Correlation of Type 1 and Type 3 Fimbrial Genes with the Type of Specimen and the Antibiotic Resistance Profile of Clinically Isolated Klebsiella pneumoniae in Baghdad

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ABSTRACT

*Klebsiella pneumoniae* is a member of coliform bacteria that causes wide ranges of infections including circulatory, respiratory system, urinary tract infections (UTIs), and wounds infections. This study aimed to find the correlation between type 1 and 3 fimbrial genes expression with multidrug resistance (MDR) *K. pneumoniae* isolates towards antibiotics. Sixty clinical isolates of *K. pneumoniae* were collected from three main types of samples including blood, wound and burn swabs, and urine samples. The diagnosis was confirmed by VITEK-2 system and 16s rRNA housekeeping gene. The antibiotic sensitivity profile included 16 antimicrobial agents, with extended-spectrum beta-lactamase production. PCR technique was applied to detect four genes of type-1 fimbrial genes: (*usher*-1, *chaperon*-1L, *chaperon*-1S, and *fim-H1*), beside type-3 fimbrial genes: (*MrkA, MrkB, MrkC, MrkD*, and *MrkF*). The results showed that *K. pneumoniae* isolates were hundred percent (100%) resistant towards ampicillin, no resistance (0%) was recorded towards tigecycline and ertapenem, while the percentages of resistance for ceftazidim, cefepime, amikacine, and amipenem were 15%, 20%, 51.7%, and 50% respectively, and the isolates showed about (13-71%) resistance to the rest antimicrobials agents. The production of extended-spectrum beta-lactamase was in 40 (66.67%) of total the 60 isolates. There was no relationship according to the statistical analysis between the types of specimen with the antibiotic resistance rates. For fimbriae type 1 genes, the largest occurrence (90%) was reported in *Chaperon-1* gene and the lowest one was in *Usher-1* gene (56.6%), while it was above 70% in *Chaperon-1L* gene and *fim-H1* gene of the total *K. pneumoniae* isolates. The percentages of type 3 genes *MrkA, MrkB, MrkC, MrkD*, and *MrkF* were: 28.3, 76.6, 85, 51.6, and 63.3% respectively. The type 1 fimbrial genes had no significant correlation among them; however, the type-3 fimbrial genes had significance in their presence at 0.01 and 0.05 levels, as they are located on the same Mrk operon. Finally, the correlation between type 1 and 3 fimbrial genes with the type of specimen and antibiotic resistance was not significant at all.

KEYWORDS: antibiotic resistance; *fimH1; usher-1* gene; *Mrk* genes; *Klebsiella pneumoniae*.

**Article Info**

Received 12/01/2022
Accepted 12/03/2022
Published 25/09/2022

**A r t i c l e i n f o**

*Klebsiella pneumoniae* is a member of coliform bacteria that causes wide ranges of infections including circulatory, respiratory system, urinary tract infections (UTIs), and wounds infections. This study aimed to find the correlation between type 1 and 3 fimbrial genes expression with multidrug resistance (MDR) *K. pneumoniae* isolates towards antibiotics. Sixty clinical isolates of *K. pneumoniae* were collected from three main types of samples including blood, wound and burn swabs, and urine samples. The diagnosis was confirmed by VITEK-2 system and 16s rRNA housekeeping gene. The antibiotic sensitivity profile included 16 antimicrobial agents, with extended-spectrum beta-lactamase production. PCR technique was applied to detect four genes of type-1 fimbrial genes: (*usher*-1, *chaperon*-1L, *chaperon*-1S, and *fim-H1*), beside type-3 fimbrial genes: (*MrkA, MrkB, MrkC, MrkD*, and *MrkF*). The results showed that *K. pneumoniae* isolates were hundred percent (100%) resistant towards ampicillin, no resistance (0%) was recorded towards tigecycline and ertapenem, while the percentages of resistance for ceftazidim, cefepime, amikacine, and amipenem were 15%, 20%, 51.7%, and 50% respectively, and the isolates showed about (13-71%) resistance to the rest antimicrobials agents. The production of extended-spectrum beta-lactamase was in 40 (66.67%) of total the 60 isolates. There was no relationship according to the statistical analysis between the types of specimen with the antibiotic resistance rates. For fimbriae type 1 genes, the largest occurrence (90%) was reported in *Chaperon-1* gene and the lowest one was in *Usher-1* gene (56.6%), while it was above 70% in *Chaperon-1L* gene and *fim-H1* gene of the total *K. pneumoniae* isolates. The percentages of type 3 genes *MrkA, MrkB, MrkC, MrkD*, and *MrkF* were: 28.3, 76.6, 85, 51.6, and 63.3% respectively. The type 1 fimbrial genes had no significant correlation among them; however, the type-3 fimbrial genes had significance in their presence at 0.01 and 0.05 levels, as they are located on the same Mrk operon. Finally, the correlation between type 1 and 3 fimbrial genes with the type of specimen and antibiotic resistance was not significant at all.

**KEYWORDS:** antibiotic resistance; *fimH1; usher-1* gene; *Mrk* genes; *Klebsiella pneumoniae*.
INTRODUCTION

*Klebsiella pneumoniae* is a multidrug resistant (MDR) bacterium that causes septicemia, lung infections, liver abscesses and urinary tract infection, which is considered as an opportunistic pathogen in both hospital and community-acquired diseases [1]. The adhesion properties of *K. pneumoniae* are generally mediated by type-1 and type-3 fimbriae, which are consists of globular proteins that enables *K. pneumoniae* attach to the host cells as the first step in infectious process [2]. Type-1 fimbrial genes are essential for colonization, invasion and persistence of *K. pneumoniae* in the UTI. They are expressed from a chromosomal *Fim* gene cluster consisting of eight genes, while the type 3 belong to the chaperoneusher class of fimbriae and are encoded by five genes in addition to other genetic mechanisms in *K. pneumoniae* (Mrk A, B, C, D and F) [3].

Type 1 and type 3 fimbrial adhesins are mainly involved together with the antimicrobial MDR as there is a strong association that obviously explained the persistence of the MDR isolates for long time in the hospital environment and the difficulty of their eradication of *K. pneumoniae* [4]. Correlation of fimbriae type 1 and 3 with antibiotic resistance was well-characterized in *K. pneumoniae* [5]. Many pathogens initiate the colonization using fimbriae that mediate adhesion to host and environmental surfaces, facilitate invasion into the host tissues and promote bacterial interactions, which might suggest that the bacterium exhibits important resistance mechanisms that lead to the increasing pathogenicity and virulence [6]. *Klebsiella pneumoniae* harboring *fimH*, *mrkD* and other specific fimbrial genes that are sensitive to limited antibiotics, like meropenem, but not to others. These isolates of clinical *K. pneumoniae* are capable to develop MDR phenotype [7]. On the other hand, resistance genes encoded via some plasmids were evaluated with the presence of the fimbrial genes [8]. The current study aimed to detect the presence of type 1 and type 3 fimbrial genes in *K. pneumoniae* isolates and find their relationship with type of specimen and antibiotic resistance profile.

MATERIALS & METHODS

Sample collections

Sixty clinical isolates of *K. pneumoniae* were collected from three main types of samples including blood (20), wound and burn swabs (20) and urine samples (20) from three hospitals in Baghdad.

Bacterial Identification

The phenotypic examination for the isolates was first determined by culturing on selective media including MacConkey agar and blood agar media (Himedia, UK). VITEK-2 (Bio-Merieux, France) compact system for Gram-negative bacteria was used to detect the genus and species of the isolated bacteria via biochemical tests to ensure the primary identification of *K. pneumoniae*.

DNA Extraction

DNA extraction was performed (according to the instruction of DNA extraction wizard kit, Promega, USA), then concentration and purity of the extracted DNA were measured. The genotypic identification of the isolates was conducted using 16s rRNA housekeeping gene (HKG) for all isolates. The sequences of 16s rRNA primers were (5’AGAGTTTGATCCTGGCTCAG3’) for the forward primer, and (5’TACGTTACCTTGTT ACGACTT3’) for the reverse primer [9]. PCR mixture consists of 12.5μl of green master mix (Promega/ USA), 3μl of template DNA, 1.5μl of each forward and reverse primers of 16S rRNA gene and 6.5μl of nuclease-free water. After mixing by vortex, the mixtures placed in the thermocycler and all stages conditions were previously set in the thermocycler programme. Initial denaturation temperature was performed at 94 °C for 5 minutes for one cycle, followed by 40 cycles consisting of the 3 stages of PCR: Denaturation at 94 °C for 1 minute, annealing at 60 °C temperature for minute and extension at 72 °C for minutes. Finally, the final extension stage was done at 72 °C for 5 minutes and one cycle.

Antibiotic Sensitivity test by VITEK-2 Compact System

Antibiotic sensitivity against many antimicrobial agents was determined using VITEK-2 system by AST cards simultaneously with the detection of bacterial genus in this technique. In VITIK-2
system, the susceptibility towards different antibiotics had been interpreted according to CLSI (2021) for Enterobacteriaceae [10]. The antibiotics were used: AMP: ampicillin (10μg), NIT: nitrofurantoin (30μg), CAZ: ceftazidim (30μg), CZ: cefazolin (30μg), CRO: ceftriaxone (30μg), CPM: cepfepime (30μg), GM: gentamicin (10μg), P/T: pipracilin/tazobactam (100/10μg), FOX: cefoxitin (30μg), IMP: imipenem (10μg), SXT: sulfa/trimethoprim (1.25/23.75μg), AK: amikacin (30μg), CIP: ciprofloxacin (5μg), LVX: levofloxacin (5μg), TGC: tigecycline (15μg), ETP: ertapenem (10μg), in addition to the production of ESβLs: extended-spectrum beta lactamase (+/-) by tested K. pneumoniae isolates.

Molecular analysis of type 1 and 3 fimbrial genes

Fimbrial genes (type 1 and type 3) were amplified by PCR for 40 cycles with the same concentrations prepared for 16s rRNA gene. All primers were designed in the current study by geneious software program, and their sequences are clarified in Table 1. Electrophoresis was then applied at 100 volt for 45 minutes using 1% agarose. The PCR product with forward primer or reverse primer for all detected genes were sent to Macrogen Company (South Korea) to be sequenced by genetic DNA analyzer using Sanger sequencing method. The results of sequence were analyzed using genius software depending on the comparison with the standard strain, which available in the National Centre for Biotechnology Information (NCBI).

<table>
<thead>
<tr>
<th>Gene symbol</th>
<th>Forward primer sequence (5’…3’)</th>
<th>Reverse primer sequence (5’…3’)</th>
<th>Size (bp)</th>
<th>Annealing temperature (℃)</th>
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</thead>
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<tr>
<td>Type 1 Fimbriae genes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Usher-1</td>
<td>AATCGCCCCAGAGACTATATT</td>
<td>CTTGCTTTTACTGGTGACA</td>
<td>874</td>
<td>57.4</td>
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<tr>
<td>Chaperon-1L</td>
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<td>CGCATTTATTATTACGCCGC</td>
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<td>AAATGTCGTACAGTTTGCGA</td>
<td>205</td>
<td>53.4</td>
</tr>
<tr>
<td>Fim-H1</td>
<td>GCCAACGTCTACGTTAACCTG</td>
<td>ATATTTTACGGTGCTGAAAA</td>
<td>180</td>
<td>53.4</td>
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<tr>
<td>Type 3 Fimbriae genes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MrkA</td>
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<td>GCACTAACAGGATGACGTAA</td>
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<tr>
<td>MrkB</td>
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<td>TTTATTTTTAGTITTTGCGGCC</td>
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<td>58.5</td>
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<tr>
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<td>CAGCAACACAAAAGATAGC</td>
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<tr>
<td>MrkF</td>
<td>TAAATATCTGCGCTCCATCC</td>
<td>GGATTCGCGAAAAAACACTAT</td>
<td>419</td>
<td>57.4</td>
</tr>
</tbody>
</table>

RESULTS AND DISCUSSION

Identification of K. pneumoniae

The colonies of K. pneumoniae characterized on blood agar media as circular, viscous, convex, smooth with distinct edges, lactose fermentation with no blood hemolysis. Bacterial diagnosis was confirmed using Gram-negative cards (ID-GN76) and confirmed using 16s rRNA gene. Gel electrophoresis results showed 16s rRNA band at 1500 bp. Figure 1: a and b illustrate the pairwise alignment of 16s rRNA with standard strain of NCBI coded as (CP009775).
As clarified in the figures above, the pairwise identity with *K. pneumoniae* standard strain (CP009775) showed 99.8% identity. Bacterial PCR products had a positive result of 16s rRNA gene confirming a *K. pneumoniae* homogenic data; by using DNA database from NCBI [11]. Moreover, molecular diagnosis using 16s rRNA gene was confirmed by other studies [12, 13].

**Phenotypic detection of antibiotic resistance pattern by VITEK-2 system**

As shown in Figure 2, *K. pneumoniae* isolates had a great resistance rate to the most antibiotics. The sensitivity percentage recorded towards ampicillin was the lowest (0%), while the highest sensitivity was towards ertapenem and tigecycline (100%).

On the other hand, *K. pneumoniae* isolates showed moderate sensitivity ranging between 40-71% for gentamycin, pipracillin/tazobactam, cefoxitin, imipenem, sulfamethoxazole/trimethoprim, amikacin, ciprofloxacin, and levofloxacin in a dramatic increasing pattern. The ESβL-negative isolates were (66.7%) of total isolates. For a less extent, sensitivity for ceftriaxone and cefepime was 20%, for nitrofurantoin was 15%, and finally it was 15% for both of ceftazidime and cefazolin. The results of this study are close to those obtained by [14, 15] regarding antimicrobial resistance rates.

![Figure 1](image1.png)

**Figure 1.** (a) Gel electrophoresis of PCR product with 1500 bp of 16s rRNA gene in 1% agarose at 100V for 45 minutes. (b) Alignment of 16s rRNA gene with the same gene of NCBI standard strains of *K. pneumoniae* (CP009775).
For the antibiotic aspect, the ability of bacteria to develop resistance through diverse mechanisms or multiple pathways they possess by acquisition of new characteristics almost has been in random pattern which give the rise to elevating antibacterial resistance [16]; besides the development of invasive infections, like liver abscesses, endocarditis, meningitis, and septic arthritis, are considered as a serious issue which help in elevating MDR towards multiple antibiotics which struggle the antibacterial effect, but not towards newer drugs [17].

For the antibiotic resistance within the three specimen groups, there was no significant differences as shown in Figure 3 regarding to the source of bacterial isolation. The same result is recorded in the study of [13, 14] in which K. pneumoniae isolated from wounds, burns, sputum, urine, and ear swabs showed various resistance towards different antibiotics, which might be related to the disease progress and other underlying conditions [18, 19].

**Figure 2.** Antibiotic sensitivity of all K. pneumoniae strains. AMP: ampicillin (10μg), NIT: nitrofurantoin (300μg), CAZ: ceftazidim (30μg), CZ: cefazolin (30μg), CRO: ceftriaxone (30μg), CPM: cefepime (30μg), GM: gentamicin (10μg), P/T: piperacilin/tazobactam (100/10μg), FOX: cefoxitin (3μg), IMP: imipenem (10μg), SXT: sulfa/trimethoprim (1.25/23.75μg), AK: amikacin (30μg), CIP: ciprofloxacin (5μg), ESBLs: extended-spectrum beta lactamase (+/-), LVX: levofloxacin (5μg), TGC: tigecycline (15μg), ETP: ertapenem (10μg).

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**Figure 3.** The antibiotic resistance within specimens’ groups.

### Detection of fimbrial genes

Gel electrophoresis results illustrated the precise size of PCR amplicons for fimbrial genes of type 1 and 3 as previously mentioned in Table 1. The electrophoresis was performed at 100V for 45 minutes in 1% agarose (Figure 4).

More than half (56.6%) of total K. pneumoniae isolates owned Usher-1 genes, with a majority in urine samples. Many pathogens initiate a UTI using fimbriae that mediate adhesion to host and environmental surfaces, facilitate invasion into the host tissues and promote bacterial interactions to form biofilms by the swans of fimbriae assembled by chaperon-usher pathway.

As shown in Figure 5(a), the identity with the NCBI strain (CP071150) was 99.1%. The results are close to those [15, 20] of usher-1 detection.
Figure 4. Gel electrophoresis in 1% agarose at 100V for 45 minutes of all studied genes. (a) Chaperon-1L gene (361bp). (b) Chaperon-1S gene (205bp). (c) Usher-1 gene (874bp). (d) FimH-1 gene (180bp) (e) MrkA gene (358bp). (f) MrkB gene (346bp). (g) MrkC gene (1155bp). (h) MrkD gene (628bp). (i) MrkF gene (419bp).

Chaperon-1L gene, which is specific to *K. pneumoniae* adherence and pathogenicity, appeared in (71.6%) of bacterial isolates in this study. Figure 5(b) shows the identity of chaperon-1L gene with the standard NCBI strain (CP066855) which was 95.5%. The results seem close to that of [21, 22] about chaperon genes. Chaperon-1S gene, which is specific to *K. pneumoniae* adherence and pathogenicity, had the occurrence of 90% in bacterial isolates of this study. Figure 5(c) shows the chaperon-1S pairwise sequence alignment identity of 98.9% with NCBI strain (CP058940). The results are similar to those of [21, 22] about Chaperon-1S genes. The gene fim-H1 was detected in (78.3%) of all clinical isolates, most of them were isolated from blood samples (17 of 20 samples). Fim-H1 pairwise alignment with NCBI strain (CP068684) of 98.8% identity is clarified in Figure 5(d). The results are similar to those in the study of [12, 23]. All the studied of type 1 fimbrial genes were prevalent in urine samples and the highest percentage was for Ch-1S gene, followed by 80% for both of Ch-1L and Fim-H1 gene; and 65% for Usher-1 gene. The high occurrence of type 1 fimbrial genes in urine samples explains how they together work to enable successful attachment, spread and persistence in the host urinary tract [3].
Figure 5. Gene pairwise sequence alignment of fimbrial type-1 genes with the same genes of NCBI standard strains of *K. pneumoniae*. (a) Usher-1 alignment with (CP071150) standard strain. (b) Chaperon-1L alignment with (CP066855) standard strain. (c) Chaperon-1S alignment with (CP058940) standard strain. (d) Fim-H1 alignment with (CP068684) standard strain.

The percentages of type 3 fimbrial genes were: (28.3%, 76.6%, 85.0%, 51.6% and 63.3%) respectively. *MrkA* gene was present in 28.33% of total isolates. Figure 6(a) shows the identity is 98.4% with *K. pneumoniae* standard strain (CP043357). The results are close to the results of (3 and 19) about *MrkA* gene occurrence. *MrkB* gene was present in 76.66% of *K. pneumoniae* isolates. The pairwise sequence alignment was 97.4% identity with *K. pneumoniae* standard strain (CP073287) as shown in Figure 6(b). *MrkC* was present in 85% of *K. pneumoniae* isolates, with an identity of 94.4% in pairwise sequence alignment approach in comparison with NCBI strain CP047595 (Figure 6(c)). The results agreed with [23-25] who confirmed the occurrence of *MrkB* and *MrkC* genes. *MrkD* gene was present in 51.66% of *K. pneumoniae* isolates, with an identity of 99.8% with NCBI strain CP084497 (Figure 6(d)). The results are similar to the results obtained by [12, 8, 26]. *MrkF* gene was present in 63.33% of total isolates. Figure 6(e) below illustrated the pairwise sequence alignment identity of 99% with the NCBI strain (CP04335). The results matched with [23, 25] based on *MrkF* gene presence.

The prevalence of type 3 fimbrial genes showed the highest rates in urine *K. pneumoniae* isolates for *MrkC* (95%), *MrkF* (75%) and *MrkA* (40%), while *MrkB* gene was mostly prevalent in swabs (80%) and *MrkD* was (80%) in blood samples. Fimbria type 3 genes are synthesized by *K. pneumoniae* to facilitate the adhesion to cells including endothelial cells and uroepithelial cells. They also mediate adhesion to several cell types of the host tissues [6].
Relation of gene occurrence with the type of specimen
As clarified in Figure 7, there was no relation of genes with the specimen type and the presence of fimbrial genes. These agreed with [21, 22, 26, 27] who mentioned the absence of the relationship between gene occurrence and the specimen’s source.

The absence of the association between the source of isolation and the genetic occurrence of *K. pneumoniae* isolates might be due to the high genetic diversity among the bacterial different clinical isolates in this study. The prevalence of one or more of the fimbrial genes among certain clinical *K. pneumoniae* isolates may contribute to the downregulation or overexpression of these genes [8]

Relation of antibiotic resistance with gene presence
As shown in Figure 8, the results are indicating the non-significant differences of antibiotic resistance rates among fimbrial gene. This result is similar to the results of [13, 28, 29]. The study of other entire factors and underlying diseases of the patients may suggest an expression to this negative relationship [19].
The characteristic of MDR \textit{K. pneumoniae} is closely related to the antibiotic resistance genes encoded by plasmid and transferable genetic elements, and consequently \textit{K. pneumoniae} continuous to accumulate these genes in the context of improper antibiotic use, resulting in the emergence of an extremely drug-resistant (XDR) strains; with the need to study further drug resistance mechanisms in \textit{K. pneumoniae} clinical isolates [16].

**Correlation between fimbrial genes 1 and 3 genes within all specimen groups**

The correlations between each pair of genes were calculated by Pearson-correlation. Fimbrial genes occurrence of type 1 and 3 were correlated within the same type and with the genes of the other type of fimbriae. As shown in Table 2, the significance correlations were only among fimbrial type-3 genes, whereas there was no correlation among fimbrial type-1 genes or between them with the genes of type-3 genes. The findings of this study are close to those of [19, 30] who recorded there was no significance among type 1 fimbrial genes.

In this study, the positive covariant relationship (p-value is less than 0.01 or 0.05) among the type 3 fimbrial genes is corresponding to the fact that they arranged in the same transcriptional orientation as they are located on the same operon (Mrk operon), while lack of some type 1 fimbrial genes in \textit{K. pneumoniae} leads to the overexpression of the other type-1 fimbrial genes with a negative inversive relationship [16].

![Figure 8. Antibiotics resistance rates within the studied genes.](image)

**Table 2. Correlations among fimbriae type 1 and 3 genes in \textit{K. pneumoniae} isolates.**

<table>
<thead>
<tr>
<th></th>
<th>MrkA</th>
<th>MrkB</th>
<th>MrkC</th>
<th>MrkD</th>
<th>MrkF</th>
<th>Ch-1L</th>
<th>Ch-IS</th>
<th>Usher-1</th>
<th>FimH1</th>
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</thead>
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<td>MrkA</td>
<td>-</td>
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<td>0.011*</td>
<td>0.018*</td>
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<td>0.536</td>
<td></td>
<td>0.336</td>
<td>0.239</td>
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<tr>
<td>MrkB</td>
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<td>-</td>
<td>0.016*</td>
<td>0.023*</td>
<td>0.023*</td>
<td>0.505</td>
<td>0.104</td>
<td>0.272</td>
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<tr>
<td>MrkC</td>
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<td>0.016*</td>
<td>-</td>
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<td>0.050*</td>
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</tr>
<tr>
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<td>0.023*</td>
<td>0.050*</td>
<td>-</td>
<td>0.0000**</td>
<td>0.697</td>
<td>0.104</td>
<td>0.426</td>
<td>0.169</td>
</tr>
<tr>
<td>MrkF</td>
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<td>0.023*</td>
<td>0.050*</td>
<td>0.0000**</td>
<td>-</td>
<td>0.697</td>
<td>0.104</td>
<td>0.426</td>
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<td>Ch-IS</td>
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<td>-</td>
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<td>0.512</td>
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<td>0.855</td>
<td>0.497</td>
<td>0.391</td>
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* Significant correlation at 0.05
**Significant correlation at 0.01

**CONCLUSIONS**

There was no significant relation of type 1, 3 fimbrial genes with the type of specimen or the antimicrobial resistance profile. This fact was dependent on the resistance profile of \textit{K. pneumoniae} isolates in a limited concern; resulting...
in the need to study the influence of other virulence factors in MDR.

**ACKNOWLEDGMENT**

Many thanks to the medical staff of hospitals in which samples were collected and to the college of Science, Mustansiriyah University, Baghdad, Iraq, for the support to complete this work (https://uomustansiriyah.edu.iq).

**REFERENCES**

https://doi.org/10.1128/CMR.00181-19

https://doi.org/10.1007/978-3-319-74715-6_8


https://doi.org/10.1099/jmm.0.000452

https://doi.org/10.1556/030.66.2019.006

https://doi.org/10.1016/j.eimc.2018.11.001

https://doi.org/10.3390/antibiotics9120852


https://doi.org/10.1186/s12866-017-1148-6


https://doi.org/10.22207/JPAM.13.2.41


https://doi.org/10.3390/ijerph17176278


https://doi.org/10.1038/srep38929

https://doi.org/10.1038/s41579-019-0315-1

https://doi.org/10.1097/MRM.0000000000000151


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