance genes among enteric bacteria. Consistent with studies by (Da Lage *et al.,* 2019), the high identity scores observed in BLAST analysis confirm the conservation of *sul1* among clinical isolates, suggesting robust and stable propagation of this resistance gene across *E. coli* population stable nucleotide mutations were observed in samples B2, B4, and B5. Although these mutations were identified, the amino acid sequences remained unaffected, indicating silent mutations that did not alter the protein's function. This finding supports prior research suggesting that some genetic variations in resistance genes do not necessarily alter their antibiotic resistance properties but may play a role in the evolutionary adaptation of *E. coli* strains under antibiotic pressure (Lamichhane *et al*., 2024; Baseri,*et al.*, 2021).

The phylogenetic tree reveals a close evolutionary relationship among *sul1* -positive *E. coli* strains, with no significant divergence within the *E. coli* clades (Masood *et al*., 2024). This result highlights the genetic stability of the *sul1* gene across different geographical locations, supporting its persistence within the bacterial population. As demonstrated by studies such as those conducted by Algarni *et al.,* 2022. Such homogeneity suggests that the gene's horizontal transfer is facilitated by integrons and other mobile genetic elements, underscoring the challenge of containing antibiotic resistance within bacterial communities (Kumavath, 2025).

These findings have implications for clinical and public health strategies. The persistence and spread of *sul1* -mediated resistance in *E. coli* necessitate enhanced surveillance and stringent antibiotic stewardship practices, especially in settings where diarrhea-related infections are prevalent (Jiang *et al.*, 2019). Limiting unnecessary use of antibiotics, particularly sulfonamides, and improving diagnostic capabilities for rapid detection of resistant strains may help curb the further spread of MDR bacteria. The integration of molecular studies like this one into routine surveillance programs could also help monitor emerging resistance patterns, providing a framework for developing targeted treatment strategies (Hemati *et al*., 2024).

In summary, this study emphasizes the clinical importance of the *sul1* gene in sulfonamide resistance among diarrheagenic *E. coli* and highlights the role of molecular epidemiology in understanding the transmission dynamics of resistance genes. The high prevalence and stable genetic characteristics of *sul1* among *E. coli* isolates call for sustained efforts to address the spread of MDR pathogens and reinforce the importance of antibiotic stewardship in managing infectious diseases.

This study confirms the high prevalence of the *sul1* gene (detected in 100% of isolates via 822 bp amplicons) in diarrheagenic *E. coli* strains, underscoring its role in sulfonamide resistance and Multidrug Resistance (MDR) dissemination. The genetic homogeneity observed in phylogenetic analysis—clustering all isolates within *E. coli* clades—aligns with prior reports of *sul1* stability across geographical regions (Royer *et al.* 2021; Masood *et al*., 2024). The use of primers from Van den Bogaard *et al*. (2001) validated the specificity of *sul1* amplification, reinforcing methodological consistency with established protocols.

Silent mutations (e.g., nucleotide substitutions at positions 176 and 319) were identified in 60% of samples (B2, B4, B5, B1, B2), corroborating findings by Lobinska *et al.,* (2023) that such mutations may facilitate evolutionary adaptation without altering phenotypic resistance. This genetic plasticity, combined with *sul1*'s association with integrons (Baharoglu,, Garriss, & Mazel, 2013), highlights its persistence in bacterial populations, particularly in low-resource settings where diarrheal infections are endemic (Baseri, *et al.* 2021; Pato and Brown, 1963).

The study's limitations include a small sample size (n=60) and regional focus, which may limit generalizability. However, the 99% sequence similarity to global reference strains (e.g., GenBank CP05437.1) emphasizes the gene's widespread conservation. These findings align with Algarni *et al*. (2022), who noted similar resistance dynamics in *Salmonella*, and support urgent calls for antibiotic stewardship.

Conclusion

This study underscores the significant role of the *sul1 gene* in mediating sulfonamide resistance in *Escherichia coli* strains isolated from diarrheal cases. The high prevalence of *sul1* -positive isolates suggests that sulfonamide resistance is widespread, potentially due to the gene's association with mobile genetic elements that facilitate horizontal gene transfer. Phylogenetic analysis revealed close genetic relationships among these isolates, indicating the persistence and propagation of this resistance gene within local bacterial populations. The findings highlight the importance of continuous molecular surveillance of resistance genes in pathogenic *E. coli* to inform treatment protocols and mitigate multidrug-resistant infections.

Recommendation

Strengthen Surveillance Programs: Implement routine genetic screening of *E.coli* isolates in healthcare and community settings to monitor *sul1* gene prevalence and track emerging resistance patterns.

Promote Prudent Antibiotic Use: Encourage rational antibiotic use, particularly of sulfonamides, in clinical and veterinary settings to limit the spread of resistance.

Enhance Infection Control Measures: Focus on improving sanitation and hygiene practices in high-risk areas to reduce the transmission of resistant *E. coli* strains, especially among vulnerable populations.

Invest in Further Research: Support studies that explore additional resistance mechanisms in *E. coli* to develop alternative therapeutic strategies and identify potential targets for new antimicrobials.

These steps could help reduce the burden of antibiotic-resistant E. *coli* infections, particularly in resource-limited areas, and support global antimicrobial stewardship efforts.

Conflict of Interest

The authors declare that they have no competing interests.

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