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Research Article

COMPARISON OF GENOTYPE AND PHENOTYPIC EXPRESSION OF ADHESINS - P FIMBRIAE AND TYPE 1 FIMBRIAE IN UROPATHOGENIC *ESCHERICHIA COLI*

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ABSTRACT

Background: Uropathogenic *Escherichia coli* possess surface adhesins called as fimbriae or pili which binds the bacteria to urothelial cells in the urinary tract. P fimbriae coded by papG commonly responsible for upper UTI and Type1 fimbriae coded by fimH present in commensal *E. coli* and with more frequency in pathogenic *UPEC.* Hemagglutination is followed for phenotypic adhesin detection and genotype studied by PCR. Variation in the phenotypic expression occurs based on the regulation, blocking or masking of the genes were influenced by the environmental it survives. Different intercross expression of fimbriae unrelated to the gene remains unclear. There is no data in comparison of phenotype and genotype, yet many studies provided data of either one type. **Aim & Objective:** The aim of the study was to determine and to compare the fimbriae detection by phenotypic and genotypic methods. **Materials & methods:** A total of 212 UPEC isolates were processed for P, Type1 fimbriae detection by hemagglutination. papG, fimH genes detected using PCR and gel documentation done after gel electrophoresis to detect specific base pair band. **Results:** Phenotypically 16% and 13.2% isolates expressed P and Type 1 fimbriae, no fimbriae in 70.8% isolates. Genotypically, papG (46.2%), fimH (92%), no genes (4.2%) and UPEC with single, association of adhesin genes were found with 9 different patterns. **Conclusion:** More studies regarding pathogenic character of fimH gene and intercross fimbrial expression not coded by the specified genes must be studied further to know their significance.

INTRODUCTION

Uropathogenic *Escherichia coli* are one of the different pathotype of extraintestinal *E. coli* which causes urinary tract infection [(1)]. The key role played behind the infection is always the virulence factors. The attachment of bacteria to the specific host cell called adhesion is the main factor for further action of rest of the virulence factors. Adhesion is the common property of pathogenic microorganisms towards host structures avoiding the swept back from flow of urine from the urinary tract in case of UTI. There are several surface adhesins for the UPEC which adheres to different area inside the urinary tract like uroplakins, uroepithelial cells, bladder epithelial cells and the cells in the kidney etc., specific type of adhesin binds to specific cell types. P fimbriae, type 1 fimbriae, afimbrial adhesin (afa), s fimbrial adhesin (sfa) etc., are the surface adhesins which are fimbrial and non-fimbrial mediates adhesion for colonization of the host cells [(2)]. The study reviews are that P fimbriae and Type 1 fimbriae are the two major adhesins which can be studied genotypically and also can detect its expression under invitro method.

PAP (Pyelonephritis associated pili) called as P fimbriae is encoded by pap gene possessing many subunits (pap A - K) adhere to the Gal - Gal receptor of the cells in the urinary tract determining the tissue and host specificity. P fimbriae is mainly responsible for upper UTI as it binds to tubular epithelial cells and muscular layers of bladder as well as to the loose adherent substances on the human intestinal tract, stimulates inflammation and overcomes the natural immune defence mechanism It agglutinates human erythrocytes despite the presence of mannose (MRHA - mannose resistant hemagglutination). The expression of these P fimbriae is subjected to rapid, random phase variations based on the environmental influences [(3)]. Adhesion of it leads to production of cytokines, local inflammation and pain associated with UTI.

Type 1 fimbriae encoded by the gene fimH, are expressed both in UPEC strains derived from cystitis and pyelonephritis patients as well as in gasterointestinal disease causing

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Enteropathogenic *E.coli*. Many studies explained type 1 fimbriae expression as Mannose sensitive hemagglutination (MSHA) using human blood group O erythrocytes. These pili have fimH adhesion in its tip, which is one of the contact point strengthening the filamentous form of UPEC towards the urothelial cells [4].

The genotypic presence of adhesion genes and its expression may vary as, "Phenotype is the observable triat and genotype is the endowment of the host". Phenotype = Genotype + Environment (development depends on environmental influence). Mostly the variation in genes does not always affect the expression of phenotype as the genes occupy only 2 percent of the total genome. But the phenotype expression may vary if there is variation in specific genotype [5]. This study had been proposed to determine the prevalence of phenotypic P and Type1 fimbirae and genotypic papG and fimH genes. Then to compare the genotype and its phenotypic expression as there is no data regarding these comparison.

MATERIALS AND METHODS

Bacterial isolates

A total of 212 *E. coli* isolated from suspected UTI patients were processed by standard culture method of identification and confirmed by biochemical tests. The study was carried out after obtaining approval from Institutional Ethical Committee (IEC).

Phenotypic fimbrial detection by haemagglutination of Human group O erythrocytes [6]

The strains of *E. coli* was inoculated into 5 ml of 1% nutrient broth and incubated at 37°C for 4 days for full fimbriation as there may be development of surface pellicle. Blood group "O" red blood cells were then washed thrice in normal saline and made up to a 3% suspension in fresh saline. They were used immediately or within a week when stored at 3-5°C. The test was carried out on Venereal Disease Research Laboratory (VDRL) slides. One drop (100µl) of bacterial suspension was mixed with one drop of erythrocytes and one drop of phosphate-buffered saline (PBS) with and without 3% D-mannose on a VDRL slide. The slide was rotated for five minutes at room temperature and the presence or absence of macroscopic haemagglutination was noted.

MRHA - Clumping of RBCs with and without presence of mannose

MSHA - Clumping of RBCs without mannose and no clumping in the presence of mannose

Escherichia coli ATCC 25922 is as a control strain for MSHA

Genotypic detection of papG and fimH gene by PCR

DNA extraction

DNA extraction was performed by using boiling lysis method. *E. coli* isolates were cultured in Luria Bertani broth at 37° C for 18 hours. Bacteria were pelleted from 1.5 ml LB broth using centrifuge at 3000 rpm then suspended in 200 ml of sterile deionized water and kept in waterbath at 100°C for 10 min. After centrifuging, the supernatant was used as template DNA and stored at -20°C [7].

PCR amplification

PCR amplification of virulence genes were used to reveal the prevalence of adhesin virulence genes papG, fimH using specific primers. The amplification of virulence genes was carried out in a Thermal Cycler (Eppendorf Master Cycler) after standardizing the PCR conditions: an initial denaturation at 94°C for 2 min, followed by 30 cycles of denaturation at 94°C for 1 min, annealing at 60°C for 30 s, and extension at 72°C for 1min 30 sec, with a final extension at 72°C for 5 min.

Table 1 Virulence gene primer sequences

Gene		Primer sequence (5'-3')	Product size (bp)	References
papG	F	GACGGCTGTACTGCAGGGTGTGGCG	328	Johnson *et al.*,2005 [8]
	R	ATATCCTTTCTGCAGGGATGCAATA		
fimH	F	AACAGCGATGATTTCCAGTTTGTGTG	410	Farmer et al.,1999 [9]
	R	ATTGCGTACCAGCATTAGCAATGTCC		

The electrophoresis of PCR products was performed on 1% agarose gel with a 100-bp DNA ladder as molecular size. The gels were stained with ethidium bromide and visualized in a Gel documentation system.

RESULTS

A total of 212 *E. coli* isolates from suspected UTI patients were studied for P and Type 1 fimbriae by both genotypic and phenotypic methods. Table 2 represents the results of phenotypic fimbrial expression by hemagglutination of human O erythrocytes. 16% and 13.2% of the isolates expressed MRHA (P fimbriae) and MSHA (Type 1 fimbriae) respectively and in 70.8% isolates there was no hemagglutination (No fimbrial expression) under invitro condition.

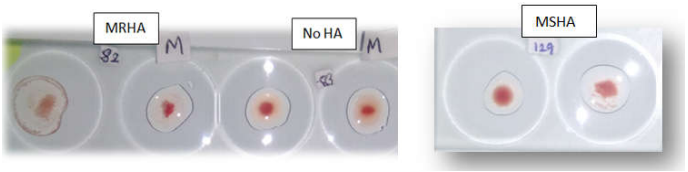


Figure 1 Phenotypic expression of Pfimbriae (MRHA), Type 1 fimbriae (MSHA), No HA - No hemagglutination, M - 3% D-mannose

Table 2 Fimbrial detection by phenotypic method - Hemagglutination

Fimbrial detection	No. of *E. coli* showed phenotypic expression	Percentage
P fimbriae (MRHA)	34	16.0
Type 1 (MSHA)	28	13.2
No hemagglutination	150	70.8

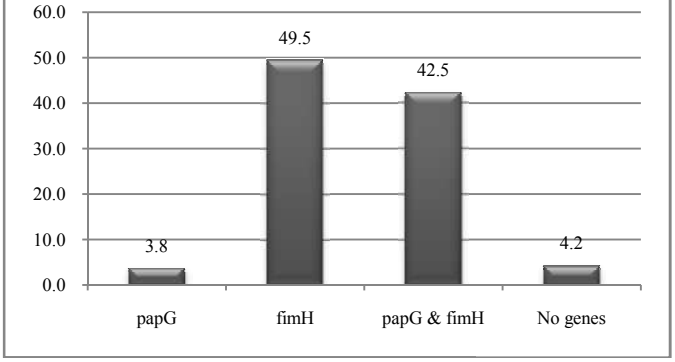
Table 3 represents the genotypic detection of papG and fimH genes encoding P and Type 1 fimbriae by polymerase chain reaction. All over 98% of isolates possess papG and 92% possess fimH genes. 4.2% of the isolates possess none of the two genes.

Table 3 Genotypic detection of adhesion (fimbrial) genes

Adhesin gene	No. of *E. coli*	Percentage
papG	98	46.2
fimH	195	92.0
No genes	9	4.2

Certain isolates had single gene, papG alone were found in 3.8%, fimH 49.5% and 42.5% isolates had association of both the genes, were represented in figure 2.

Figure 2: Percentage of adhesin genes



All the *E. coli* isolates which carry the genes do not always express the genes by encoding its specific characterization.

Figure 2: Molecular detection of papG and fimH gene by PCR, L lane is 100 bp ladder, NC - negative control. papG (328 bp) were positive in lane 2,3 and 7 in the gel picture 1 and picture 2 represents positive for fimH (410 bp) in lanes 2, 3, 4, 5, 6 and 7.

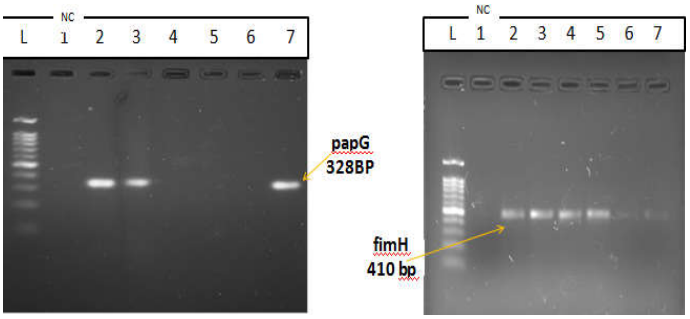


Table 4 Comparison of genotype and phenotype expression of adhesin genes among *E. coli* isolates

Adhesin Patterns	Genotypic detection - Phenotypic expression	No. of isolates
Pattern I	papG - No HA	5
Pattern II	fimH - No HA	76
Pattern III	papG, fimH - No HA	60
Pattern IV	papG - P fimbriae	1
Pattern V	fimH -Type 1 fimbriae	13
Pattern VI	papG, fimH - P fimbriae	17
Pattern VII	papG, fimH -Type 1 fimbriae	13
Pattern VIII	papG -Type 1 fimbriae	2
Pattern IX	fimH - P fimbriae	16

Among 212 isolates, 9 different genotypic patterns were identified, isolates which possess papG there was no phenotypic fimbrial expression - 5, fimH gene with no hemagglutination - 76. In certain isolates both genes were present but no fimbrial expression - 60, both genes with expression of P fimbriae and with expression of Type 1 fimbriae were 17 and 13 isolates respectively. Only 1 isolate possessing papG gene expressed P fimbriae, 13 isolates with fimH showed type 1 fimbriae. In contrast to other patterns, 2 isolates with papG gene showed type 1 fimbriae and fimH with the expression of P fimbriae were 16 isolates. The comparison of genotype and their phenotypic expression under invitro with different pattern were represented in the table 4.

DISCUSSION

UPEC has been strengthened with multiple virulence factors to confer pathogenesis in the urinary tract. For any bacterium, either to get symbiotic association to the host (ex., Commensal *E. coli* in the intestine) or to cause infection (ex., UPEC) in the host, the first necessity for binding to the host cells was supported by surface adhesins made up of many structural protein subunits encoded by specific genes.

P fimbriae encoded by papG mediates binding to the α-D-galactopyranosyl-(1-4)-β-D-galactopyranoside receptor (Gal-Gal receptor) present on the lining of host cells in the upper urinary tract and erythrocytes. Once after attachment it activates the immune response, stimulates inflammation and strengthens their colonization in the tubular epithelium leading to tubular, nephron obstruction, renal infilteration, pain associated with UTI and followed by the entire pathophysiology of pyelonephritis [10].

Type 1 fimbriae encoded by the gene fimH the commonly found virulence factor, which is mannose sensitive as its binding to the erythrocytes is blocked by mannose [11]. It is present in all subsets of *E. coli* including commensal strains where the fimbriae binds only to trimannose residues and those type 1 fimbriae binding to monomannose and trimannose residues, also express as the virulence factor of UPEC. These fimbriae binds the *E. coli* to urothelial mannosylated glycoproteins called as uroplakins expressed in the urothelium of the uethra and bladder causing adhesion and invasion of cells. It colonizes the centre of the tubule and promotes the formation of intracellular bacterial communities (Biofilm) which is responsible for the acute stage if the infection. FimH adhesion tip internalizes the UPEC within urothelial cells through which escapes the natural immune response [11, 12].

Phenotypic expression of P fimbriae and Type 1 fimbriae

In this study 16% out of 212 isolates expressed P fimbriae (MRHA), 13.2% with Type 1 fimbriae (MSHA) which is lower compared to the study Fatima et al., were P and Type 1 fimbriae were found in 30% and 18% respectively, there were no phenotypic fimbrial expression in 32% of the isolates where it was found to be higher, 70.8% in our study. The comparative study results of hemagglutination carried out in different places were shown in the table 5.

Table 5 Comparative study of hemagglutination with human erythrocytes

S.No.	Place	No. of strains	P fimbriae		Type 1 fimbriae		Reference [13, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24 respectively]	Year
			No.	%	No.	%		
1	New Delhi	170	75	44	80	47	Mandal et al	2001
2	Bangalore	191	68	36	ND*	-	Raksha et al	2003
3	CMC Vellore	163	48	30	51	32	Rebecca et al	2005
4	Lucknow	160	40	25	55	34	Manjula et al	2006
5	Poland	66	28	42	ND*	-	Dorota et al	2007
6	Bijapur	200	60	30	72	36	Yasmeen Kausar et al	2009
7	Haryana	220	68	30.9	ND*	-	Prabhat et al	2010
8	Aligarh	120	75	30	45	18	N. Fathima et al	2012
9	Bangalore	93	38	40.9	ND*	-	Shruthi N et al	2012
10	Kurnool	200	120	60	152	76	Vijayalakshmi et al	2014
11	Haryana	135	46	45.5	55	54.5	Mittal et al	2014
12	Bellary	75	31	41.3	14	81.7	Vishalakshi et al	2014
13	Tehran, Iran	156	98	62.8	58	37.2	Tabasi et al	2015

14	Lucknow	172	ND*	-	70	46.7	Sugandha et al	2016
15	Tamil Nadu	212	34	16	28	13.2	Present Study	2018

ND* - Not Done

Genotypic assay for detection of adhesion genes

Genotype defines the phenotype based on the selection process. The genotypic assay which is highly specific, informative is PCR method, here this study was carried out to detect adhesion-encoding virulence factor [25]. The distributions of papG and fimH genes in our study were 46.2% and 92% respectively. Similar to other studies fimH gene is the commonest gene compared to papG (Table 3). Association among the two genes represents their presence in the same pathogenic islands (PAI's) in the genome (Figure-1). Variations among the distribution of genes were noted in other studies which may be due to different geographical regions (Table - 6).

Table 6 Genotypic comparative study of adhesion genes

S.No.	Total no. of isolates	papG (%)	fimH (%)	References [25 to 35]	Place	Year
1	112	74	ND*	Bogylova et al	Czech Republic	2002
2	162	32.7	97.5	Tiba et al	Sao Paulo	2008
3	204	25	ND*	Oliveira et al	Brasil	2011
4	123	50.4	79.67	Aazam Krimian et al	Falavarjan, Iran	2012
5	100	14	ND*	AV Shetty et al	Mangalore	2013
6	60	30.2	ND*	Dormanesh et al	Shahrekord, Iran	2014
7	205	20.5%	ND*	Mohajeri et al	Kermanshah, Iran	2014
8	150	16.6	ND*	Nemati et al	Kashan, Iran	2015
9	91	63.7	65.9	Mayasaa et al	Egypt	2015
10	172	25.6	87.2	Sugandha et al	Lucknow	2016
11	148	20.3	89.9	Munkhdelger et al	Mongolia	2017
12	119	29.4	82.4	Pootong et al	Thailand	2018
13	50	48	66	Husam et al	Sudan	2018
14	210	24	68.5	Bahadori et al	Shiraz, Iran	2018
15	212	46.2	92	Present Study	Tamil Nadu	2018

ND* - Not Done

Variation in genotype and phenotypic expression

Variation in genes leading to serious disruption or function of the gene is said to be mutational variation while the change contributing to the normal genetic variability is polymorphism, which s based on environmental influence [5].

P fimbriae expression is regulated based on several environmental and nutritional factors and the mechanism of methylation-dependent phase variation controls its phenotypic expression [36].

Type1 fimbriae posses many subunits, fimH phase variation is controlled at the transcriptional level in the invertible element which is flanked by σ70 promoter repeats at both the ends. Leucine-responsive protein (LRP), integration host factor (IHF), and the histone-like protein (H-NS) binds to the DNA sequences around, within the invertible element thus assists or blocks the switch on and off mechanisms of the enzyme recombinases, which is required for the expression of all the subunits involved in the structural formation of Type 1 fimbriae, basically influenced by the environment [36].
Looking into other studies, this study was the specific one as it determines and compares the genotype for adhesins and their simultaneous phenotypic expression. There is a significant percentage difference between the expressions of gene; out of 195 isolates possessing fimH gene only 28(14.4%) isolates expressed Type1 fimbriae. The percent of expression of fimH is lower compared to Sugandha et al., out of 150 UPEC isolates 70 (46.7%) isolates showed phenotypic expression, no other studies has correlated this fimH and its expression. Regarding pyelonephritis associated pili, 34 (34.7%) isolates expressed P fimbriae out of 98 papG possessing isolates. This is the first study to compare both genotype and phenotype expression of papG and P fimbriae.

In our study there were 9 different patterns for adhesins were identified by comparing the genotype and its phenotypic expression under invitro. The genes were present but there is absence of phenotypic expression as Pattern I, II, III and the reason may be alteration or mutation in the specific genes in the entire genome which can be further analyzed by genomic sequences. Certain isolates showed the correct expression of existing genes as Pattern IV, V and in case where both the genes are found but either one of the gene will get expressed as Pattern VI, VII which may be because of environmental factors favoring either one of the gene to express as the influence of one gene is more than the other or mutation of any one of the genes or based on binding receptor factor.

In contrast to the other patterns of expressing the respective gene products, isolates possessing papG expressed MSHA (Type 1 fimbriae) and fimH gene expressed MRHA (P fimbriae). The phenotypic method of hemagglutination was repeated for those particular isolates, but the result being the same as earlier. This is the new information obtained, it depicts as there might be cross interaction between the fimbriae that the genetic codons codes for one fimbriae gets transferred to other bacteria getting combined or mutated, both of which expresses different phenotype, the non-fimbrial adhesins would have bind to the RBCs and reveals this interchanged reaction, studies should be conducted for the in-depth knowledge to know their function and significance.

CONCLUSION

More studies should be carried out on fimH genes to get clear idea of its specific characteristics as a commensal and to be as a pathogenic strain in a host cell. And also the intercross phenotypic expression of the genes must be studied further to know their significance.

References

1. Ronald A, The etiology of urinary tract infection: traditional and emerging pathogens. *Am J Med*. 2002; 8:113
2. L. Siegfried, Marta Kmeiovat, Hana puzova, Maria Molokacovas and J. Filkag. Virulence-associated factors in Escherichia co/i strains isolated from children with urinary tract infections. *J. Med. Microbiol*. 1994; 41: 127-132
3. Georgi Slavchev, Emiliya Pisareva2 and Nadya Markova. Virulence of uropathogenic *Escherichia coli. Journal of culture collections. 2009; 6: 3-9*
4. Thomas E. Andersen, Surabhi Khandige, Michelle Madelung, Jonathan Brewer, Hans J. Kolmos and Jakob Moller-Jensen. *Escherichia coli* Uropathogenesis *In Vitro*: Invasion, Cellular Escape, and Secondary

Infection Analyzed in a Human Bladder Cell Infection Model Infection and Immunity 2015; 1858-1867
5. Beurton, Peter, Raphael Falk, Hans-Jorg Rheinberger., eds. *The Concept of the Gene in Development and Evolution*. Cambridge, UK: Cambridge UniversityPress, 2000.
6. J. Vijayalakshmi, R. Hymavathi, A. Renuka Devi, M. V. Ramanamma. G. Swarnalatha, A. Surekha, V. Anitha Lavanya, R. Somasekhar "Study of Virulence Factors in Uropathogenic Escherichia Coli". *Journal of Evolution of Medical and Dental Sciences* 2015; 4(8): 1297-1305.
7. Farzaneh Firoozeh, Mahmood Saffari, Foroogh Neamati, Mohammad Zibaei. Detection of virulence genes in Escherichia coli isolated from patients with cystitis and pyelonephritis. *International Journal of Infectious Diseases*, 2014; 29: 219-222
8. Johnson, j.r.; kuskowski, m.a.; gajewski, a. et al. - Extended virulence genotypes and phylogenetic background of Escherichia coli isolates from patients with cystitis, pyelonephritis, or prostatitis. *J. infect. Dis*., 2005; 191: 46-50.
9. Farmer JJ. Enterobacteriaceae: introduction and identification (1999). In: Murray PR, Baron EJ, Phaller MA, Tenover FC, Yolken RH, editors. Manual of clinical microbiology. 7th Ed., Washington, D.C.: American Society for microbiology; 1999, 452-455.
10. Barbara Kot. Virulence Factors and Innovative Strategies for the Treatment and Control of Uropathogenic Escherichia coli. *Escherichia coli* - Recent Advances on Physiology, Pathogenesis and Biotechnological Applications. 2017; 25 - 44.
11. Bahrani-Mougeot F, et al., Uropathogenic *Escherichia coli* In Donnenberg MS (ed.), *Escherichia coli: virulence mechanisms of a versatile pathogen.* Academic Press. San Diego, Calif. 2002; 239-268.
12. Abdollah Derakhshandeh, Mohammad Motamedifar, Maryam Bahadori, Zahra Naziri, Mohsen Alinejad, Azar Motamedi Boroojeni, Roya Firouzi. Genetic relatedness of the *Escherichia coli* fecal population and strains
13. causing urinary tract infection in the same host. Microbiology Open Access by John Wiley & Sons Ltd. 2018;
14. Mandal P, Kapil A, Goswami K, Das B, Dwivedi SN. The uropathogenic Escherichia coli cause urinary tract infections. *Indian J Med Res*. 2001; 114:207-11.
15. Raksha R, Srinivasa H, Macaden RS. Occurrence and Characterisation of uropathogenic E.coli in urinary tract infections. *Ind J of Medical Microbiolgy* - 2003, Oct: 102-107.
16. Rebecca Naveen, Elizabeth Mathai. Some Virulence Characteristics of Uropathogenic E. coli in different patient groups. *Ind J of Med Res*, 2005, Aug: 143-147.
17. Dorota wojnicz. Virulence Factors of Uropathogenic Escherichia Coli Strains Isolated from Children with Chronic Pyelonephritis. Adv Clin Exp Med, 2007; 16(5): 651-657
18. Yasmeen Kausar, Sneha K Chunchanur. Characterization of Uropathogenic E. coli in relation to virulence Factors.
19. K Prabhat Ranjan, Neelima Ranjan, Arindam Chakraborty, D R Arora "An Approach to Uropathogenic *Escherichia coli* in Urinary Tract Infections". *Journal of Laboratory Physicians* 2010; 2(2)
20. Nazish Fatima, Mithlesh Agrawal, Indu Shukla and Parvez Anwar khan "Characterization of Uropathogenic E. coli in relation to virulence Factors". Open Access Sceintific Reports 2012; 1(7)
21. Shruthi N et al., Virulence Factors in *E. coli* Isolated From Antenatal Catheterized Cases Journal of Clinical and Diagnostic Research. 2012; 6(10): 1699-1703
22. Mittal S, Sharma M, Chaudhary U. Study of virulence factors of uropathogenic *Escherichia coli* and its antibiotic susceptibility pattern. *Indian J Pathol Microbiol* 2014; 57:61- 64.
23. B Vishalakshi, KR Suresh. Virulence Markers, Extended Spectrum Beta Lactamase detection and Antibiotic Susceptibility pattern of Uropathogenic E Coli in Clinically Suspected Urinary Tract Infections. *Research Journal of Pharmaceutical, Biological and Chemical Sciences*, 2014; 5(6): 72 - 81.
24. Mohsen Tabasi, Mohammad Reza Asadi Karam, Mehri Habibi, Mir Saeed Yekaninejad, Saeid Bouzari, Phenotypic Assays to Determine Virulence Factors of Uropathogenic Escherichia coli (UPEC) Isolates and their Correlation with Antibiotic Resistance Pattern. Osong Public Health Res Perspect 2015; 6(4): 261-268
25. Sugandha Srivastava, Jyotsna Agarwal, Bharti Mishra, Richa Srivastava. Virulence versus fitness determinants in *Escherichia coli* isolated from asymptomatic bacteriuria in healthy nonpregnant women. *Indian Journal of Medical Microbiology*, 2014; 34(1): 46 - 51
26. Tiba, m.r.; yano, t. & leite, d.s. Genotypic characterization of virulence factors in *Escherichia coli* strains from patients with cystitis. Rev. Inst. Med. trop. S. Paulo 2008; 50(5): 255-260.
27. Bogyiová E, Kmetová M, Biros E, Siegfried L. Detection of *pap*-, *sfa*- and *afa*-specific DNA sequences in *Escherichia coli* strains isolated from extraintestinal material. Folia Microbiol (Praha) 2002; 47:723–726.
28. F.A. Oliveira, K.S. Paludo, L.N.V.S. Arend, S.M.S.S. Farah, F.O. Pedrosa4, E.M. Souza, M. Surek, G. Picheth and C.M.T. Fadel-Picheth, Virulence characteristics and antimicrobial susceptibility of uropathogenic Escherichia coli strains. Genetics and Molecular Research, 2011; 10(4): 4114 - 4125
29. Aazam Karimian, Hassan Momtazand Mahbobe Madani. Detection of uropathogenic *Escherichia coli* virulence factors in patients with urinary tract infections in Iran. *African Journal of Microbiology Research*, 2012; 6(39): 6811-6816
30. AV Shetty, SH Kumar, M Shekar, AK Shetty, I Karunasagar "Prevalence of adhesive genes among uropathogenic *Escherichia coli* strains isolated from patients with urinary tract infection in Mangalore". *Indian Journal of Medical Microbiology* 2014; 32(2): 175-178
31. Mohajeri P, Khademi H, Ebrahimi R, Farahani A, Rezaei M. Frequency distribution of virulence factors in uropathogenic *Escherichia coli* isolated from

Kermanshah in 2011-2012. *Int J App Basic Med Res* 2014; 4:111-6.
32. Foroogh Neamati; Farzaneh Firoozeh; Mahmood Saffari; Mohammad Zibaei, Virulence Genes and Antimicrobial Resistance Pattern in Uropathogenic *Escherichia coli* Isolated From Hospitalized Patients in Kashan, Iran. *Jundishapur J Microbiol*. 2015; 8(2): e17514.
33. Maysaa El Sayed Zaki, Ahmed Elewa. Evaluation of Uropathogenic Virulence Genes in *Escherichia coli* Isolated from Children with Urinary Tract Infection. *International Journal of Advanced Research* 2015;3 (3): 165-173
34. Yandag Munkhdelger, Nyamaa Gunregjav, Altantsetseg Dorjpurev, Nishi Juniichiro, Jav Sarantuya. Detection of virulence genes, phylogenetic group and antibiotic resistance of uropathogenic *Escherichia coli* in Mongolia. *J Infect Dev Ctries* 2017; 11(1):51-57.
35. Pootong, A, Mungkornkeaw, N, Norrapong, B and Cowawintaweewat, S. Tropical Biomedicine Phylogenetic background, drug susceptibility and virulence factors of uropathogenic *E. coli* isolate in a tertiary university hospital in central Thailand. 2018; 35(1): 195-204.
36. Husam Eldin M. Hassan, Hisham N. Altayb, Mogahid M. El Hassan and Miskelyemen, Elmekki. Genotypic detection of the virulence factors of uropathogenic Escherichia coli isolated from diarrheic and urinary tract infected patients in Khartoum State, Sudan. African Journal of Microbiology Research 2018; 12(9): 230-236.
37. Bann JG, et al., Adhesive pili of the chaperone-usher family. In Donnenberg MS (ed.), *Escherichia coli: virulence mechanisms of a versatile pathogen.* Academic Press. San Diego, Calif. 2002; 289-306.

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