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Comparison of virulence-associated traits between a UPEC strain HEC4 and a APEC strain E058

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Abstract: Since avian pathogenic *Escherichia coli* (APEC) and human uropathogenic *Escherichia coli* (UPEC) may encounter similar challenges when establishing infection in the extra-intestinal locations of the hosts, they may share a similar content of virulence genes and capacity to cause disease. One APEC and one UPEC isolates were compared by their content of virulence genes and other traits. The two strains showed overlap in terms of their virulence genotypes, including their possession of certain genes associated with a large transmissible plasmid of APEC, and also shared some biochemical activities. Study of the pathogenicity of UPEC in chicks showed the similar symptoms and lesions compare to those caused by APEC. Based on these results, the potential whether APEC might serve as a reservoir of plasmid-linked and virulence genes for UPEC should be considered.

Keywords: avian pathogenic *Escherichia coli*; uropathogenic *Escherichia coli*; virulence genes; pathogenicity

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1 INTRODUCTION

Avian colibacillosis refers to any localized or systemic infection such as an acute fatal septicemia or subacute pericarditis and airsacculitis caused entirely or partly by avian pathogenic *Escherichia coli* (APEC). It is a common systemic disease of economic importance in poultry and is seen worldwide. Urinary tract infections (UTIs) are among the most common bacterial infections, causing significant morbidity in women around the world. Most community-acquired UTIs are due to uropathogenic *E. coli* (UPEC) infections. *Escherichia coli* differ from commensal intestinal *E. coli* in having specialized virulence factors which promote extraintestinal infection[1]. UPEC are uniquely endowed with various virulence traits, enabling them to survive and grow in urine and other extraintestinal environments. The abilities of UPEC to grow extraintestinally may enable them to cause a variety of diseases, not just urinary tract ones. This broad potential to cause disease has led Russo & Johnson[2,3] to propose that UPEC be incorporated in a new category known as extraintestinal pathogenic *E. coli* (ExPEC). Based on the extraintestinal diseases in poultry of APEC[4], it is also known as ExPEC.

Bacterial population modelling reveals a close relationship between avian pathogenic *E. coli* and urinary pathogenic *E. coli*. ExPEC virulence factors exhibit distinct patterns of phylogenetic distribution. APEC may serve as reservoirs of virulence genes for human UPEC. In order to test the hypothesis, a systematic comparison of virulence-associated gene content, serotype and phylogenetic lineage of a large collection of human UPEC and avian pathogenic E. coli (APEC) isolates also confirmed the considerable overlap between APEC and human ExPEC with respect to these traits[5].

In this study, an APEC strain E058 and a UPEC strain HEC4 were selected for further investigation. Strain E058 was selected from over 400 APEC isolates with different pathogenicity in chicks, and other typical characteristics by biochemical reactions were evaluated previously[6]. HEC4 was isolated from a typical UTI woman patient and also identified with presence of more than 40 virulence-associated genes. It is anticipated that factors may direct host specificity of human and animal ExPEC isolates could be identified by pathogenomics[5]. As a first step in assessing the potential common pathogenicity between APEC and UPEC, UPEC isolate HEC4 and APEC isolate E058 were examined for their biological characteristics and pathogenicity in chicks, and compared for the content of virulence genes, including many implicated in ExPEC virulence as well as those associated with APEC plasmids. The results showed some similarities and differences between the APEC isolate E058 and UPEC isolate HEC4 studied here.

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2 MATERIALS AND METHODS

2.1 Bacteria

APEC strain E058 was isolated, identified previously[6].

UPEC strain HEC4 was kindly provided by Dr. Qunying Xi of Yangzhou No. 1 People's Hospital, which isolated from a typical UTI woman patient and identified by the traditional biochemical methods.

2.2 Primers and templates

All primers used in amplification of the virulence genes were showed in Table 1. Most primers were designed by using the primer premier 5.0 software program (Premier Biosoft International) and synthesized commercially (Sangon Inc., Shanghai, China). Template DNA for all amplifications was generated as described elsewhere[7].

2.3 Hemolytic reaction

APEC isolate E058 and UPEC isolate HEC4 were plated on 5% sheep blood agar plates and incubated overnight at 37℃. The plates were then examined for "greening" or clearing of the agar around areas of bacterial growth as an indication of alpha or beta hemolytic activity[8].

2.4 Fermentation of lactose

APEC isolate E058 and UPEC isolate HEC4 were plated on MacConkey agar and incubated overnight at 37℃. Isolate was considered positive for lactose utilization if pink colonies were observed[8].

2.5 Virulence genes detection by PCR

E058 and HEC4 were examined using PCR assays for the presence of several genes (Table 1) known for their association with ExPEC or APEC virulence. Targeted genes and their descriptions are summarized in Table 1. The APEC genes of interest have been associated with APEC plasmids, such as pTJ100[9,10]. Targeted genes were amplified in procedures documented elsewhere[11]. Amplification for these groups of genes was performed in 25 μl reaction mixtures that included 2μl template DNA, 14μl ddH_2O, 2.5μl 10 × PCR buffer (Takara), 4μl 25 mM $MgCl_2$, 0.3μl AmpliTaq Gold Taq (5 U μl^{-1}) (Takara), 0.2μl of each 10 mM dNTP (Sangon) and 2μl 0.2 mM upper and lower primers.

All samples were subjected to horizontal gel electrophoresis in 1% agarose, and the size of the amplicons was determined by comparison to the 100bp DNA marker (Invitrogen). Positive and negative controls were examined with each amplification procedure, and all amplification procedures were repeated three times to reduce the possibility of false negatives.

Table 1 PCR primers of ExPEC/ APEC virulence genes

Gene	Primer sequence (5′→3′)	Amplicon size (bp)	Annealing Temp(℃)	Reference
Adhesins				
fimH	ATGAAACGAGT-TATTACCCTGTTTGTTATTGATAAACAAAAGTCACGCC	854	57	AF089840
sfaDE	CTCCG-GAGAACTGGGTGCATCTTACCGGAGGAGTAATTACAAACCTGGCA	408	63	Le Bouguenec *et al*. (1992)
afaBC	GCTGGGCAG-CAAACTGATAATTCTCCATCAAGCTGTTTGTTCGTCCGCCG	793	61	Le Bouguenec *et al*. (1992)
papC	GACGGCTGTACTGCAGGGTGTGGCGATATCCTTTCTGCAGGGATGC AATA	328	63	Le Bouguenec *et al*. (1992)
focG	AGCACAGGCAGTGGATACGTAATACTTCCCGCACCAGC	414	57	S68237
bmaE	GCTATCGCAAGCAGTGTCCAATGTCTGGTCTCCGAAG	414	58	M15677
papGI	TTATTATTCGTGCTCAGGTCC CGTATTAGTGGCAGGTAGGTG	433	57	AF237476
papGII	CCTTGTGTGTGTCTGGTGAGTCAGTCAGACGGGTTGTTTC	415	57	AY212280
papGIII	TGTCAGGCTGTAATGATGCTCCTGTCCAGATGTGTTTGCTTC	368	58	AF237473
felA	GGCAGTGGTGTCTTTTGGTGGGCCCAGTAAAAGATAATTGAACC	279	58	L07420
gafD	AAGTGGATTTACCCGTGGGCGACAACTTCATCACCG	451	55	L33969
iha	TAACCACTCTGGCTTCCGTAGCTGACAGAATCATCCACAAGG	592	58	AF126104
pTJ100-related genes				
sitA	ACAACTTTTACCATCATCGCGTTCCGCAATCTGCTTAC	415	55	AY598030
iutA	ACAGCCGACAACTGGACTCGTAATCGTCGTCGCCCTG	440	64	AY602767
iroN	TCCTGGTTGGGTTGAATACAGCCAGAGGCCCACTA	800	58	AF449498
cvaC	CACACACAAACGGGAGCTGTTCTTCCCGCAGCATAGTTCCAT	635	63	Johnso & Stell(2000)
iss	GTGGCGAAAACTAGTAAAACAGCCGCCTCGGGGTGGATAA	760	63	Foley *et al*. (2000)
tsh	TTATTCTCTTCGCTACAGGATGACAGGCTACCGACAG	685	59	AF218073

Continued to table 1

Gene	Primer sequence(5′→3′)	Amplicon size (bp)	Annealing Temp(℃)	Reference
traT	CTTGTTCGCATTTGAGACCTGTGGTTTGTAATAAAGGTAA	685	52	AY214164
Protectins				
kpsM	GCGCATTTGCTGATACTGTTGCATCCAGACGATAAGCATGAGC	272	64	Johnson & Stell(2000)
Iron-related				
ireA	ATTACACGCTGATTCTGGTCCTTCTGGCTTTCAGTCGG	440	57	AF320691
feoB	GTGTAGGTAACTGGGCTGGAGTGAATCTGCTTCGTTGAG	478	57	X71063
iucCD	ATCCATGATTTGCAGACGGATGACAATCCGGTACCAGCA	460	59	AY553855
*irp*2	AAGGATTCGCTGTTACCGGATCGGCCAGGATGATTCGTCG	295	58	L18881
fyuA	ACACGGCTTTATCCTCTGGCGGCATATTGACGATTAACGAA	953	57	Z29675
Toxin				
*cnf*1	ACGCAGTTTCAGTGATGGTGTCATAGTAGATGCCGCTCAG	534	57	X70670
*cnf*2	AATCTAATTAAAgAgAACCATgCTTTgTATATCTA	543	44	U01097
cdtA	CTCAAGTAGAGGGAGGACCTCTGGCTTAACAATAGTGGC	601	55	AJ508930
hlyA	TTGTCAGGACGGCAGATGCGTTCAGGTGCTTTGTATTG	472	57	AF037578
hlyE	ACTCTCAATCGGCATCCACCTTCAACAACCCCAGCAG	453	57	AF052225
vat	TCGCTTACCCTGACTATCCCCTTACCAGACATACCGC	441	57	AY151282
Miscellaneous				
*flicH*7	ATCGGTGGAAGCCAGGCATACGGCAGCATCACTGGATTCACCCG	350	63	U47614
malX	GCTGGCGACTAATAACCCTATCCCATTGCTCTGCTAAG	411	57	AF081286
kfiB	GAGGGATTTAGAATACGAGACGCATTATGGGAGGTAGTCG	579	57	X77617
ibeA	TTAGTTGTTGGTGGTGGTCCCGAGTAAATTATTGCCCTGC	361	55	AY248744
leoA	TGCGTATTGCTCTGTTGGCTGTCAGGCTGGTCACTTC	399	57	AF170971
ompA	GACTGGTTAGGTCGTATGCCAACAACAGACTGAGCACGG	618	58	V00307

2.6 Pathogenicity in chicks

The pathogenicity test was performed by inoculation of the air sac with a calculated dose of overnight broth culture in LB. Forty five -3 - week old specific pathogen-free (SPF) chicks were subdivided into 3 groups of 15 chicks each. Because of its high pathogenic to chicks, APEC strain E058 was inoculated with the lower dose of 0.2mL of cell suspension containing 1×10^6 colony forming unit (CFU) per bird in the first group. The second group of 15 chicks were inoculated with 1×10^8 CFU of UPEC isolate HEC4 per bird. 15 chicks were inoculated by *E. coli* K-12 strain as a negative control group, with the same dose and the same way as that of APEC isolate E058. All the chicks were observed daily for clinical signs, at random, 3 chicks of each group were killed at 6, 12, 24, 48 and 72 hours post-inoculation (HPI). Selected tissues were examined for pathological changes at every HPI.

E. coli was reisolated and viable counted by 100μl heart blood cultured in plates for assessing gross pathogenicity.

3 RESULTS

3.1 Lactose fermentation and hemolytic activity

APEC isolate E058 was positive for lactose fermentation and negative for hemolytic reaction, so was UPEC isolate HEC4.

3.2 Prevalence of virulence genes

As shown in Table 2, both strains shared some similarity in most virulence genes, but several differences were also observed.

Table 2 Prevalence of virulence genes in HEC4 and E058[a]

	fimH	*afaBC*	*sfaDE*	*focG*	*papC*	*bmaE*	*papGI*	*papGII*	*pa* Ⅲ	*felA*
HEC4	+	+	+	+	+	−	−	+	−	+
E058	+	−	−	−	+	−	−	+	−	+
	gafD	*iha*	*sitA*	*iutA*	*iroN*	*cvaC*	*iss*	*tsh*	*traT*	*kps*M
HEC4	−	+	+	+	+	−	+	+	+	+
E058	−	−	+	+	+	+	+	+	+	+
	*ire*A	*feo*B	*iuc*CD	*irp*2	*fyu*A	*cnf*1	*cnf*2	*cdtA*	*hly*A	*hlyE*
HEC4	−	+	+	+	+	−	−	−	+	−
E058	+	+	+	+	+	−	−	−	−	−
	vat	*flicH*7	*mal*X	*kfi*B	*ibeA*	*leo*A	*omp*A			
HEC4	−	−	+	−	−	+	+			
E058	+	−	+	−	−	−	+			

a: fimH (type 1 fimbria), afaBC (afimbrial adhesion), sfa/foc (S and F1C fimbria), focG (F1C fimbria), papC, papG (P fimbrial major subunit or adhesin, respectively), bmaE (M fimbria), felA (fimbriae F11), gafD (G fimbria), iha (adhesin), sitA (S. flexneri putative iron transport gene), iutA (aerobactin receptor), iroN (siderophore receptor), cvaC (microcin V), iss (increased serum survival), tsh (temperature sensitive hemagglutinin), traT (the serum survival gene), kpsM (group 2 capsule), ireA (siderophore receptor), feoB (ferrous iron uptake gene), iucCD (aerobactin system), fyuA, irp2 (yersiniabactin system), cnf1, cnf2 (cytotoxic necrotizing factor 1), cdtA (cytolethal distending toxin protein A), hlyA, hlyE (hemolysin), vat (vacuolating autotransporter toxin), flicH7 (H-antigen type), malX (marker for pathogenicity island from strain CFT073), kfiB (K5 capsule gene cluster), ibeA (invasion) leoA (labile enterotoxin output), ompA (outer membrane protein A). (+): positive (−): negative

3.3 Pathogenicity in chicks

In the pathogenicity test of chicks, no chicks died during 72-hour observings in every group except the euthanized ones. APEC isolate E058 and UPEC isolate HEC4 challenged groups were listless with ruffled feathers and indication of stopping feeding and drinking. Additional symptoms of labored breathing and diarrhea were evident. No symptoms were showed in K-12 challenged group.

At every HPI, 3 chicks were dissected to examination of pathological changes and bacteria reisolation. APEC isolate E058 caused more severe airsacculitis than UPEC isolate HEC4 at every HPI. The consistent gross and pathological lesions were of fibrinous to caseous exudate in the air sacs, heart sac and on the surface of the heart. After inoculation 48 hours, the lesions were more severe and typical, even fibrin appeared to the surface of the liver. Otherwise, the group inoculated with *E. coli* K-12 appeared no lesions.

Both APEC E058 isolate and UPEC HEC4 isolate were reisolated and cultured in MacConkey agar plates at 37℃ for 18 ~ 24h and the CFU of the challenged isolates mentioned above in 100μl heart blood was counted, the result was shown in the figure 1.

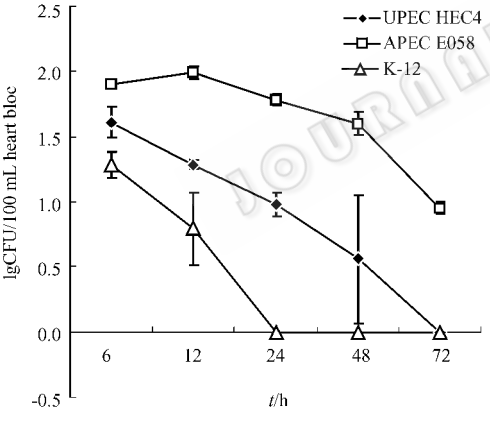

Fig.1 The CFU of the challenged isolates in 100μl heart blood at different intervals.

4 DISCUSSION

In the last years, knowledge on virulence traits and evolution of ExPEC was accumulated. ExPEC strains cause frequent infections of man and animals. Bacterial population modelling reveals a close relationship between avian pathogenic *E. coli* (APEC) and urinary pathogenic *E. coli* (UPEC). ExPEC virulence factors exhibit distinct patterns of phylogenetic distribution, and certain virulence gene associations are considered to reflect the existence of different pathotypes[12].

Genome sequence of avian pathogenic *Escherichia coli* Strain O1:K1:H7 shares strong similarities with human ExPEC genomes[13]. It was also found an APEC plasmid, pAPEC-O2-ColV, which contains many of the genes associated with APEC virulence and also shows similarity in content to a plasmid and pathogenicity island of human UPEC[14].

In this study, we compared genotypes of the UPEC isolate HEC4 to the APEC isolate E058, of special interest are certain genes, found on large plasmids in APEC such as pTJ100[9,10] are known to occur on UPEC plasmids and pathogenicity islands (PAIs)[5]. All of the APEC plasmid-related genes studied here were found in the APEC isolate E058. Several of these pTJ100- associated genes (*iss*, *sitA*, *iutA*, *ironN*) were also found in the UPEC isolate HEC4. APEC and their plasmids may be transmitted to humans, evaluation of APEC plasmids as possible reservoirs of urovirulence genes for human UPEC may be warranted. *cvaC*, which encodes colicin V[15], was proposed to confer enhanced virulence through their carriage of other specific virulence factors, including the aerobactin system and serum survival genes, such as *traT* and *iss*[16]. Otherwise the gene *cvaC* was not detected in HEC4. In UPEC, this gene may be more likely found on non-ColV plasmids or within chromosomal PAIs[17~21] than on pTJ100-like plasmids[5], which indicates that a similar heterogeneity and diversity may exist among closely related plasmids as among functionally related islands in *E. coli*.

There were also discernable notable differences between the two isolates. From the table 2, the genes specially associated UPEC adhering effect (*afaBC*, *sfaDE*, *focG*, *iha*), were not found in E058. A possible explanation for this discrepancy might lie in the different host-specific environments[22,23,24].

The results obtained in biology assays of UPEC isolate HEC4 and APEC isolate E058 showed UPEC isolate HEC4 could cause the similar symptom and lesions in chicks compared to those caused by APEC isolate E058. Otherwise, lower CFUs in 100μl heart blood and shorter existing hours of viable bacteria in blood of UPEC isolate HEC4 than that of APEC isolate E058 could also be observed, which was possibly because of their different propensity to the reservoir.

Our results suggested that consequently, it seems reasonable that the possibility of certain clonal types of APEC being implicated in human disease cannot be ruled out. Further research will be necessary to determine if APEC can actually overcome the hurdles necessary for transmission to humans via the foodchain and establishment of disease in humans or serve as a reservoir of virulence genes contributing to uropathogenesis.

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尿道致病性大肠杆菌 HEC4 株和禽源致病性大肠杆菌 E058 株毒力相关特性的比较

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摘 要 对人尿道致病性大肠杆菌(uropathogenic *Escherichia coli*, UPEC)HEC4 株和禽致病性大肠杆菌(avian pathogenic *Escherichia coli*, APEC)E058 株进行毒力基因和其他相关特性的比较,结果显示,它们具有一些共同的毒力基因,包括一些存在于 APEC 中一个大的可传递质粒上的基因;同时,它们也具有一些相似的生化特性。对 SPF 鸡的致病性试验显示,这两株分离株具有相似的致病力。因此,对于 APEC 和 UPEC 的相关性,以及 APEC 是否有可能导致人尿道感染或者成为 UPEC 的毒力基因贮主,有待进一步研究。

关键词:尿道致病性大肠杆菌;禽源致病性大肠杆菌;毒力基因;致病性

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