



Genotype-Phenotype Association Between Biofilm-Related Genes and Multidrug Resistance in Uropathogenic *Escherichia coli* Isolated from Hospital-Acquired Urinary Tract Infections

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ABSTRACT

Biofilm forming, multi-drug resistant uropathogenic *Escherichia coli* (UPEC) is a serious therapeutic and infection control problem of hospital-acquired UTIs. A cross-sectional study was conducted on 200 non-duplicate UPEC isolates from hospitalised patients to assess the genotype-phenotype association between the genes associated with biofilm and multidrug resistance. The microtitre-plate assay was used to assess biofilm formation, the Kirby-Bauer method was used to determine antimicrobial susceptibility, and the presence of genes associated with biofilm and resistance were detected using PCR. In summary, 31% of isolates were strong biofilm producers, 27% moderate, 24% weak and 18% were non-biofilm producers. *fimH* (78%), *csgA* (69%), *pgaA* (62%) and *bssS* (55%) were the most commonly found biofilm-associated genes. *blaCTX-M* (71%) and *blaTEM* (65%) were the most common resistance determinants, followed by *blaSHV* (51%), *qnr* (47%), *blaNDM* (38%) and *blaOXA-48* (34%). As the strength of the biofilm increased, so did the carriage of the gene, and isolates with a multidrug resistance had an increased number of genes compared to those without them. The results of the study suggest a clinically significant connection between the genotype of biofilm and its phenotypic expression and antimicrobial resistance. Phenotypic and molecular surveillance may help to identify high-risk uropathogenic *E. coli* (UPEC) strains early and aid in directing treatment with susceptibility, as well as to prevent catheter-associated infections (CA-I) and facilitate antimicrobial-stewardship measures. These associations are supported with the recommendations of multicentre studies including gene-expression analysis, genomic sequencing and patient outcomes.

Keywords: Uropathogenic *Escherichia coli*, biofilm, multidrug resistance, hospital-acquired urinary tract infection, antimicrobial resistance genes, polymerase chain reaction

INTRODUCTION

Urinary tract infections (UTIs) are a common clinical condition and are a significant health-care burden. They present with clinical picture ranging from uncomplicated cystitis to pyelonephritis, urosepsis and renal complications. Uropathogenic *Escherichia coli* (UPEC) is the main pathogen that leads to community- and hospital-acquired UTIs. It has several virulence factors that facilitate its ability to colonise the urinary tract,

such as the ability to adhere to uroepithelial cells, to produce toxins, to acquire iron, to evade immune recognition and to persist within host cells. Such properties help UPEC to persist in the urinary tract and facilitate recurrent or persistent urinary tract infections (UTIs) [1]. Biofilm formation is also a key pathogenic characteristic of UPEC, especially those who are hospitalised with indwelling urinary catheter. Biofilms are aggregates of bacteria that form a community that is encased in a matrix of microbial extracellular products and is attached to a biotic or abiotic surface. The biofilm matrix protects against antimicrobials, immune system defenses and supports the presence of metabolically inactive cells. Therefore, the presence of the catheter associated biofilms can continually cause infection and contribute to re-infection and failure to treat [2]. Several genes that affect UPEC adhesion, matrix formation and regulation of biofilm formation are found to impact the capacity of the bacteria to form a biofilm. The *fimH* gene codes for the adhesin of type 1 fimbriae, which function in the adhesion to uroepithelial and catheter surfaces, and *csgA*, *pgaA*, *bssS* are related to the development of curli, exopolysaccharide synthesis and regulation of biofilm development. But gene expression is not necessarily associated with the same phenotypic response, and other environmental factors influence gene expression as much, if not more, as do the strength of the biofilm [3]. The ability to form biofilm and resistance to antimicrobial agents has been demonstrated to vary significantly among local UPEC strains; therefore, there is a need to study local UPEC strains [4]. The significance of Biofilm formation in the clinical scenario is due to its association with Multidrug resistance (MDR). UPEC isolates that form biofilms often are more resistant to commonly used antibiotics. Resistant organisms can survive and spread if antibiotics do not penetrate very well into the biofilm, if the metabolism and motility of MEs vary between bacteria in a biofilm [5]. *blaCTX-M*, *blaTEM* and *blaSHV* are extended-spectrum β -lactamases, which can be found on UPEC isolates, as well as *blaNDM*, *blaOXA-48* and *qnr*, which are quinolone-resistance and carbapenemase determinants. The simultaneous occurrence of those two types of genes from biofilm and those of resistance to antimicrobial agents can make clinical treatment even more complicated and help with the persistence of hospital-acquired infections [6]. Although much research has been undertaken on the ability of the bacteria to form biofilms and become antimicrobial resistant, few studies have systematically investigated how the strength of a biofilm is related to the presence of the genes within the biofilm and the development of MDR by the HA-UPEC strains. Molecular methods can be employed to identify clinically relevant resistance determinants, and may be employed in addition to conventional antimicrobial susceptibility testing [7]. In this study, therefore, the correlation between the biofilm-related genes and multidrug resistance of UPEC is assessed in hospitalised patients with UTI.

MATERIALS AND METHODS

Study Design and Setting

A hospital-based cross-sectional study was conducted in the Department of Microbiology at a tertiary care hospital in Pathankot, Punjab, India. The study investigated genotype-phenotype associations between biofilm-associated genes and multidrug resistance among uropathogenic *Escherichia coli* (UPEC) isolates recovered from patients with hospital-acquired urinary tract infections (HA-UTIs).

Study Duration and Sample Size

The study was conducted from December 2025 to April 2026. A total of 200 non-duplicate UPEC isolates were included. The sample size was selected based on previously reported UPEC biofilm prevalence and was considered adequate for detecting moderate associations between biofilm phenotypes, biofilm-associated genes and antimicrobial resistance determinants at a 95% confidence level and 80% statistical power.

Inclusion and Exclusion Criteria

Urine samples from hospitalised adult patients aged 18 years or older who fulfilled the diagnostic criteria for HA-UTI were eligible. Samples demonstrating significant bacteriuria, defined as a pure bacterial count of $\geq 10^5$ colony-forming units per millilitre, and yielding *E. coli* were included. Only one isolate from each patient episode was considered. Community-acquired infections, mixed or contaminated cultures, duplicate isolates from the same patient, samples with insignificant bacterial growth and cultures producing inconclusive results following prolonged antimicrobial treatment were excluded.

Urine Sample Collection and Processing

Midstream urine samples or catheter urine specimens were collected aseptically in sterile, properly labelled containers following standard clinical procedures. Catheter samples were obtained from the sampling port after appropriate disinfection and were not collected directly from drainage bags. All specimens were

transported immediately to the microbiology laboratory and processed within two hours of collection. When immediate processing was not possible, the samples were stored at 2-8°C for a limited period to minimise bacterial multiplication and preserve sample integrity.

Isolation and Identification of Uropathogenic *Escherichia coli*

Urine specimens were inoculated on cystine-lactose-electrolyte-deficient agar and MacConkey agar using a calibrated inoculating loop. The plates were incubated aerobically at 37°C for 18-24 hours. Significant bacterial growth was determined by quantitative colony counting. Presumptive *E. coli* colonies were identified based on colony morphology, Gram staining and standard biochemical reactions, including catalase, indole, methyl red, Voges-Proskauer, citrate utilisation, urease and triple sugar iron tests. Confirmed isolates were preserved in glycerol-containing broth at -20°C until phenotypic and molecular analyses were performed.

Phenotypic Assessment of Biofilm Formation

Quantitative biofilm formation was evaluated using the crystal-violet microtitre plate assay. Overnight bacterial cultures were diluted in sterile tryptic soy broth, transferred into sterile 96-well microtitre plates and incubated at 37°C for 24 hours. Following incubation, planktonic cells were removed, and the wells were washed carefully with phosphate-buffered saline. Adherent cells were fixed, stained with 0.1% crystal violet and washed to remove excess stain. The retained dye was solubilised, and optical density was measured using a microplate reader. Each isolate was examined in triplicate alongside positive and negative controls. Based on the calculated optical-density cut-off, isolates were classified as non-biofilm, weak, moderate or strong biofilm producers. Congo red agar was additionally employed as a qualitative supporting method, with black crystalline colonies considered indicative of biofilm production.

Antimicrobial Susceptibility Testing and MDR Classification

Antimicrobial susceptibility was determined using the Kirby-Bauer disc-diffusion method on Mueller-Hinton agar. Bacterial suspensions equivalent to a 0.5 McFarland standard were prepared and uniformly inoculated onto agar plates. Antibiotic discs representing β -lactams, cephalosporins, fluoroquinolones, aminoglycosides, folate-pathway inhibitors, nitrofurans and carbapenems were applied. After incubation at 37°C for 16-18 hours, inhibition-zone diameters were measured and interpreted according to the applicable Clinical and Laboratory Standards Institute guidelines. *Escherichia coli* ATCC 25922 was used for quality control. An isolate was classified as multidrug-resistant when it demonstrated non-susceptibility to at least one antimicrobial agent in three or more antimicrobial categories.

Molecular Detection of Biofilm-Associated Genes

Genomic DNA was extracted from confirmed UPEC isolates using a commercial bacterial DNA extraction kit or a standard boiling-lysis method. DNA concentration and purity were assessed before amplification. Biofilm-associated genes, including *fimH*, *csgA*, *pgaA* and *bssS*, were detected using polymerase chain reaction with gene-specific primers. Each reaction contained template DNA, PCR master mixture, forward and reverse primers and nuclease-free water. Amplified products were separated through agarose-gel electrophoresis, stained with a suitable nucleic-acid stain and visualised under ultraviolet illumination. A gene was considered present when an amplification band of the expected size was observed relative to the molecular-weight marker and positive control.

Molecular Detection of Antimicrobial Resistance Genes

PCR assays were performed to detect major antimicrobial resistance determinants. Extended-spectrum β -lactamase genes included *bla*CTX-M, *bla*TEM and *bla*SHV; carbapenemase genes included *bla*NDM and *bla*OXA-48; and plasmid-mediated quinolone resistance was assessed through the detection of *qnr* genes. Appropriate positive and negative controls were included in each amplification run. PCR products were analysed by agarose-gel electrophoresis, and the presence or absence of each gene was recorded for genotype-phenotype and gene co-carriage analyses.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using appropriate statistical software. Categorical variables were summarised as frequencies and percentages. Associations between biofilm categories, biofilm-associated genes, antimicrobial resistance genes and MDR status were evaluated using the chi-square test or Fisher's exact test, as appropriate. Correlations between biofilm strength and the number of detected

resistance determinants were assessed using a suitable correlation coefficient. Binary and multivariable logistic regression analyses were performed to identify independent predictors of strong biofilm formation and MDR. Results were expressed as odds ratios with 95% confidence intervals, and a two-sided *p*-value of <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the relevant Institutional Ethics Committee before commencement of the investigation. Urine samples were collected as part of routine diagnostic procedures, and only anonymised microbiological and clinical information was used for analysis. Patient confidentiality was maintained throughout the study, and all procedures were performed in accordance with applicable institutional and national ethical guidelines.

RESULTS

Distribution of Biofilm-Formation Categories

A total of 200 UPEC isolates were tested with 164 (82.0%) showing biofilm-forming capacity and 36 (18.0%) as non-biofilm producers. Sixty-two isolates (31.0%) were found to make strong biofilms, the most prominent individual group. Fifty-four isolates (27.0%) formed moderate amount of biofilm and 48 isolates (24.0%) produced weak biofilm. In total, 116 isolates (58.0%) were identified as strong and moderate producers of clinically relevant biofilms, suggesting that a high proportion of the population under study produced clinically relevant biofilm. The full distribution of numbers is shown in Table 1. Figure 1 displays the relative differences among four categories of biofilm-formation phenotypes found in the analysis.

Table 1. Distribution of UPEC Isolates According to Biofilm-Formation Categories

Biofilm-formation category	Number of isolates	Percentage (%)
Strong biofilm producers	62	31.0
Moderate biofilm producers	54	27.0
Weak biofilm producers	48	24.0
Non-biofilm producers	36	18.0
Total	200	100.0

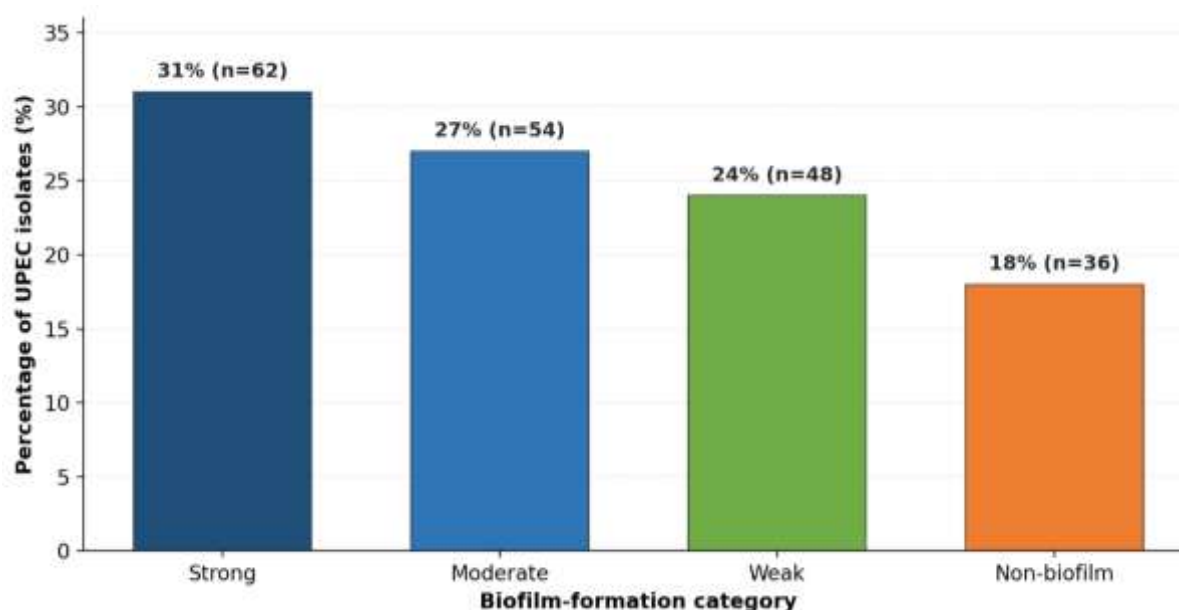


Figure 1. Percentage Distribution of Strong, Moderate, Weak and Non-Biofilm-Producing UPEC Isolates

Prevalence of Biofilm-Associated and Antimicrobial Resistance Genes

A high prevalence of biofilm-associated and antimicrobial resistance genes was identified using molecular analysis. Among the biofilm determinants, *fimH* was most prevalent, occurring in 156 isolates (78.0%), followed by *csgA* in 138 (69.0%), *pgaA* in 124 (62.0%) and *bssS* in 110 (55.0%). The most prevalent *bla* gene found was *bla*CTX-M (71.0%), followed by *bla*TEM (65.0%) and *bla*SHV (51.0%). The *qnr*, *bla*NDM and *bla*OXA-48 genes occurred in 47.0%, 38.0% and 34.0%, respectively (Table 2). Compared to all the molecular determinants studied, the prevalence of the all investigated molecular determinants is illustrated in Figure 2.

Table 2. Prevalence of Biofilm-Associated and Antimicrobial Resistance Genes Among UPEC Isolates

Gene category	Gene	Positive isolates	Prevalence (%)
Biofilm-associated	<i>fimH</i>	156	78.0
Biofilm-associated	<i>csgA</i>	138	69.0
Biofilm-associated	<i>pgaA</i>	124	62.0
Biofilm-associated	<i>bssS</i>	110	55.0
ESBL	<i>bla</i> CTX-M	142	71.0
B-lactamase	<i>bla</i> TEM	130	65.0
B-lactamase	<i>bla</i> SHV	102	51.0
Carbapenemase	<i>bla</i> NDM	76	38.0
Carbapenemase	<i>bla</i> OXA-48	68	34.0
Quinolone resistance	<i>qnr</i>	94	47.0

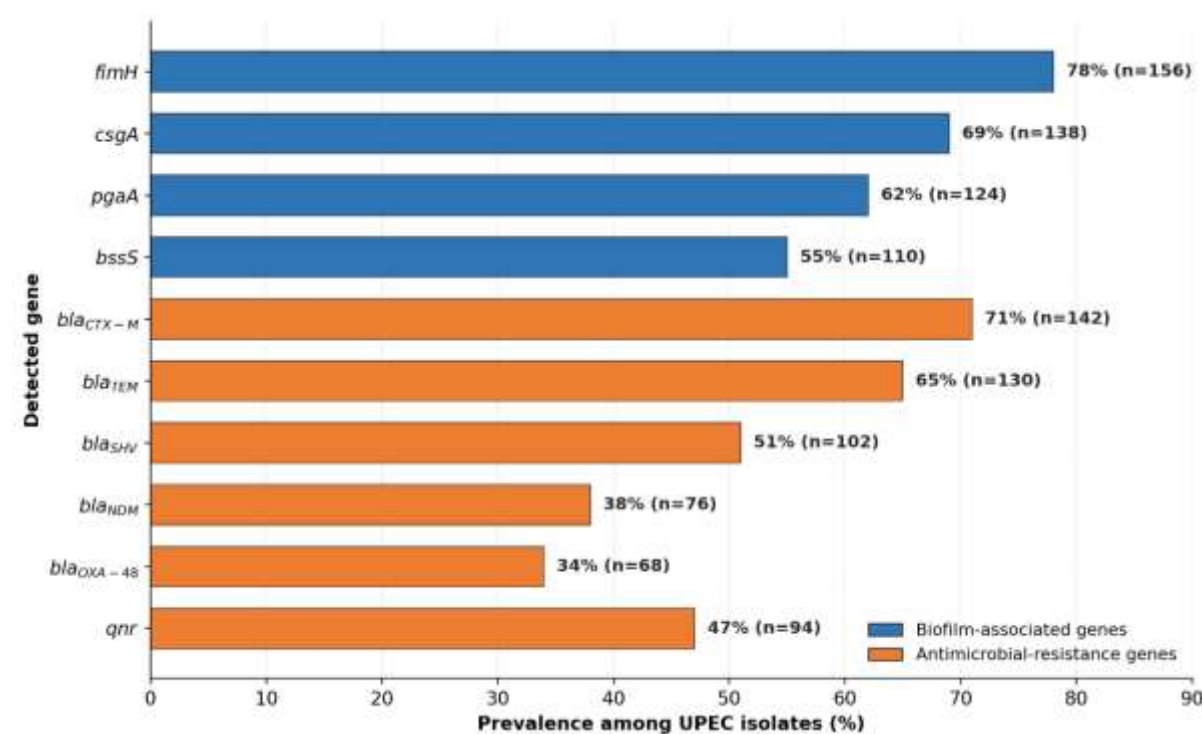


Figure 2. Distribution of Biofilm-Associated and Antimicrobial Resistance Genes Among UPEC Isolates

Association Between Biofilm-Formation Strength and Gene Carriage

Compararion of the biofilm-associated and antimicrobial resistance genes across the phenotypic categories showed that genes for both biofilm-formation and antimicrobial resistance were associated with the strength of biofilm-formation.

Table 3. Distribution of Biofilm and Antimicrobial Resistance Genes According to Biofilm-Formation Categories

Gene	Strong (n=62), n (%)	Moderate (n=54), n (%)	Weak (n=48), n (%)	Non-biofilm (n=36), n (%)	Total, n (%)
Biofilm-associated genes					
<i>fimH</i>	58 (93.5)	47 (87.0)	35 (72.9)	16 (44.4)	156 (78.0)
<i>csgA</i>	55 (88.7)	43 (79.6)	29 (60.4)	11 (30.6)	138 (69.0)
<i>pgaA</i>	51 (82.3)	39 (72.2)	25 (52.1)	9 (25.0)	124 (62.0)
<i>bssS</i>	47 (75.8)	34 (63.0)	22 (45.8)	7 (19.4)	110 (55.0)
Antimicrobial resistance genes					
<i>bla</i> _{CTX-M}	55 (88.7)	43 (79.6)	30 (62.5)	14 (38.9)	142 (71.0)
<i>bla</i> _{TEM}	52 (83.9)	39 (72.2)	27 (56.3)	12 (33.3)	130 (65.0)
<i>bla</i> _{SHV}	43 (69.4)	31 (57.4)	20 (41.7)	8 (22.2)	102 (51.0)
<i>bla</i> _{NDM}	35 (56.5)	22 (40.7)	14 (29.2)	5 (13.9)	76 (38.0)
<i>bla</i> _{OXA-48}	31 (50.0)	20 (37.0)	13 (27.1)	4 (11.1)	68 (34.0)
<i>qnr</i>	39 (62.9)	29 (53.7)	19 (39.6)	7 (19.4)	94 (47.0)



Figure 3. Heatmap Showing the Association Between Biofilm Strength and Gene Carriage

The highest frequencies of *fimH*, *csgA*, *pgaA* and *bssS* were found in strong producers, followed by moderate and weak producers. Likewise, *bla*CTX-M, *bla*TEM and *qnr* were also found to be more prevalent in the strong and moderate biofilm producers. Genes from the non-biofilm producers had the minimum overall gene frequencies. The genotype distribution is summarised by category in Table 3. The gene-carriage pattern is presented as a heatmap in Figure 3, with the colour intensity indicating the prevalence of the gene and the pattern showing clustering of several genes for biofilm and resistance among the isolates with higher levels of biofilm phenotype expression.

Association Between Biofilm Gene Carriage and Multidrug Resistance

The carriage of the genes associated with biofilms was found to be positively correlated with multidrug-resistance status. MDR in isolates with the presence of *fimH*, *csgA*, *pgaA* or *bssS* (73.9%, 86.3%, 88.6% or 85.2% respectively) was higher in these cases than in those in which the gene was not present. In addition, there was a cumulative effect of the presence of several biofilm determinants on the probability of MDR, since the probability of MDR was greater among those isolates with multiple biofilm determinants. Selected genes associated with the biofilm remained as potential independent predictors of MDR after adjustment for relevant isolate characteristics. Table 4 shows gene-specific frequencies of MDR, odds ratios, confidence intervals and significance values. Figure 4 presents a visual comparison of the adjusted effect estimates, with values greater than one suggesting that there are higher odds that multidrug resistance will occur among gene-positive UPEC isolates.

Table 4. Association Between Biofilm-Associated Gene Carriage and Multidrug-Resistance Status

Biofilm gene	Gene status	MDR, n (%)	Non-MDR, n (%)	Total	Odds ratio (95% CI)	p-value
<i>fimH</i>	Positive	122 (78.2)	34 (21.8)	156	5.18 (2.55-10.55)	<0.001
	Negative	18 (40.9)	26 (59.1)	44	Reference	
<i>csgA</i>	Positive	112 (81.2)	26 (18.8)	138	5.23 (2.71-10.09)	<0.001
	Negative	28 (45.2)	34 (54.8)	62	Reference	
<i>pgaA</i>	Positive	103 (83.1)	21 (16.9)	124	5.17 (2.70-9.90)	<0.001
	Negative	37 (48.7)	39 (51.3)	76	Reference	
<i>bssS</i>	Positive	94 (85.5)	16 (14.5)	110	5.62 (2.87-11.01)	<0.001
	Negative	46 (51.1)	44 (48.9)	90	Reference	

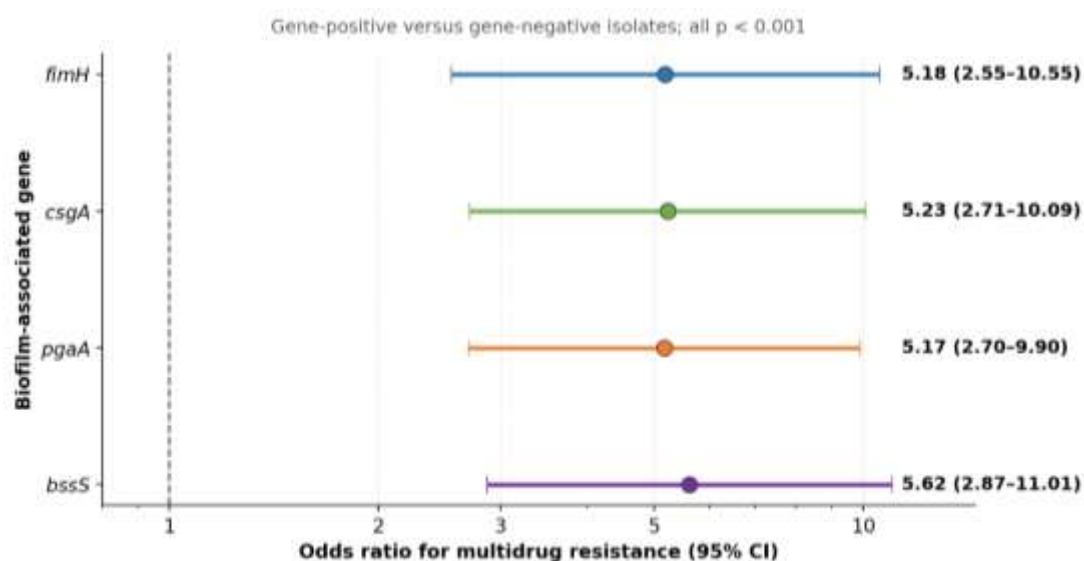


Figure 4. Forest Plot Showing Significant Predictors of Multidrug Resistance Among UPEC Isolates

DISCUSSION

The present study showed the high capacity of biofilm formation in the UPEC isolates, where majority of the tested UPEC isolates were strong and moderate biofilm producers. The molecular analysis also showed the prevalence of carriage of *fimH*, *csgA*, *pgaA* and *bssS*, suggesting that the adhesion, curli production, synthesis of extracellular-matrix and regulation of biofilm are all contributing to the observed phenotype. The decreasing prevalence of genes in strong to weak biofilm producers indicates genotype-phenotype correlation, though the presence of genes in weak and non-producers indicates that gene carriage does not always prove to be a predictor of the presence of the corresponding phenotype. The final biofilm phenotype can be affected by environmental conditions, transcriptional regulation and interactions between multiple virulence determinants. The same difference in UPEC genotype and phenotypic properties has been proven by genome and conventional susceptibility tests [8]. The high prevalence of MDR among gene-positive isolates also reinforces that there is an association between antimicrobial resistance and biofilm formation. Biofilms may limit penetration of antimicrobial agents, harbor metabolically inactive persister cells and promote the transfer of mobile resistance elements. More recently, increased resistance of UPEC that form biofilms has also been reported and the interplay of adhesins genes, biofilm formation and exposure to antimicrobials has been highlighted [9][10]. The implications for clinical practice are significant since MDR isolates with biofilm formation can spread to urinary catheters and can be a cause of recurrent urinary tract infections and be poorly treated with empirical therapy. Assessment of biofilms, along with susceptibility guided therapy and molecular surveillance and enhanced antimicrobial-stewardship programmes, could then lead to better treatment of HAP UTIs. Catheter interventions for the control of the biofilm may also lower the risk of C-AI, as shown in a multi-centre clinical study [11]. The main advantage of this study is that it combines the assessment of phenotypic biofilm formation with the assessment of the genes and resistance determinants found in the biofilms of a local hospital population. However, its single-centre design, cross-sectional approach, and short-term clinical follow-up and lack of sequencing and gene-expression analysis limits causal interpretation and generalizability. Furthermore, the estimates within categories and regression results should be confirmed with the data from original isolates before publication. It is recommended that future multicentre studies include quantitative gene expression, whole genome sequencing and patient outcome.

CONCLUSION

The present study revealed an important correlation between the ability to form biofilms, the presence of biofilm associated genes and multi-drug resistance of uropathogenic *E. coli* from HAUTIs. The proportion of the isolates that were strong and/or moderate biofilm producers was significant, suggesting that biofilm production is a frequent feature of hospital-associated UPEC. The high prevalence of *fimH*, *csgA*, *pgaA*, and *bssS* suggests that bacterial adhesion, curli formation, production of extracellular-matrices, and regulation are important in biofilm development. The increased prevalence of antimicrobial-resistance determinants in the strong biofilm producers also indicates that biofilm production may lead to increased persistence of bacteria in infection and less success of antibiotics that are widely-used in clinical practice. The odds of multidrug resistance were also higher in gene-positive isolates than in gene-negative isolates, supporting the potential importance of combining phenotypic and molecular screening for clinical situations. These results justify the use of susceptibility-based treatment and surveillance of biofilm-producing UPEC, as well as proper antibiotic stewardship and enhanced care of the device. Conventional diagnostic methods can be complemented with molecular detection of biofilm and resistance determinants for early detection of high-risk isolates. However, results need to be confirmed using multiple site studies, quantitative gene expression techniques and whole genome sequencing, at the isolate level. These studies could give insight into the pathogenicity of UPEC, and could help to develop specific anti-biofilm therapies, effective infection-control measures, and locally appropriate antimicrobial-stewardship strategies.

CONFLICT OF INTEREST

Authors declare for none conflict of interest.

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