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Rukiya Saad Jassam
Department of Biology, College of Science, University of Baghdad, Baghdad, Iraq,
Roqia.Saad2302@sc.uobaghdad.edu.iq

Husam Sabah Auhim
Department of Biology, College of Science, University of Baghdad, Baghdad, Iraq,
husam.auhim@sc.uobaghdad.edu.iq

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RESEARCH ARTICLE

Correlation Between Prevalence of Some Colibactin Genes, Biofilm Formation, and Antimicrobial Resistance in Uropathogenic *E. coli* in Iraq

Rukiya Saad Jassam, Husam Sabah Auhim *

Department of Biology, College of Science, University of Baghdad, Baghdad, Iraq

ABSTRACT

Colibactin is a genotoxin produced by Enterobacteriaceae via a polyketide synthase (pks) island cluster. There is less knowledge regarding the distribution of colibactin genes in *E. coli* isolates in Iraq and its correlation with biofilm and antibiotic susceptibility. Therefore, this study aimed to investigate the frequency of some colibactin genes (*CIbA* and *CIbQ*) in uropathogenic *E. coli* in Iraq and evaluate the correlation with biofilm and antimicrobial resistance. Between October 2023 and January 2024, 70 *E. coli* isolates were isolated from 120 females diagnosed with UTIs. Isolates were identified first by biochemical methods and confirmed molecularly by amplification of *16S rRNA* gene with specific primers. PCR was employed to detect the *CIbA* and *CIbQ* genes. Biofilm production was assessed by the microtiter plates method, and antibiotic susceptibility was determined by the disc diffusion method. Results indicate a high prevalence of pks+ *E. coli* (40%, 28/70) compared to pks- *E. coli* (60%, 42/70) with non-significant differences ($p > 0.05$). *E. coli* produce different levels of biofilm with significant differences ($p < 0.05$). However, a statistically significant correlation has been found between *E. coli* (pks+) and a moderate biofilm formation with a significant difference ($p < 0.05$). pks+ *E. coli* isolates are more sensitive to all antibiotics under investigation, with a significant relation to fluoroquinolone group. Interestingly, current and previous results show that pks+ *E. coli* prevalence in Iraq has gradually increased over the last five years. High rates of pks-positive isolates in Iraqi urinary tract infections indicate the spread of highly genotoxic isolates.

Keywords: *CIbA*, *CIbQ*, Colibactin, *E coli*, Urinary tract infections

Introduction

Colibactin is a bacterial toxin initially discovered in *Escherichia coli* strains harbouring polyketide synthase (pks) genes by Nougayrede *et al.* in 2006.[1] Colibactin is a genotoxin that has been associated with various detrimental effects on cellular processes, including chromosome abnormalities, double-strand DNA breaks, cell cycle arrest, and immune cell death.[2] The island known as pks harbours a total of 19 genes responsible for synthesising colibactin, denoted as *ClbA* to *ClbR*. It is worth noting that the full complement of pks genes is necessary to create colibactin in its complete form.[3,4] Researchers have used two genes to detect the pks cluster, which are *ClbA* and *CIbQ* genes, due to their being close to the 5‘ and 3‘end of the pks cluster.[5] Recently, researchers have shown keen interest in investigating the frequency and implications of bacteria carrying the pks island, a potential determinant of the onset of colorectal cancer. This is mostly due to the disproportionate presence of *E. coli* strains harbouring pks genes in

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* Corresponding author.
E-mail addresses: Roqia.Saad2302@sc.uobaghdad.edu.iq (R. S. Jassam), husam.auhim@sc.uobaghdad.edu.iq (H. S. Auhim).

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biopsies obtained from individuals diagnosed with colorectal cancer.[6]

Additional members of the Enterobacteriaceae family, such as *Klebsiella pneumoniae*, *Enterobacter aerogenes*, and *Citrobacter koseri*, have been documented to possess the colibactin island.[7] Notably, the pks gene cluster identified in *K. pneumoniae* exhibits complete sequence identity (100%) with the pks gene cluster present in *E. coli*, suggesting that its regulation and functionality are conserved.[8] Moreover, it has been established through research that there exists a correlation between colibactin and the pks cluster with the hypervirulence of *K. pneumoniae*. The existence of the pks cluster has been found to be correlated with both gut colonisation and mucosal invasion by *K. pneumonia*.[9] Colibactin has been found to increase the likelihood of severe outcomes associated with bacterial infections, including meningitis and tumorigenesis. The hypothesis posits that the presence of pks-positive strains may serve as a possible biomarker for the occurrence of tumours and the effectiveness of anticancer treatments.[10] The prevalence of pks-positive members in the Enterobacteriaceae family varies from one country to another, as documented by researchers.[11–13]

E. coli is a predominantly responsible agent of urinary tract infections locally and internationally, especially in women.[14,15] Limited information is available on the prevalence of the colibactin cluster in *E. coli* isolated from urinary tract infections in Iraq, and on its association with biofilm production and antibiotic susceptibility. Therefore, it is important to determine the distribution of colibactin-producing *E. coli* among Iraqi patients with UTIs and to assess its possible correlation with biofilm formation and antibiotic susceptibility. This would aid in understanding the epidemiological aspects of the potentially genotoxic effects of *E. coli*, their clinical implications, and potential strategies for eliminating these strains.

Materials and methods

Collection of specimens and bacterial identification

A total of 120 urine samples were collected from two hospitals in Baghdad, the Baghdad Teaching Hospital and AL-Yarmok Hospital, between October 2023 and January 2024. The samples were directly cultured on a selective medium MacConkey agar, and then incubated at 37°C for 24 hours. In addition to microscopic examination, a selective Eosin methylene blue (EMB) agar and biochemical tests, including oxidase and catalase, were used to primarily identify bacterial isolates that have been growing on MacConkey agar. Molecular techniques eventually confirmed the identification by targeting the *16S rRNA* gene with specific primers. The identified bacterial isolates were cultured and stored at 4°C for subsequent examination.

Oligonucleotide primers

The gene sequences were downloaded from NCBI: the accession numbers for the *16S rRNA* gene (J01859.1) and the *CIbA* and *CIbQ* genes (CP147555.1). In silico oligonucleotide primers were designed using Serial Cloner 2-6-1 and Amplifx software. The primers were manufactured by Macrogen Company; for *16SrRNA* gene the primer pair was16S-Ec-F: 5‘-TAGCTGGTCTGAGAGGATGACCA-3‘, 16S-Ec-R: 5‘-CCAACATTTCACAACACGAGCTGAC-3‘, with an amplicon size of (802 bp), for the *CIbA* gene the primer pair was CIbA-F: 5‘-CGCTATGATCAGTTTCATTTGTGAG-3‘, CIbA-R:5‘-GAGAAATAAACAGGTGAACCTCTATATTT3-‘, with an amplicon size of (500 bp), for the *CIbQ* gene the primer pair was CIbQ-F:5‘ -GCCATATTCAGGTGGTTCTGC3-‘, CIbQ-R:5‘-CTATGCCCAAAAA TGGCGTAGTC-3‘, with an amplicon size of (207 bp). Thermo Fisher Scientific’s Tm calculator was used to calculate primer annealing temperatures.

Whole bacterial genomic extraction

This study used a newly developed method for rapidly extracting entire bacterial genomes, which has potential applications in gene investigation. This approach integrates and modifies the boiling method[16] and the colony PCR method.[17] Briefly, 5 ml of nutrient broth was inoculated with a pure single bacterial colony and incubated for 24 hr. at 37°C. Then, cells were collected by centrifugation at 13000 rpm for 15 min, washed twice with 1 ml D.W., and resuspended in 1 ml D.W. Subsequently, the cells were standardised to an optical density (O.D.) of 1 at 600 nm. Next, a volume of 0.3 ml was transferred from the standardised bacterial culture and placed into an Eppendorf tube, and cells were collected by centrifuging at 13000 rpm for 15 mins. Subsequently, 0.75 ml of Tris-EDTA buffer (TE) was used to suspend the cells, which were then heated to 100°C for 10 mins. The genomic DNA was separated from denatured proteins and cellular debris by a centrifuge at 13000 rpm for 20 mins. The supernatant containing genomic DNA was used as a DNA template directly in the polymerase chain reaction (PCR) without further purification, at a ratio of 1:9 (supernatant to PCR reaction mixture).

Table 1. Composition of PCR mixture and PCR conditions.

Composition of PCR mixture	
Materials	**Volume in μl**
Go Taq G2 Green Master Mix (2X)	50
Forward Primer (10 μM/ μl)	5
Reverse Primer (10 μM/ μl)	5
Nuclease-free water	40
Total reaction mixture	100

Aliquot 9 μl into PCR tubes, and 1 μl of supernatant containing DNA was added to each tube

PCR conditions					
	30 reaction cycles				
Initial denaturation	Denaturation	Annealing	Extension	Final Extension	Hold
5 min (95°C)	30 s (95°C)	30 s (57°C)	1 min (72°C)	5 min (72°C)	5 min (4°C)

Polymerase chain reaction (PCR)

The conventional PCR technique was used to amplify the target gene. The *16Sr RNA* gene was used for molecular identification of *E. coli* isolates, and the *CIbA* and *CIbQ* genes were used to detect the colibactin cluster island. Table 1 presents the composition of the reaction mixture and the reaction conditions.

Gel electrophoresis technique

The agarose gel electrophoresis technique was employed to examine the PCR results using a 2% agarose gel (w/v). The gel was prepared by dissolving 2 g of agarose powder (Promega, USA) in 100 ml of 1X TAE (40 mM Tris-acetate, 1 mM EDTA, pH 8.8), then boiling the mixture until all gel particles were dissolved. Subsequently, 4 μl ethidium bromide (10 mg/ml, Promega, USA) was added to the solution after it had cooled to 50–60°C. Afterwards, the agarose was poured into a gel tray and solidified for 30 minutes at room temperature. 5 μl of PCR product was loaded and run alongside 3 μl of a 100 pb DNA marker (Promega, USA) for 60 minutes; electrophoresis was carried out at 100 V. The visualisation of DNA bands was facilitated by employing a UV viewer illuminator system (Fisher Scientific, UK).

Biofilm formation assay

For the biofilm formation assay, a microtiter plate technique was applied as described by Djordjevic *et al.*[18] Briefly, all isolates were cultivated in 37°C brain heart infusion (BHI) broth for 24 hours. After that, it was calibrated with McFarland standard 0.5. A sterile 96-well U-shaped bottom polystyrene microplate with three wells contained 200 μl of an isolated culture. The plate was covered and aerobically incubated for 24 hours at 37°C. BHI broth wells were used as negative controls. After incubation, three gentle rinses with distilled water were performed, followed by drying for 1 hour at 65 °C, then fixation in methanol for 30 minutes and washing once with distilled water. The plates were stained for 10 minutes at room temperature with 200μL of 1% crystal violet solution. After that, the plates were rinsed with distilled water and dehydrated at 37°C for 30 minutes. The adhering cells were resolubilised with 200 μl of glacial acetic acid, and the optical density (OD) at 580 nm for each well was measured using a Microplate ELISA reader (Diagnostic Automation, Inc., USA). The cut-off optical density (ODC, which is known as 3 standard deviations (SD) over the average OD of the negative control: ODC = average OD of negative control + (3 × standard deviations of negative control) was determined by calculating three standard deviations above the average optical density of the negative control. The isolates were categorised into four groups based on their ODc value: OD $\leq$ ODC = non-producer, ODC $<$ OD $\leq$ 2 × ODC = weak biofilm producer, 2 × ODC $<$ OD $\leq$ 4 × ODC = moderate biofilm producer and 4 × ODC $<$ OD = strong biofilm producer.

Antibiotic susceptibility test

Using CLSI 2023 recommendations as standards, the antibiotic susceptibility patterns were evaluated by the disk diffusion method using Mueller-Hinton agar plates as described by Velican *et al.*[19] The antimicrobial disks that were employed were Ampicillin (AM, 25 μg), Piperacillin (PRL,100 μg), Ceftriaxone (FOX, 30 μg), Cefixime (CFM, 30 μg), Ciprofloxacin (CIP, 5 μg), Levofloxacin (LEV, 5 μg), Amikacin (AK, 30 μg), Gentamycin (GN, 10 μg), Impenem (IMP, 10 μg) and Meropenem (MEM, 10 μg). *E. coli* isolates were classified as resistant, sensitive, and

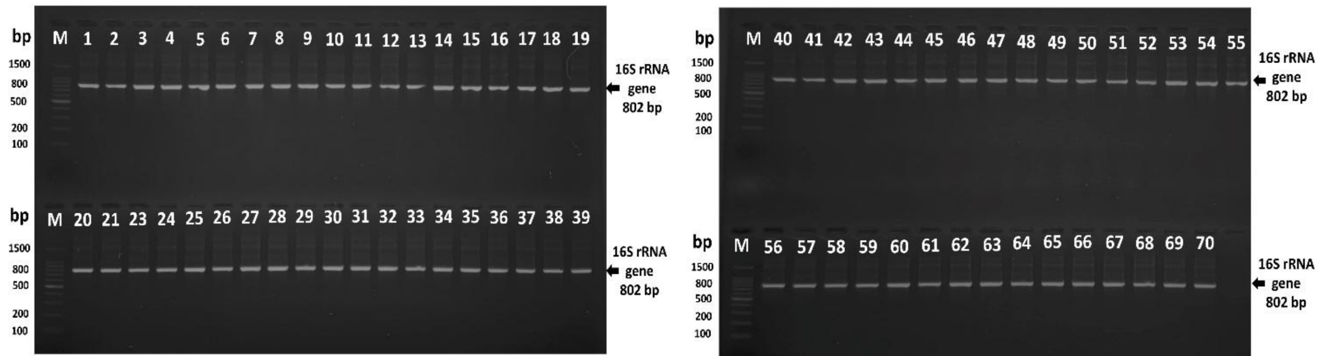

Fig. 1. Agarose gel electrophoresis (2%) of PCR amplicon showing *16S rRNA* gene (802 bp). Numbers 1 to 70 correspond to *E. coli* isolates. M corresponds to the DNA marker (100 bp).

intermediate after incubation at 37°C for 24 hours, as mentioned by Velican *et al.*[19] by comparison of inhibition zone diameter to the CLSI, 2023 guidelines.

Statistical analysis

All features appeared as frequencies and percentages. A Pearson-Chi-square test was conducted to find significant differences in percentages. $P \leq 0.05$ was measured as significant. SPSS v. 22.0 and Excel 2013 statistical software were used for data analysis.

Results

Bacterial isolation and identification

A total of 70 *E. coli* isolates (58.33%, 70/120) were isolated from 120 urine samples collected from females diagnosed with UTIs in Iraqi hospitals compared to (41.67%, 50/120) of other bacterial isolates with non-significant differences ($p > 0.05$). Primary identification of *E. coli* isolates relied on microscopic examination, culturing characteristics on selective and differential media, and some biochemical tests. All isolates were Gram-negative and rod-shaped. The isolates appeared as pink colonies on MacConkey agar and produced a green metallic sheen colour on EMB medium. All isolates were positive to the catalase test and negative to the oxidase test. In addition, the identity of *E. coli* isolates was verified by targeting the *16S rRNA* gene with specific primers using conventional PCR, as shown in Fig. 1. A clear single DNA band (802bp) identifies bacterial isolates as *E. coli*.

Molecular detection of colibactin genes (ClbA and ClbQ)

Tow genes (*ClbA* and *ClbQ*) were targeted to detect the presence of colibactin island (pks) in *E. coli* isolates obtained from UTIs in Iraq. Results reveal that (40%, 28\70) of *E .coli* isolates have both genes (*ClbA* and *ClbQ*), which are pks$^+$, compared to (60%, 42\70) of *E. coli* isolates that lack *ClbA* and *ClbQ* genes, which are considered as pks-negative, with a non-significant difference ($p > 0.05$), as shown in Figs. 2 and 3

Table 2. Frequency and percentage of biofilm types in *E. coli* isolates.

Biofilm	N	%
Strong	27	38.57%
Moderate	23	32.85%
Weak	19	27.14%
Non-produce	1	1.42%
Total	70	100%
P value	$P < 0.001$***	

A correlation between biofilm formation and possessing the colibactin island (pks+)

The results of biofilm formation demonstrated that 98.57% of the isolates were positive for biofilm formation (69/70). In comparison, 1.42% of isolates (1/70) were non-biofilm formers with significant differences ($p < 0.05$). The positive biofilm-forming isolates revealed different levels of biofilm formation. Strong biofilm formers (38.57%), followed by moderate biofilm formers (32.85%) and weak biofilm formers (27.14%), as illustrated in Table 2.

Results showed that most isolates of *E. coli* that possess pks+ led to the formation of a strong biofilm (51.86%,14/27), a weak biofilm (52.64%,10/19), a moderate biofilm (13.03%, 3/23), and a non-producing biofilm (100%,1/1). The differences among biofilm types in pks-positive isolates were significant ($p < 0.05$). The differences between strong and weak biofilm with pks positive were non-significant ($p > 0.05$), whereas the differences

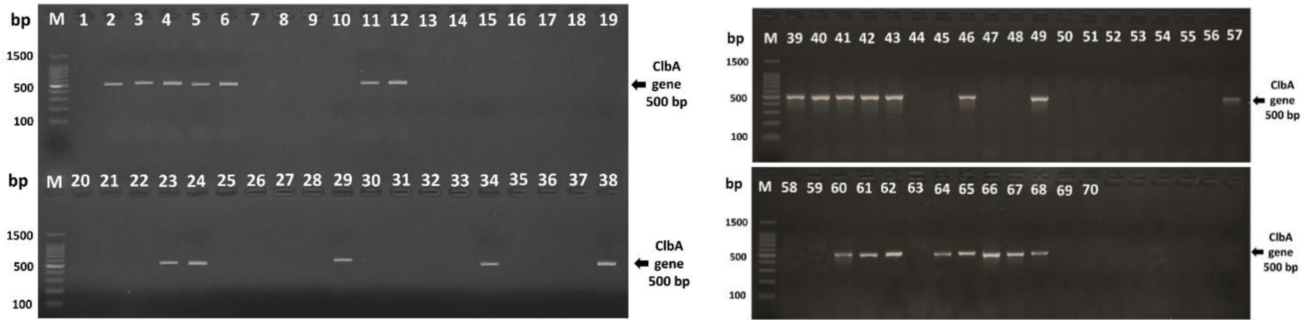

Fig. 2. Agarose gel electrophoresis (2%) of PCR amplicon showing *ClbA* gene (500 bp). Numbers 1 to 70 correspond to *E. coli* isolates. M corresponds to the DNA marker (100 bp).

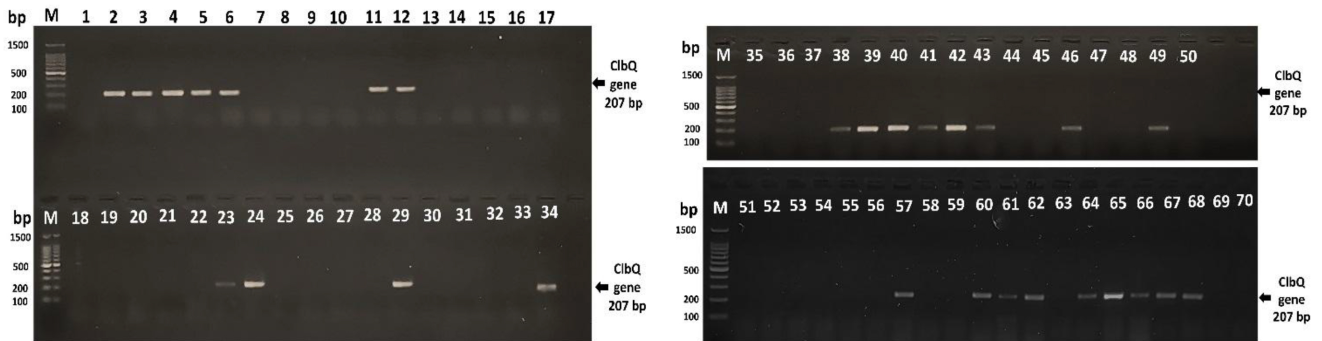

Fig. 3. Agarose gel electrophoresis (2%) of PCR amplicon showing *ClbQ* gene (207 bp). Numbers 1 to 70 correspond to *E. coli* isolates. M corresponds to the DNA marker (100 bp).

between moderate biofilm were significant ($p < 0.05$). On the other hand, the present findings showed that most *E. coli* isolates that are pks-negative formed a moderate biofilm (86.95%, 20/23), with a significant difference compared with pks-positive isolates (13.03%, 3/23). These results indicate a correlation between possessing pks and moderate biofilm formation. However, there are no significant differences between pks-positive and pks-negative isolates in strong or weak biofilm formation ($p > 0.05$), as demonstrated in Table 3.

Correlation of colibactin island (pks+) with antibiotic susceptibility

All isolates (70) were tested against 10 antimicrobial agents belonging to five different antibiotic groups, with two antimicrobial agents for each group. The antibiotics chosen in this study were the standard therapeutic options for UTIs. The isolates were found to have a broad spectrum of resistance from 100% against Piperacillin to 2.85 % against Imipenem and Meropenem; a wide range of sensitivity from 85.71% against Meropenem to 0 % against Piperacillin, Ampicillin and Amikacin, and a wide range of intermediate susceptibility from 18.57% against Ceftriaxone to 0% against Piperacillin and Gentamicin, as shown in Fig. 4.

Based on the antibiotic susceptibility results, it can be seen that all colibactin-producing isolates showed low levels of antibiotic resistance toward all antibiotics under investigation compared with non-colibactin-producing isolates, as illustrated in Table 4. However, a statistically significant correlation has been found between colibactin-producing isolates and resistance to the fluoroquinolone group (Ciprofloxacin and Levofloxacin) as well as Amikacin, with a significant difference ($p > 0.05$).

Results and discussion

Urinary tract infections (UTIs) are common human diseases that pose significant health risks to hospitalised and out-of-hospital patients.[20] *E. coli* is the main cause of UTIs, which cause inflammation in the kidneys, ureters, and urethra. However, *E. coli* strains synthesising colibactin have been found to develop cancer diseases such as colorectal cancer and increase resistance to chemotherapeutic medicines by enhancing epithelial to mesenchymal transition.[21]

E. coli appeared as pink colonies owing to their ability to ferment the lactose and produce a distinctive

Table 3. A correlation of biofilm types with pks-positive and pks-negative *E. coli* isolates.

Biofilm types	pks– N	pks– %	pks+ N	pks+ %	Total N	Total %	P value
Strong	13	48.14%	14	51.86%	27	39%	P > 0.05
Moderate	20	86.95%	3	13.05%	23	33%	P < 0.001***
Weak	9	47.36%	10	52.64%	19	27%	P > 0.05
Non-produce	0	0%	1	100%	1	1%	1.00
Total	42	60 %	28	40 %	70	100 %	P > 0.05
P value	P < 0.001***		P < 0.01**		P < 0.001***		

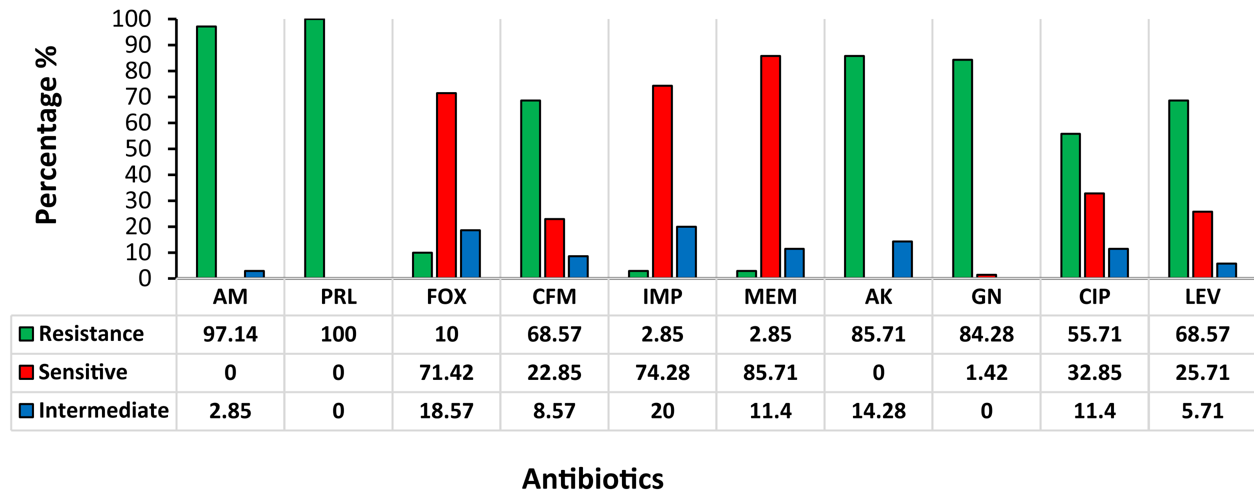

Fig. 4. Antibiotic susceptibility profile of isolated *E. coli*, expressed as a percentage (%). Ampicillin (AM), Piperacillin (PRL), Ceftriaxone (FOX), Cefixime (CFM), Ciprofloxacin (CIP), Levofloxacin (LEV), Amikacin (AK), Gentamycin (GN), Imipenem (IMP) and Meropenem (MEM).

metallic green sheen colour on EMB medium (because of the metachromatic characteristics of the dyes, the motility of *E. coli* using flagella, and the production of strong acid end-products during fermentation), which allows a rapid distinction of *E. coli* from other lactose-fermenting bacteria.[22] Primary identification is considered an essential step to isolate bacteria from UTIs, and this identification helps to exclude other uninteresting bacteria, such as *Klebsiella* and *Pseudomonas*, which may be involved in UTIs, thus saving time and resources.[23,24] However, molecular identification based on amplification of a specific gene is more reliable than other identification methods and is widely employed for pathogen detection and identification.[25,26] A study conducted by Muhaimeed *et al.* revealed that 100% of the primarily identified *E. coli* were confirmed by the *16S rRNA* gene with an amplicon size of 180 bp.[27]

UTIs are primarily caused by *E. coli*, according to local and international investigations that have shown a high prevalence of *E. coli* in urine samples from UTIs.[28,29] The percentage of *E. coli* isolates in the current study was 58.33% (70/120). These results are consistent with a local study conducted by Abood *et al.*,[30] who isolated *E. coli* from urine with an isolation percentage of (58.09%). Other local studies showed higher percentages of isolation (70 %)[31] and (65.8%).[32] The high percentage of *E. coli* isolated from UTIs could be attributed to bacterial adaptation to the urinary tract environment and their ability to survive in harsh conditions. Moreover, it possesses powerful virulence factors and the ability to form biofilm. Furthermore, women have a higher susceptibility to UTIs in comparison to men, mostly due to differences in their urinary system anatomy.[33]

Colibactin is a genotoxic secondary metabolism that damages DNA. First discovered in *E. coli*,[1] this toxin gained research interest due to its association with colorectal cancer. The distribution of this toxin in *E. coli* and other members of Enterobacterales is a worrying situation, especially when a correlation between this toxin and other virulence factors has been established.[34] The results showed a high prevalence of colibactin-producing isolates amongst *E. coli* isolates obtained from UTIs in Iraq; the prevalence percentage was 40% compared to previous local studies that reported lower percentages of 8%,[7] 11.18%,[12] and 14%.[35] However, the percentage of pks-positive isolates in Spain was 19.9%, which was reported as a high prevalence percentage.[36] Based on the previous investigation, it is noteworthy that the prevalence of pks-positive isolates in Iraq has shown

Table 4. A correlation of antibiotic susceptibility with pks-positive and pks-negative *E. coli* isolates.

		Ampicillin			
Genotype	Sensitive	Resistance	Intermediate	Total	P value
Colibactin (+)	0 (0%)	27 (96.42%)	1 (3.58%)	28 (100%)	P < 0.001***
Colibactin (-)	0 (0%)	41 (97.62%)	1 (2.38%)	42 (100%)	P < 0.001***
P value	1.00	P > 0.05	P > 0.05	P > 0.05	
Piperacillin					
Colibactin (+)	0 (0%)	28 (100%)	0 (0%)	28 (100%)	1.00
Colibactin (-)	0 (0%)	42 (100%)	0 (0%)	42 (100%)	1.00
P value	1.00	P > 0.05	1.00	P > 0.05	
Ceftriaxone					
Colibactin (+)	21 (75%)	3 (10.71%)	4 (14.29%)	28 (100%)	P < 0.001***
Colibactin (-)	29 (69.04%)	4 (9%)	9 (21.96%)	42 (100%)	P < 0.001***
P value	P > 0.05	P > 0.05	P > 0.05	P > 0.05	
Cefixime					
Colibactin (+)	6 (21.42%)	20 (71.42%)	2 (7.16%)	28 (100%)	P < 0.001***
Colibactin (-)	10 (23.80%)	28 (66.66%)	4 (9.54%)	42 (100%)	P < 0.001***
	P > 0.05	P > 0.05	P > 0.05	P > 0.05	
Imipenem					
Colibactin (+)	20 (71.42%)	2 (7.16%)	6 (21.42%)	28 (100%)	P < 0.001***
Colibactin (-)	33 (78.57%)	1 (2.39)	8 (19.04)	42 (100%)	P < 0.001***
P value	P > 0.05	P > 0.05	P > 0.05	P > 0.05	
Meropenem					
Colibactin (+)	24 (85.72%)	2 (7.14%)	2 (7.14%)	28 (100%)	P < 0.001***
Colibactin (-)	36 (85.71%)	0 (0%)	6 (14.29%)	42 (100%)	P < 0.001***
P value	P > 0.05	1.00	P > 0.05	P > 0.05	
Amikacin					
Colibactin (+)	0 (0%)	21 (75%)	7 (25%)	28 (100%)	P < 0.001***
Colibactin (-)	0 (0%)	39 (92.85%)	3 (7.15%)	42 (100%)	P < 0.001***
P value	1.00	P < 0.05*	P > 0.05	P > 0.05	
Gentamicin					
Colibactin (+)	1 (3.58%)	23 (82.14%)	4 (14.28%)	28 (100%)	P < 0.001***
Colibactin (-)	0 (0%)	36 (51.4%)	6 (8.5%)	42 (100%)	P < 0.001***
P value	1.00	P > 0.05	P > 0.05	P > 0.05	
Ciprofloxacin					
Colibactin (+)	11 (39.28%)	13 (46.42%)	4 (14.30%)	28 (100%)	P > 0.05
Colibactin (-)	13 (30.95%)	25 (59.52%)	4 (9.53%)	42 (100%)	P < 0.001***
P value	P > 0.05	P < 0.05*	1.00	P > 0.05	
Levofloxacin					
Colibactin (+)	11 (39.29%)	17 (60.71%)	0 (0%)	28 (100%)	P < 0.0*
Colibactin (-)	7 (16.66%)	31 (73.80%)	4 (9.53%)	42 (100%)	P < 0.001***
P value	P > 0.05	P < 0.05*	1.00	P > 0.05	

a progressive increase over the past five years. In 2020, the percentage was 8%,[7] which rose to 11.18% in 2022,[12] further increasing to 14% in 2023;[35] in the current study, in 2024, the percentage reached 40%. This is an alarming indication that genotoxic isolates are spreading rapidly and gradually in urinary tract infections in Iraq. Similarly, two surveys in Taiwan have shown an increasing trend in the infection rate of pks-positive *K. pneumoniae* strains. These surveys revealed an increase from 16.7% to 25.6% of pks-positive *K. pneumoniae* strains in cases of bloodstream infections.[37] The widespread occurrence of pks-positive isolates could be attributed to the horizontal transfer of plasmids containing colibactin cluster within the bacterial population; the colibactin cluster has many characteristics of a horizontally transferred genetic segment, including (i) the existence of a P4-like integrase gene, (ii) the existence of flanking direct repeats (16-bp), (iii) *asnW* tRNA locus for the chromosomal insertion, (iv) a higher CG content compared to the core genome.[38] The high prevalence of pks-positive isolates is a concerning sign of the widespread distribution of a highly genotoxic strain in urinary tract infections in Iraq, with possible transfer of the colibactin cluster to other bacteria.

Findings regarding biofilm formation revealed that the majority of bacterial isolates (98.57%) formed biofilm under laboratory conditions; this outcome aligns with other local investigations that showed that all *E. coli* isolated from UTIs were 100% biofilm-formers;[39,40] this could explain the high percentage of isolates from UTIs. However, isolates showed different levels of biofilm formation: weak, moderate, and

strong biofilm producers, with a high percentage for strong biofilm producers, followed by moderate and weak biofilm producers. Different factors affect the adhesion of *E. coli,* such as the presence of *F1C pili, P pili, S pili, Type 1 pili, Dr adhesins*, and *Fimbriae* genes; different distributions of these adhesion factors affect the initial number of cells that succeed in adhesion and biofilm formation. Moreover, the variation in the quality and quantity of autoinducers produced by each isolate [41,42] could explain the isolates' ability to generate different levels of biofilm.

Different genes and factors control biofilm formation and antibiotic susceptibility. Also, the production of colibactin is controlled by 19 genes, which form a polyketide synthase (pks) island. [43–45] Therefore, the correlation between the colibactin cluster and biofilm formation and antibiotic susceptibility appears complex due to the number of genes involved. However, the current study found a statistically significant correlation between the presence of colibactin genes *clbA* and *clbQ* with moderate biofilm producer isolates. Most pks-negative isolates formed a moderate biofilm (86.95%) compared to pks-positive isolates (13.05%). These results align with a prior study indicating a statistically significant association between the adhesin-encoding genes *pgaA*, *papA*, *sfa* and *fimH* and the existence of some colibactin genes, including *clbA*, *clbB*, *clbN*, and *clbQ*. [46] Another research establishes that most colibactin-producing isolates exhibited moderate to strong biofilm production. [47] Regarding the correlation between pks-positive isolates and antibiotic susceptibility, it has been found that all pks-positive isolates exhibit low levels of antibiotic resistance to all antibiotics under study compared with non-producers, with a significant correlation with fluoroquinolone antibiotics and Amikacin. This is consistent with the findings of other investigations, which established that most colibactin-producing isolates showed low rates of antibiotic resistance. [36,48,49] The results from a study by Morgan *et al.* showed that fluoroquinolones reduced the expression of colibactin genes (*clbA*, *clbQ*, and clbM) and cytotoxicity at various sub-minimum inhibitory concentrations (sub-MICs), indicating a direct correlation between colibactin genes and fluoroquinolones. [50] The colibactin genes could directly or indirectly affect other genes, especially those responsible for biofilm formation and antibiotic susceptibility. Our findings corroborate the theory that colibactin genes may be associated with other biosynthetic pathways, especially when linking colibactin toxin to other toxin genes such as cnf-1 and hlyA, and to siderophore-encoding genes *iroN* and *irp1*. [36] In addition, the polyketide synthase (pks) island is involved in siderophores production, such as salmochelins, yersiniabactin, and enterobactin, by the function of phosphopantetheinyl transferase ClbA. [51] Furthermore, it has been proposed that *E. coli*, which possesses a colibactin cluster and hemolysin, has benefits in colonisation and the development of colorectal cancer. [12] Moreover, a study conducted by Morgan and his colleagues using rats as a model of ascending UTI reported that *E. coli* containing *clbA*, *clbQ*, and *cnf-1* genes caused severe infection between 48 and 72 hours. [49]

Conclusion

This study enhances our understanding of the frequency of pks-positive *E. coli* and its association with biofilm formation and antibiotic resistance in Iraq. The prevalence of uropathogenic *E. coli* isolates obtained from UTIs containing colibactin genes (*clbA* and *clbQ*) was significantly higher than in previous local investigations. This prevalence has gradually increased over the last five years. This finding suggests that genotoxic isolates are rapidly and progressively expanding in cases of UTIs in Iraq. It will be worth checking for colibactin genes in *E. coli* isolated from patients with UTIs and paying closer attention to these isolates to prevent potential subsequent detrimental effects. The correlation between biofilm production and the presence of colibactin genes (*clbA* and *clbQ*) could explain the spread of the colibactin cluster by facilitating horizontal transfer. The isolates containing colibactin genes (*clbA* and *clbQ*) exhibited greater susceptibility to all antibiotics being studied than isolates without colibactin genes. This can serve as a potential indicator of the presence of colibactin genes in *E. coli* isolates. Moreover, the low rate of antibiotic resistance in colibactin-producing isolates makes their elimination easier. Nevertheless, additional investigations are necessary to clarify the relationship between colibactin genes (*clbA* and *clbQ*) and other colibactin genes with biofilm formation, antibiotic resistance, and other virulence factors, such as cytotoxic necrotizing factor 1 and Shiga-like toxins.

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images, that are not ours, have been

included with the necessary permission for re-publication, which is attached to the manuscript.
- Author(s) sign on ethical consideration's approval.
- No animal studies are present in the manuscript.
- Ethical Clearance: The project was approved by the local ethical committee at the University of Baghdad approved the project.

Authors' contribution statement

R.S.J. and H.S.A. contributed to the research design and implementation, results analysis, and manuscript writing.

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العلاقة بين انتشار بعض جينات الكوليباكتين وتكوين الأغشية الحيوية ومقاومة المضادات الحيوية في بكتريا الايشيريشيا القولونية المسببة للأمراض البولية في العراق

رقية سعد جاسم، حسام صباح أوهيم

قسم علوم الحياة، كلية العلوم، جامعة بغداد، بغداد، العراق.

الخلاصة

الكوليباكتين هو احد السموم الجينية تنتجه البكتيريا المعوية عبر مجموعة جينات تعرف باسم pks island (pks). هناك معرفة قليلة فيما يتعلق بتوزيع جينات الكوليباكتين في عزلات الإشريكية القولونية في العراق وارتباطها بالأغشية الحيوية والحساسية للمضادات الحيوية. لذلك، هدفت هذه الدراسة إلى دراسة تواتر بعض جينات الكوليباكتين(ClbA و ClbQ) في بكتيريا الإشريكية القولونية في العراق وتقييم علاقتها مع الأغشية الحيوية ومقاومة المضادات الحيوية. بين أكتوبر 2023 ويناير 2024، تم عزل 70 عزلة من الإشريكية القولونية من 120 أنثى تم تشخيص إصابتهن بالتهاب المسالك البولية. تم التعرف على العزلات أولاً بالطرق البيوكيميائية وتم تأكيدها جزيئياً عن طريق تضخيم جين 16S rRNA باستخدام بادئات قليل النوكليوتيد المحددة. تم استخدام PCR للكشف عن جينات ClbA وClbQ .تم تقييم إنتاج الأغشية الحيوية بطريقة microtiter plate ، وتم تحديد الحساسية للمضادات الحيوية بواسطة طريقة انتشار القرص. اشارت النتائج إلى ارتفاع معدل انتشار العزلات الموجبة للكوليباكتين (40%، 70/28) مقارنة مع العزلات السالبة للكوليباكتين (60%، 70/42) مع اختلافات غير معنوية ($P>0.05$). تنتج الإشريكية القولونية مستويات مختلفة من الأغشية الحيوية مع اختلافات معنوية ($P<0.05$). ومع ذلك، تم العثور على علاقة ذات دلالة إحصائية بين الايشيريشيا القولونية الموجبة للكوليباكتين وتكوين الغشاء الحيوي المعتدل مع وجود فرق كبير ($P <0.05$). كما اظهرت العزلات الموجبة للكوليباكتين أكثر حساسية لجميع المضادات الحيوية قيد الدراسة، مع وجود علاقة معنوية لمجموعة (Fluoroquinolone). ومن المثير للاهتمام أن النتائج الحالية والسابقة تظهر أن انتشار الايشيريشيا القولونية الموجبة للكوليباكتين في العراق قد زاد تدريجياً خلال السنوات الخمس الماضية. تشير المعدلات المرتفعة للعزلات الإيجابية للكوليباكتين في التهابات المسالك البولية العراقية إلى انتشار العزلات عالية السمية الجينية.

الكلمات المفتاحية: ClbQ، ClbA، الكوليبكتين، الايشيريشيا القولونية، التهاب المسالك البولية.