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ORIGINAL ARTICLE

Virulence factors, antibiotic resistance phenotypes and O-serogroups of *Escherichia coli* strains isolated from community-acquired urinary tract infection patients in Mexico

Gloria Luz Paniagua-Contreras [a], Eric Monroy-Pérez [a], José Raymundo Rodríguez-Moctezuma [b], Pablo Domínguez-Trejo [b], Felipe Vaca-Paniagua [a], Sergio Vaca [a,*]

[a] *FES Iztacala, Universidad Nacional Autónoma de México, Estado de Mexico, Mexico*
[b] *Unidad Médica Familiar Number 64, Instituto Mexicano del Seguro Social, Juárez, Mexico*

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multidrug-resistance;
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Abstract *Background/Purpose:* Uropathogenic *Escherichia coli* (UPEC) strains isolated from patients with community-acquired urinary tract infections (UTIs) were assessed to determine the prevalence of virulence genes, antibiotic resistance, and the O-serogroup of the strains.
Methods: Consenting patients with community-acquired UTI were enrolled at Unidad Médica Familiar Number 64 (Instituto Mexicano del Seguro Social, Estado de Mexico, Mexico) and 321 urine samples were collected. Polymerase chain reaction (PCR) was used to assess 24 virulence genes and 14 O-serogroups. The Kirby-Bauer method was used to evaluate the antibiotic susceptibility of the isolated strains to 12 commonly used antibiotics.
Results: A total of 194 strains were identified as *E. coli* using standard biochemical tests, followed by PCR amplification of 16S ribosomal RNA gene. Only 58.2% of the strains belonged to the assessed 14 O-serogroups. The serogroups O25, O15, O8, and O75 were present in 20.6%, 17%, 6.1%, and 4.6% of strains, respectively. The most frequently occurring virulence genes among UPEC strains included *kpsMT* (92.2% strains), *usp* (87.1%), *irp2* (79.3%), *iha* (64.9%), *fim* (61.3%), *set* (36%), *astA* (33.5%), *pap* (24.7%), and *papGII* (21.1%). In addition, 97% of the strains were multi-drug resistant (coresistance to 3–11 antibiotics).

* Corresponding author. FES Iztacala, Universidad Nacional Autónoma de México. Avenida de Los Barrios 1, Los Reyes Iztacala, Tlalnepantla, 54090, Estado de México, México
E-mail address: vacasergio@gmail.com (S. Vaca).

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Conclusion: The isolated UPEC strains predominantly belonged to three serogroups (O25, O15, and O8), harboured numerous virulence genes, and are multiresistant to antibiotics. The findings of this study could be used to orient UTI treatment strategies and in epidemiological studies in Mexico.

Introduction

Urinary tract infection (UTI) is one of the most commonly occurring bacterial infections[1] and 70—95% of the UTIs are caused by uropathogenic *Escherichia coli* (UPEC).[2] Virulence factors related to UPEC include genes encoding adhesins (*pap*, *papG* allele I, *papG* allele II, *papG* allele III, *sfa*, *afal*, *fim*, *iha*, and *tsh*); genes related to the iron acquisition systems (*iuc*, *iroN*, and *irp2*); protectin genes (*kpsMT* and *iss*); and genes encoding toxins (*set*, *astA cnf1*, *hlyA*, *vat*, *usp*, and *cva/cvi*).[3,4]

UPEC strains are routinely serotyped using the common O antisera and the serogroups O1, O2, O4, O6, O7, O8, O15, O16, O18, O21, O22, O25, O75, and O83 are preferentially associated with UPEC strains.[3,5] Serogroup assays are used for accurate *E. coli* identification and for epidemiological investigations of *E. coli* outbreaks.

The treatment of UTIs caused by UPEC often requires antimicrobial therapy. However, currently there has been a rise in the occurrence of antibiotic-resistant UPEC strains, and UTIs are considered a serious health concern.[1]

Given the clinical importance of UTIs, the aim of this work was to determine the prevalence of virulence genes, resistance to antibiotics, and the O-serogroup of UPEC strains isolated from patients with community-acquired UTIs in Mexico.

Methods

Sample collection and *E. coli* strains

Three hundred and twenty-one urine samples were collected from patients with community-acquired UTIs (247 women and 74 men; age range, 20—70 years) from Unidad Médica Familiar (UMF) Number 64 (Instituto Mexicano del Seguro Social), located in Estado de Mexico, Mexico, from August 2013 to December 2013. Patient inclusion criteria were as follows: persons with signs and symptoms of UTI and accepting to participate in the study by signing the informed consent letter were enrolled. Patients undergoing antibiotic treatment were excluded. The local ethics committee of UMF approved the study. The samples were cultured on blood agar and MacConkey agar (Bioxon, Mexico, Mexico) at 37°C overnight. The identification of *E. coli* was performed by IMViC tests, and confirmed by 16S ribosomal RNA (rRNA) gene polymerase chain reaction (PCR) amplification as described elsewhere.[6,7] For each test, *E. coli* ATCC 11775 was used as the control strain. An UTI caused by *E. coli* was recognized if it was accompanied by symptoms suggestive of infection and a culture of a clean-catch urine sample with at least 10^5 colony forming units of *E. coli* per milliliter.

DNA extraction

DNA extraction was performed using the boiling method. Bacteria were grown in brain-heart infusion broth (BHI; Bioxon) at 37°C overnight. The bacterial overnight growth culture (1.5 mL) was pelleted, the culture supernatant was discarded, and the cell pellet was suspended in 200 μL of sterile water. The resuspended pellet was then incubated at 100°C for 10 minutes and centrifuged at 10,000*g* for 5 minutes. The pellet was discarded and the DNA, present in the supernatant, was stored at −20°C until required for assays.

Antibiotic susceptibility testing

The standard disc diffusion method of Kirby-Bauer by using Mueller Hinton agar (Bioxon) was performed to evaluate antibiotic susceptibility. The antibiotic susceptibility of gram-negative bacteria was assessed by using 12 different antibiotic-loaded discs (Bio-Rad, Mexico). Results were interpreted in accordance with the Clinical and Laboratory Standards Institute guidelines.[8] The following antibiotics were used at the indicated concentrations: ampicillin (10 μg), cefalotin (30 μg), cefotaxime (30 μg), carbenicillin (100 μg), ceftriaxone (30 μg), chloramphenicol (30 μg), pefloxacin (5 μg), gentamicin (10 μg), nitrofurantoin (300 μg), netilmicin (30 μg), amikacin (30 μg), and trimethoprim-sulfamethoxazole (25 μg). For each test, *E. coli* ATCC 11775 was used as the control strain. In accordance with the manufacturer of the discs (Bio-Rad), strains were considered resistant to carbenicillin if the diameter of inhibition halo was $\leq$ 19 mm; and resistant to pefloxacin if the diameter of inhibition halo was $\leq$ 14 mm.

Detection of virulence genes and serogroups of UPEC strains

The primer sequences and PCR cycling conditions described by Momtaz et al[9] were used to assess the prevalence of the following virulence genes: (1) adhesins: *pap* (pilus associated with pyelonephritis), *papG* allele I, *papG* allele II, *papG* allele III, *sfa* (S fimbriae), *afal* (afimbrial adhesin I), *fim* (type-1 fimbriae), *iha* (nonhemagglutinating adhesin), and *tsh* (temperature-sensitive hemagglutinin); (2) nutrition: *iuc* (aerobactin), *iroN* (iron), and *irp2* (iron-repressible protein); (3) protectins: *kpsMT* (K-antigen) and *iss* (increased serum survival protein); and (4) toxins: *set*

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Table 1 Virulence genes and serogroups of UPEC strains

UPEC serogrup (n = 194)		Adhesins No. of strains (%)									Nutrition			Protectins		Toxins							*she* PAI		
	No. (%)	*pap*	*papGI*	*papGII*	*papGIII*	*sfa*	*afa*	*fim*	*Iha*	*tsh*	*iuc*	*iroN*	*irp2*	*kpsMT*	*iss*	*astA*	*cnf1*	*hlyA*	*vat*	*usp*	*cva/cvi*	*set*	*sigA*	*sap*	*pic*
		48 (24.7)	23 (11.8)	41 (21.1)	12 (6.2)	24 (12.3)	25 (12.8)	119 (61.3)	126 (64.9)	10 (5.1)	32 (16.5)	23 (11.8)	154 (79.2)	179 (92.2)	7 (5.9)	65 (33.5)	14 (7.2)	30 (15.4)	11 (5.6)	169 (87.1)	9 (4.6)	70 (36)	8 (4.1)	3 (1.5)	5 (2.5)
01	5 (2.5)	1	—	3	1	1	1	4	3	—	1	2	4	4	—	3	1	1	—	4	—	3	—	—	—
02	1 (0.5)	—	—	1	—	—	—	1	1	—	—	—	1	1	—	—	—	—	—	1	—	1	—	—	—
04	0 (0)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
06	2 (1.0)	—	1	—	1	1	—	—	2	—	—	2	—	2	—	1	—	—	—	2	—	—	—	—	2
07	1 (0.5)	—	—	—	—	—	—	1	—	—	—	—	1	1	—	—	—	—	—	1	—	—	—	—	—
08	12 (6.1)	1	3	2	—	4	1	7	5	5	4	3	9	11	1	6	1	3	1	9	2	2	—	1	—
015	33 (17)	12	5	9	5	6	6	22	25	—	5	1	31	31	1	8	4	7	3	29	—	16	1	—	—
016	7 (3.6)	1	2	1	—	1	—	5	5	—	—	1	7	7	—	2	—	1	—	7	—	2	—	—	—
018	0 (0)	—	—	—	—	—	—	—	5	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
021	2 (1.0)	—	—	—	—	1	1	2	1	—	—	—	1	1	—	2	—	—	—	2	—	—	—	—	—
022	1 (0.5)	—	—	—	—	—	—	—	—	—	—	—	1	1	—	1	—	—	—	1	—	—	—	—	—
025	40 (20.6)	18	4	19	1	8	7	30	39	3	11	2	36	40	1	13	5	14	1	34	1	21	4	1	—
075	9 (4.6)	2	4	—	1	—	—	5	8	2	2	1	9	9	—	5	—	—	2	5	2	6	—	1	—
083	0 (0)	—	—	—	—	—	—	—	—	—	—	—	—	0	—	—	—	—	—	—	—	—	—	—	—
Not detected	81 (41.7)	13	4	6	3	12	—	42	32	0	9	11	54	71	4	24	3	4	—	74	4	19	3	0	3

UPEC = uropathogenic *Escherichia coli*.

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(*Shigella* enterotoxin 1), *astA* (enteroaggregative heat stable toxin), *cnf1* (cytotoxic necrotizing factor 1), *hlyA* (hemolysin), *vat* (vacuolating autotransporter toxin), *usp* (uropathogenic specific protein), and *cva/cvi* (colicin V plasmid operon genes). To determine the presence of *set* gene in the *she* pathogenicity island (*she* PAI), the *sigA*, *pic*, and *sap* genes, which are also present in pathogenicity-associated islands (PAIs), were detected using PCR as described elsewhere.[10]

The primers and PCR conditions used for detection of UPEC O1, O2, O4, O6, O7, O8, O15, O16, O18, O21, O22, O25, O75, and O83 serogroups were described by Li et al.[11]

Statistical analysis

The frequencies of the virulence-related genes among the serogroups of UPEC strains were analyzed using Chi-square tests. A p value < 0.05 was considered significant.

Results

Detection of serogroups and virulence genes of UPEC strains

A total of 194 strains isolated from patients with community-acquired UTIs were identified as *E. coli* by using standard biochemical tests and the identity was confirmed using PCR amplification of the 16S rRNA gene. Only 58.2% ($n = 113$) of the strains belonged to 11 of the 14 assessed serogroups (Table 1). The serogroups O25, O15, O8, and O75 were prevalent in 20.6% (40), 17% (33), 6.1% (12), and 4.6% (9) isolated UPEC strains, respectively (Table 1).

The adhesion genes with the highest prevalence were *iha* (64.9%, $n = 126$), *fim* (61.3%, $n = 119$), and *pap* (24.7%, $n = 48$). The iron acquisition genes *irp2* and *iuc* were present in 79.3% (154) and 16.5% (32) of the isolated UPEC strains, respectively. The most commonly present gene for host defense avoidance was the capsular polysaccharide production related gene, *kpsMT* (92.2%, $n = 179$). The toxin-coding genes *usp*, *set*, *astA*, and *hlyA* were present in 87.1% (169), 36% (70), 33.5% (65), and 15.4% (30) strains, respectively. The *she* PAI-associated *sigA*, *sap*, and *pic* genes were found in a very low number of the strains (Table 1).

UPEC strains belonging to serogroups O25 (20.6%, $n = 40$), O15 (17%, $n = 33$), and O8 (6.1%, $n = 12$) harbored the highest number of associated virulence factors (Table 1).

The virulence gene frequencies among the serogroups of UPEC strains were different ($p < 0.05$).

Multidrug resistance

Almost 98% of the UPEC strains were resistant to cefalotin (97.9%, $n = 190$), ampicillin (97.4%, $n = 189$), or carbenicillin (97.4%, $n = 189$). Seventy-six percent of strains ($n = 148$) were resistant to pefloxacin and 72.7% ($n = 141$) strains were resistant to cefotaxime (Table 2). Overall, a total of 188 strains (96.9%) were resistant to three to 11 antibiotics (Table 3).

Table 2 Antibiotic resistance of the UPEC strains

Antibiotic	Number (%)
Cefalotin (Cf)	190 (97.9)
Ampicillin (Am)	189 (97.4)
Carbenicillin (Cb)	189 (97.4)
Pefloxacin (Pef)	148 (76.3)
Cefotaxime (Ctx)	141 (72.7)
Trimethoprim & sulfamethoxazole (Sxt)	128 (66.0)
Ceftriaxone (Cro)	95 (48.9)
Gentamycin (Ge)	97 (50.0)
Nitrofurantoin (Nf)	87 (44.8)
Netilmicin (Net)	72 (37.1)
Chloramphenicol (Cl)	50 (25.8)
Amikacin (Ak)	28 (14.4)

Patterns of virulence genotypes, antibiotic resistance phenotypes, and serogroup of the UPEC strains

A total of 138 distinct arrangements of antibiotic resistance phenotypes and virulence markers were identified in the UPEC strains. None of the patterns was predominantly prevalent (Table 3). The most abundant pattern (No. 1; *fim/iha/irp2/kpsMT/usp/set*/cefalotin/ampicillin/carbenicillin) was represented only by 11 strains (5.6%) that belonged to serogroups O25 (6 strains), O15 (1 strain), O75 (1 strain), and to O-serogroup not assessed in this study (3 strains).

Discussion

Recently, several different prevalence rates of UPEC strains related to UTIs have been described in different countries.[12–14] Furthermore, the antibiotic resistance of UPEC strains is of increasing public health concern.[15] In this study, we identified *E. coli* in 60.4% ($n = 194$) of the 321 urine samples obtained from patients with community-acquired UTIs. Unlike diarrheagenic *E. coli* strains that have been classified into six different pathotypes,[16] UPEC strains are genetically more diverse and may share virulence determinants with diarrheagenic *E. coli*.[3] Four serogroups (O25, O15, O8, and O75) were predominantly prevalent among the UPEC strains assessed in this study. It has been previously reported that 025 was the most frequent serotype found in uropathogenic *E. coli* in Mexico City.[17]

Genes encoding fimbrial adhesive systems are the most commonly occurring virulence factors in *E. coli* that cause UTIs.[18] The adhesion factors promote colonization, invasion, and replication within uroepithelial cells[19] and precede the expression of other bacterial products such as toxins, iron acquisition systems, and host defense avoidance mechanisms (protectins), which are encoded on PAIs.[20] The most prevalent adhesion genes found in our study were *iha* (64.9%, $n = 126$), *fim* (61.3%, $n = 119$), *pap* (24.7%, $n = 48$), and *papGII* (21.1%, $n = 41$; Table 1). The adhesion genes were mainly associated with serogroups O25, O15, and O8 (Table 1). These findings are in agreement

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Table 3 Patterns of virulence genes, antibiotic resistance phenotypes, and O-serogroup of the UPEC strains

Number of pattern	Patterns of virulence genes and antibiotic resistance phenotypes	Serogroups															Number of strains	%
		01	02	04	06	07	08	015	016	018	021	022	025	075	083	Not detected		
1	*fim*/*iha*/*irp2*/*kpsMT*/*usp*/*set*/Cf/Am/Cb	—	—	—	—	—	—	1	—	—	—	—	6	1	—	3	11	5.6
2	*fim*/*iha*/*irp2*/*kpsMT*/*usp*/*astA*/Cf/Am/Cb	—	—	—	—	—	—	1	—	—	1	—	2	1	—	4	9	4.6
3	*fim*/*iha*/*iuc*/*irp2*/*kpsMT*/*usp*/*set*/Cf/Am/Ctx/Pef/Cb	—	—	—	—	—	—	1	—	—	—	—	2	—	—	4	7	3.6
4	*fim*/*iha*/*irp2*/*kpsMT*/*usp*/*astA*/*set*/Cf/Am/Cb	—	—	—	—	—	—	1	—	—	—	—	4	1	—	0	6	3.1
5	*fim*/*irp2*/*set*/Cf/Am/Cb	—	—	—	—	—	—	—	—	—	—	—	1	—	—	4	5	2.5
6	*fim*/*irp2*/*astA*/*set*/Cf/Am/Cb	—	—	—	—	—	—	1	—	—	—	—	—	—	—	3	4	2.0
7	*fim*/*papGI*/*iha*/*irp2*/*kpsMT*/*usp*/*set*/Cf/Am/Ctx/Cb	—	—	—	—	—	—	1	1	—	—	—	—	2	—	0	4	2.0
8	*fim*/*iha*/*irp2*/*kpsMT*/*usp*/Cf/Am/Cb	—	—	—	—	—	—	3	—	—	—	—	1	—	—	0	4	2.0
9	*pap*/*iha*/*irp2*/*kpsMT*/*usp*/*set*/Cf/Cro/Am/Sxt/Ctx/Net/Pef/Ge/Cb	—	—	—	—	—	—	2	1	—	—	—	—	—	—	0	3	1.5
10	*fim*/*irp2*/*kpsMT*/*astA*/Cf/Am/Cb	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	3	1.5
11	*pap*/*papGII*/*sfa*/*fim*/*iha*/*irp2*/*kpsMT*/*cnf1*/*hlyA*/*usp*/Cf/Am/Net/Pef/Ge/Cb	—	—	—	—	—	—	2	—	—	—	—	—	—	—	0	2	1.0
12	*sfa*/*hlyA*/*set*/Cf/Cro/Am/Sxt/Ctx/Pef/Nf/Ge/Cb	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	2	1.0
13	*hlyA*/*fim*/*set*/*irp2*/Cf/Am/Ctx/Pef/Cb	—	—	—	—	—	—	—	1	—	—	—	—	—	—	1	2	1.0
14	*afa*/*fim*/*astA*/Cf/Am/Ctx/Pef/Nf/Cb	—	—	—	—	—	1	—	—	—	—	—	—	—	—	1	2	1.0
15	*papGI*/*sfa*/*fim*/*iha*/*irp2*/*kpsMT*/*hlyA*/*usp*/Am/Pef/Cb	—	—	—	—	—	1	—	—	—	—	—	1	—	—	0	2	1.0
16	*fim*/*iha*/*iroN*/*irp2*/*kpsMT*/*cva-cv1*/Cf/Am	—	—	—	—	—	1	—	—	—	—	—	—	1	—	0	2	1.0
17	*pap*/*papGII*/*iha*/*irp2*/*kpsMT*/*usp*/Cf/Am/Cb	—	—	—	—	1	—	—	—	—	—	—	1	—	—	0	2	1.0
18—138	Other combinations	5	1	0	2	0	9	20	4	0	1	1	21	3	0	57	124	64.0

Ak = amikacin; Am = ampicillin, Cb = carbenicillin, Cf = cefalotin, Cl = chloramphenicol; Cro = ceftriaxone, Ctx = cefotaxime, Ge = gentamycin, Net = netilmicin; Nf = nitrofurantoin, Pef = pefloxacin, Sxt = trimethoprim and sulfamethoxazole; UPEC = uropathogenic *Escherichia coli*.

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with those previously reported for UPEC strains ($n = 162$) isolated from cystitis[21] and for UPEC strains ($n = 202$) isolated from patients with community-acquired UTIs.[22] The *fim*, *pap*, *sfa*, and *afa* genes have been associated with cystitis and pyelonephritis.[23] Previous studies have shown that *papGII* is associated with pyelonephritis and bacteraemia and that class *papGIII* is associated with cystitis; although strains associated with pyelonephritis and bacteraemia have also been shown to harbor *papGIII* genes.[24,25] In this study, *iha* was the most frequent adhesion gene detected in the UPEC strains (Table 1). It has been described that *iha* confers nonadherent *E. coli* K12 adherence capability to Hela cells, and the encoded protein (Iha) was first identified from an *E. coli* O157:H7 isolate.[26]

In this study the iron acquisition system genes, *irp2* (79.3%, $n = 154$) and *iuc* (16.5%, $n = 32$; Table 1), were the most prevalent in the serogroups O25, O15, and O8. The prevalence rates obtained in our study were higher than those previously described for UPECs,[27] but lower than those described for avian pathogenic *E. coli*, indicating that frequencies of iron acquisition system genes vary according to location and host.[28] Iron is an essential element for the survival of *E. coli* and facilitates numerous cellular activities such as peroxide reduction, electron transport, and nucleotide biosynthesis.[29]

High prevalence of virulence markers in the UPEC strains isolated from patients with community-acquired UTIs suggests that these strains are highly pathogenic. The capsule-encoding gene, *kpsMT*, is commonly prevalent in UPEC strains associated with pyelonephritis than in strains associated with other UTIs.[30] In this study, $>$ 92% (179) of the isolated strains were positive for *kpsMT* (Table 1). Furthermore, the percentage of strains with toxin-coding genes *usp*, *set*, *astA*, and *hlyA* were 87.1%, 36%, 33.5%, and 15.4%, respectively. These data indicated that the isolated strains have the potential to produce toxins. Different gene prevalence frequencies have been described for UPECs isolated in other countries.[21,22,31] Other virulence genes that were detected in UPECs were as follows: *usp*, which has been shown to occur more frequently in strains associated with pyelonephritis than with cystitis[32]; *set* encoding the ShET-1 (*Shigella* enterotoxin 1) toxin that has been shown to induce fluid accumulation in the rabbit ileal loop and may account for initial watery diarrhea that can occur in early stages of *Shigella flexneri* 2a infections[33]; *astA* encoding the EAST-1 (enteroaggregative heat-stable toxin 1) toxin that plays a role in enteroaggregative *E. coli* (EAEC) pathogenicity[34] and *hlyA* in UPEC increased urothelial damage *in vivo*.[35]

The emergence of *E. coli* isolates that have multiple-antibiotic-resistant phenotypes, which confers resistance to four or more unrelated families of antibiotics has been previously reported and is considered a serious health concern.[36,37] Most UPEC strains that were characterized in this study were resistant to cefalotin, ampicillin, carbenicillin, pefloxacin, and cefotaxime (Table 2), with 96.9% ($n = 188$) of strains showing multi-drug resistance (core-sistance to 3—11 antibiotics). This finding is in agreement with previous findings on antibiotic resistance of UPEC strains isolated from patients from other countries.[22] The high prevalence of multiple-antibiotic resistance in UPECs may be related with the frequent use of these drugs to treat UTIs in Mexico. Of particular concern is the finding that a high percentage of *E. coli* strains are resistant to trimethoprim/sulfamethoxazole (66%) and nitrofurantoin (44.8%) because these antibiotics, and ciprofloxacin, are the therapeutic agents used in the Mexican social security health system, Instituto Mexicano del Seguro Social, to treat UTIS.

The transfer of resistance determinants by mobile genetic elements, including plasmids, transposons, and gene cassettes present in integrons[38] and the alteration in *mar* locus regulation may be important factors that can contribute to the increase in the prevalence of multidrug-resistant bacteria.

In this study, different patterns of combinations of the virulence markers and antibiotic resistant phenotypes were identified in the UPEC strains (Table 3). All the strains from patterns 1—4, 7—11, and 15—17 (36%, $n = 70$) had genes for adhesins, nutrition, protectins, toxins, and resistance to cefalotin, ampicillin, and carbenicillin (resistance to 1 of the aforementioned antibiotics was absent in strains with patterns 15 and 16), and mainly belonged to serogroups O15, O25, and O8. The pattern 1 (*fim*/*iha*/*irp2*/*kpsMT*/*usp*/*set*/cefalotin/ampicillin/carbenicillin) was the most frequently present (Table 3). The association between *fimA* and *kpsMT* genes that was identified in our study was previously characterized using *E. coli* isolated from UTIs and asymptomatic bacteriuria in children.[31] In this study we found that genes *fim* and *kpsMT* were present in a high percentage of the *E. coli* strains (57.7%, $n = 110$, data not shown). The simultaneous possession of these genes may enhance the pathogenesis of *E. coli* due to the increased adhesion (fimbriae encoded by *fim*) and the evasion of phagocytosis (capsule encoded by *kpsMT*).

In conclusion, to our knowledge, in addition to the work of Molina-López et al,[17] this is the second comprehensive study that characterizes the genotype of uropathogenic *E. coli* in Mexico. The strains characterized here showed the coexistence of different combinations of virulence markers and confirmed the prevalence of antibiotic-resistant phenotypes among the distinct serogroups. The findings of our study have potential application in designing UTI treatment strategies against UTIs caused by *E. coli* and for epidemiological studies in Mexico.

Conflicts of interest

All contributing authors declare no conflicts of interest.

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