



Virulotypes of Uropathogenic *E. Coli* Isolates from Diabetic Patients in Tunisia: Occurrence of the Invasion-Associated *IbeA* Gene

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Short Communication

Extraintestinal pathogenic *Escherichia coli* (ExPEC) are an important cause of urinary tract infections (Uropathogenic *E. coli*, UPEC), neonatal meningitis and septicemia (neonatal meningitis *E. coli*, MNEC) in humans [1]. Animals are recognized as a reservoir for human intestinal pathogenic *E. coli*, but whether animals are a source for human Ex-PEC is still a matter of debate [2]. Recent studies have suggested that meat, particularly poultry, can be a source of ExPEC strain transmission to humans. In the last decade, genetic linkages between *E. coli* isolates from animal and human origins have been demonstrated especially for Extended Spectrum Beta-lactamase (ESBL) producing *E. coli* isolates. Indeed, all studies have mainly focused on the phenomenon of ESBL-production to demonstrate the transmission of *E. coli* between animal and human or vice versa. However, other genetic traits, such as some specific genes encoding virulence factors, might confirm this hypothesis. In our previous work we have reported the genetic characterization (occurrence of integrons, phylogeny, genetic relationship) of 27 *E. coli* isolates recovered from Urinary Tract Infections (UTI) of diabetic patients in Tunisia [3]. Herein, we aimed to study the genes encoding virulence factors in these isolates. Indeed, occurrence of eleven virulence genes associated with intestinal and extra-intestinal pathotypes (*hlyA*, *fimH*, *iutA*, *ibeA*, *sxt1*, *sxt2*, *eaeA*, *papC*, *papG* II, *papG* III, *IpaH*) was determined by using PCR in 27 antibiotic resistant *E. coli* strains [4,5]. The *fimH* adhesion gene was detected in 22 (81.5 %) of our isolates (Table1); this finding is in agreement with the literature, which shows that *fimH* is most frequent in isolates from UTI [6]. The *papG*II expressed as P fimbriae and gene belonging to the *pap* gene family of adherence virulence genes such as *papA*, *papC*, *papEF*, *papGII*, and *papGIII* genes, was detected in four (14.8%) isolates (Table1). However, *papC*, *papG* II, *papG* III genes were not detected. These findings highlight the importance of adhesion factors in the process of UTI. Indeed, bacterial adherence to urinary epithelia is a crucial step in the development of UTI, allowing the bacteria to persist in the urinary tract. The second most common gene in these 27 UTI isolates was *iutA*, found in 20 (70.3%) isolates, which is one of the bacterial iron acquisition systems. In urinary tract, free iron concentrations are very low for the growth of pathogenic bacteria, thus, UPEC have developed multiple ways of getting iron from the host through the expression of iron-acquisition systems that are mainly encoded by *iutA*, *fyuA* and *feoB* genes. In our isolates, genes linked to intra-intestinal *E. coli* (*hlyA*, *sxt1*, *sxt2*, *eaeA*, and *IpaH*) were not detected, which might argue for the specificity of these isolates to induce UTI rather than gastro enteritis. The most important finding of this study was the occurrence of the invasion-associated *ibeA* gene in 6 (22.2 %) isolates. The *ibeA* was involved in the pathogenesis of NMEC and Avian Pathogenic *E. coli* (APEC) [7]. Indeed, the *IbeA* product appears to increase the ability of NMEC to invade brain micro vascular endothelial cells *via* ligand receptor interaction. In recent years, the distribution of *ibeA* in the APEC and NMEC collection was examined [7], which indicated that the *ibeA* gene has been frequently detected in APEC and MNEC isolates [8]. This finding is of great interest and argues for possible poultry to human transmission of these *ibeA*-harboring isolates especially that in our country consumption of avian meat has been increased in the last 20 years. Therefore, it is recommended to investigate the occurrence of this gene in *E. coli* from other human pathogenic samples owing to its pathogenic ability.

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Table 1: Viruotypes and phylogenetic groups of 27 *Escherichia coli* isolated from urinary tract infection.

Isolates	viruotypes	Phylogenetic groups
S1	<i>fimH, iutA</i> ,	B1
S2	<i>fimH, iutA</i>	B1
S3	<i>fimH, ibeA</i>	A
S4	<i>fimH, ibeA, iutA</i>	B1
S5	<i>fimH, ibeA, iutA</i>	A
S6	<i>fimH, iutA, pap G II</i>	B1
S7	<i>fimH, iutA, pap G II</i>	A
S8	<i>fimH, iutA</i>	B1
S9	<i>fimH, iutA</i>	A
S10	<i>iutA</i>	A
S11	<i>iutA</i>	A
S12	<i>fimH, iutA</i>	A
S13	<i>fimH, ibeA</i>	A
S14	<i>fimH, iutA</i>	A
S15	<i>fimH, iutA</i>	B1
S16	<i>fimH, iutA</i>	A
S17	<i>fimH</i>	A
S18	<i>iutA</i>	B1
S19	<i>fimH, ibeA</i>	B1
S20	<i>fimH, ibeA, iutA</i>	A
S21	<i>fimH, iutA, pap G II</i>	B1
S22	<i>iutA</i>	B1
S23	<i>fimH, iutA, pap G II</i>	B1
S24	<i>fimH, iutA</i>	B1
S25	<i>fimH</i>	A
S26	<i>fimH</i>	A
S27	-	A

References

1. Manges AR, Johnson JR. Reservoirs of Extraintestinal pathogenic *Escherichia coli*. *Microbiol Spectr*. 2015;3(5).
2. Bélanger L, Garenaux A, Harel J, Boulianne M, Nadeau E, Dozois CM. *Escherichia coli* from animal reservoirs as a potential source of human extraintestinal pathogenic *E. coli*. *FEMS Immunol Med Microbiol*. 2011;62:1-10.
3. Debbichi N, Abbassi MS, Sáenz Y, Khemiri M, Majouri D, Ben Rayena C, et al. Low antibiotic resistance rates and high genetic heterogeneity of *Escherichia coli* isolates from urinary tract infections of diabetic patients in Tunisia. *J Chemother*. 2016;28(2):89-94.
4. Chapman TA, Wu XY, Barchia I, Bettelheim K A, Driesen S, Trott D, et al. Comparison of virulence gene profiles of *Escherichia coli* strains isolated from healthy and diarrheic swine. *Appl Environ Microbiol*. 2006;72:4782-95.
5. Wu XY, Chapman T, Trott D J, Bettelheim K, Do TN, Driesen S, et al. Comparative analysis of virulence genes, genetic diversity and phylogeny between commensal and enterotoxigenic *Escherichiacoli* from weaned pigs. *Appl Environ Microbiol*. 2007;73(1):83-91.
6. Yun KW, Kim HY, Park HK, Kim W, Lim IS. Virulence factors of uropathogenic *Escherichia coli* of urinary tract infections and asymptomatic bacteriuria in children. *J Microbiol Immunol Infect*. 2014;47(6):455-61.
7. Wang S, Niu C, Shi Z, Xia Y, Yaqoob M, Dai J, et al. Effects of *ibeA* deletion on virulence and biofilm formation of avian pathogenic *Escherichia coli*. *Infect Immun*. 2011;79(1):279-87.
8. Guabiraba R, Schouler C. Avian colibacillosis: still many black holes. *FEMS Microbiol Lett*. 2015;362(15):118.