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Review Article

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Escherichia coli associated urinary tract infection: Epidemiology and possible strategies for control

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ABSTRACT

Urinary tract infection (UTI) is a prevalent condition that individuals may experience at least once in their lifetime. It is one of the most common reasons for hospital visits across all age groups, from neonates to adults. The predominant organism causing UTIs is *Escherichia* (*E.*) *coli*, followed by other microorganisms such as *Klebsiella pneumoniae*, *Staphylococcus saprophyticus*, *Citrobacter* spp., *Pseudomonas aeruginosa*, and *Proteus* spp. This review focuses on *E. coli* as the predominant causative agent for UTIs, examining its contribution to the disease burden and antibiotic susceptibility which significantly impact on human health and society. Additionally, we discuss novel approaches to combat this common threat, including the development of bio-markers for UTI treatment, the application of AI, and nanotechnology in medical field to fight against UTIs. We also observe the global distribution of uropathogenic *E. coli*, with specific attention to India, and highlight the recent trends in drug resistance patterns among the uropathogenic *E. coli* isolates enabling physicians to administer appropriate antibiotics for UTI treatment.

KEYWORDS: UTI; Uropathogenic *E. coli*; Antibiotic susceptibility; Bio-markers; Artificial Intelligence; Nanotechnology

1. Introduction

Urinary tract infection (UTI) is a commonly occurring disease caused by bacteria, which refers to the presence of microbial pathogens in the urinary tract. Depending on the site of infection, UTIs are mainly classified as bladder infection (cystitis), kidney infection (pyelonephritis) and urine infection (bacteriuria)[1]. Based on the cause of the disease, UTIs are generally classified into two major categories: i) Community acquired UTIs and ii) Hospital acquired UTIs[2]. The predominant organism in both community and hospital acquired cases is *Escherichia* (*E.*) *coli*[3−5]. Besides *E. coli*, other uropathogens causing community acquired UTIs are *Staphylococcus* (*S.*) *saprophyticus*, *Klebsiella* (*K.*) *pneumoniae*, and *Citrobacter* spp.[6–8]. Hospital acquired UTIs result from a variety of organisms besides *E. coli* like *Pseudomonas* (*P.*) *aeruginosa* and *Proteus* spp., which are rarely found in community-acquired UTIs[9,10].

Currently, an increased resistance has been observed in uropathogenic *E. coli* (UPEC) towards almost all commonly prescribed antibiotics, highlighting a serious medical issue. Due to this situation, UTI treatment has become quite challenging nowadays. In bacterial populations like *E. coli*, antibiotic resistant genes of *Staphylococcus* can spread very rapidly due to the fact that they are mainly carried on plasmids and mobile genetic elements. Moreover, increased medical tourism phenomena (phenomena of international travel for obtaining health-care) have also significantly contributed towards emergence of highly resistant UPEC strains across the world[11]. It has been recently reported that UPEC show increased resistance towards trimethoprim-sulfamethoxazole, being regarded as the first-choice antibiotic with respect to treating uncomplicated UTIs[12]. The resistance among UPEC isolates towards trimethoprim-sulfamethoxazole presently varies from 14.6% to 60.0% across all European nations[12]. Moreover, due to excessive administration of fluoroquinolones for UTI treatment, resistance among uropathogenic *E. coli* isolates towards fluoroquinolones have also shown significant increase worldwide[13]. It has been observed that UPEC isolates display greater resistance towards fluoroquinolones in developing nations, ranging from 55.5 to 85.5%

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as compared to developed nations where the resistance observed has been recorded around 5.1% to 32.0%[12]. This increasing trend of antibiotic resistance observed among UPEC immediately calls for the development and implementation of strict antibiotic prescribing policy worldwide for countering the biggest fear of emerging antibiotic-resistant UPEC isolates.

E. coli, a Gram-negative, rod-shaped, facultative anaerobe, coliform bacteria belonging to genus *Escherichia*, generally occurs in the lower intestine of warm-blooded animals (endotherms)[14,15]. The German Paediatrician Theodor Escherich first discovered *E. coli* during 1885 in the intestinal flora of infants which was later named after him in the year 1920[16]. *E. coli* is the predominant microbe of the human colonic flora which colonises the infant gastrointestinal tract within hours of life and thereby live as commensals[17]. Most strains of *E. coli* remain harmless, but certain groups like Enterotoxigenic *E. coli* and Enteropathogenic *E. coli* can cause serious food poisoning and sometimes may also lead to food contamination incidents inside its host[18,19].

A specialized strain of *E. coli*, known as Extraintestinal pathogenic *E. coli* (ExPEC), causes a variety of extraintestinal infection like UTIs, accounting for significant morbidity, lost productivity and increased health-care costs[20–22]. A typical example of Extraintestinal pathogenic *E. coli* is O157:H7 serotype. A majority proportion of bacterial population comprise of *E. coli* exhibiting enormous amount of both genetic and phenotypic diversity. *E. coli* is the most diverse bacterial species, with 20% of its genes share among all strains[23]. A group of closely related microbes that are distinguished by a common set of antigens is referred to as serotype. Presently, more than 700 different serotypes of *E. coli* are known, most of which are harmless. However, some serotypes such as O5 have been reported to cause approximately 29.3% of all UTI cases, followed by O17 and O25 (each accounting for 5.3%). In 2001, the discovery of *E. coli* isolates O11/O77/O17/O73:K52:H18-ST69 accounted for 11.0% of total *E. coli* caused UTIs[24,25].

On a molecular basis, *E. coli* has been grouped into four main phylogenetic categories: A, B1, B2 and D. Group B2 is predominate and group D is less extensive, accounting for most of the extraintestinal pathogenic *E. coli* causing UTIs[26–29]. The remaining two groups (A & B1) comprise harmless *E. coli* strains[30].

Since this was a narrative review instead of a systematic or meta-analytical review, no planned strategy was designed to screen the distribution of uropathogenic *E. coli* all across the world in an organised way. A random approach was followed to screen the distribution of UPEC isolates globally, with greater emphasis placed on South-East Asian nations, particularly India, which attributes significantly to world's population.

2. Global distribution of *E. coli* causing UTIs

2.1. ExPEC strains and prevalence

There are various lineages of ExPEC strain causing UTIs, but only one group causes the majority of the infections. According to the report of Manges *et al*. from 1995 to 2018 on *E. coli* obtained from extraintestinal infections and the gut, it was found that about 20 main ExPEC sequence types accounted 85% of all *E. coli* isolates. Out of these, ST131 became the generally occurring ExPEC sequence from year 2000 onwards, covering almost all geographical regions. The similarity in distribution of ExPEC sequence types was observed in Europe and North American studies while divergence was observed among Asian and African studies[31].

2.2. Epidemiology of serotype

A study related to molecular epidemiology of O15:K52:H1 ExPEC on global scale was conducted in Europe, indicating that sixteen (21%) out of 75 non-European O15 isolates showed similarity with the predominate serotype O15:K52:H1 in Europe. These sixteen non-European origin serotypes O15:K52:H1 were found to occur in diverse geographical regions. The site of their isolation was from extraintestinal infections in humans, accounting for 50.0% of all O15

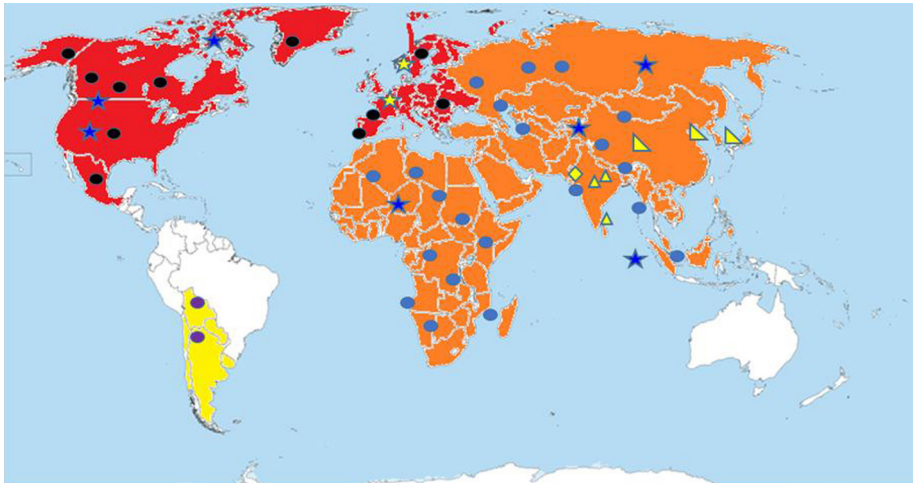

Figure 1. Global distribution of different strains of uropathogenic *E. coli*. ● ST131 in Europe and North America; ● ST131 in Afria and Asia; ☆ O15K52-H1 in Europe; ★ O15 *E. coli in* non-Europe countries; ◇ CTXM15 *E. coli* in Indian continent; ◺ CTXM14 *E. coli* in South Korea, China and Japan; △ O157:H7 *E. coli in* West Bengal, Mizoram, Andaman, and Nicobar Island; ● *bla*-CTXM-2 *E. coli* in Argentina.

isolates obtained during studies of five human clinical collections. This indicated that O15:K52:H1 serotype is extensively found beyond Europe, exhibiting previously unrecognized phenotypic and genotypic diversity, which greatly contributed causing extraintestinal infections in humans[32].

3. Distribution of *E. coli* causing UTIs in South–East Asia and India

In South-East Asia, the major causative agent for UTIs as well as bloodstream infection is extended-spectrum β-lactamase (ESBL) producing *E. coli* and *K. pneumoniae*[33]. An increasingly greater rate of more than 50.0% ESBL-producing *E. coli* and *Klebsiella* spp. occured in specific regions of Asia, Africa, and Latin America[34]. Presently, there are more than 220 different enzymes of CTX-M-β-lactamase which are divided into five subgroups on basis of their amino acid identity like CTX-M-1, CTX-M-2, CTX-M-8, CTX-M-9, and CTX-M-25[35]. Among these, the most abundantly occurring CTX-M enzymes include the CTX-M-15, which belongs to CTX-M-1 subgroup was first reported in *E. coli* from India in 2001 and also CTX-M-14, which belongs to CTX-M-9 subgroup and was discovered in 2001 from *E. coli*, *K. pneumoniae*, and *Shigella* spp. in Korea[36,37].

In South-East Asian nations like China, South Korea, and Japan, the most common ESBL is CTX-M-14-producing *E. coli*, while in South American nations like Argentina, *bla*CTX-M-2 like Enterobacteriaceae are generally found[38].

During a study conducted for ten years on *E. coli* O157:H7 serotype (one of the most prevalent causative agent for UTIs) from 1996-2005, it was found that out of 17 093 *E. coli* isolates, 242 were O157:H7. From the total *E. coli* isolates received annually, the percentage of O157:H7 ranged from 0.07% (lowest) to 3.60% (highest) during the year 1997 and 2002, respectively[39].

When geographical distribution of O157:H7 was studied in India during the 10-year survey, it was found that O157:H7 was predominately found in almost all parts of the country due to open trade and frequent dispersal among different states. Most of the cases related to *E. coli* O157 infection were reported from West Bengal (infectivity rate of 4.7%) along with north-eastern state like Mizoram (infectivity rate of 5.9%) and the Andaman and Nicobar Islands (infectivity rate of 4.2%). It may attributed to the more seafood consumption in these states and the major reservoir for O157:H7 serotype of *E. coli* are seafoods[39].

4. Antimicrobial resistance patterns in South–East Asia and India

Antimicrobial resistance presents a significant challenge to public health. Hoban *et al*. demonstrated that the antibiotic resistance patterns in UTI-causing *E. coli* serotypes. ESBL-negative isolates were found to have 90.0% inhibition rates when treated with amikacin and piperacillin–tazobactam antibiotics while ESBL-positive *E. coli* serotypes were largely resistant to ciprofloxacin and levofloxacin, showing just 14.6% and 15.9% susceptibility, respectively[40]. Similarly, another study conducted by Bryce *et al*. on occurrence of antibiotic resistance patterns worldwide among paediatric UTI cases caused by *E. coli*, indicating that out of 77 783 *E. coli* isolates in urine obtained from Organisation for Economic Co-operation and Development (OECD) countries, the pooled prevalence of resistance was 53.4% for ampicillin, 23.6% for trimethoprim, 8.2% for co-amoxiclav and 2.1% for ciprofloxacin while nitrofurantoin showed the lowest resistance of 1.3%. The reports from outside the OECD nations were significantly higher, with rates of 79.8% for ampicillin, 60.3% for co-amoxiclav, 26.8% for ciprofloxacin and 17.0% for nitrofurantoin[41].

According to Hara *et al*., during molecular characterization of multi-drug resistant *E. coli* isolates obtained from tropical environments of Southeast Asia, it was found that among 129 *E. coli* isolates occurring in lowland aqueous environments near hospitals and medical service centers of Kuala Lumpur, Malaysia, 11 *E. coli* strains were extremely resistant towards trimethoprim-sulfamethoxazole (83.7%) and nalidixic acid (71.3%) while moderate resistance was shown against ampicillin and chloramphenicol (66.7%), tetracycline (65.1%), fosfomycin (57.4%), cefotaxime (57.4%), as well as ciprofloxacin (57.4%). The level of resistance was found to be low with aminoglycosides like kanamycin (22.5%) and gentamicin (21.7%)[42]. Similarly, in another study conducted on monitoring the high abundance of *E. coli* ST131 in Southeast Asia, it was found that ST131 multilocus sequence type of *E. coli* is rapidly spreading the phenomena of antibiotic resistance on global scale[43]. In this study, almost all the C2 strains formed the SEA-C2 clone. The SEA-C2 clone was particularly derived from Asian sub-continent mainly from Southeast Asia and Singapore. The SEA-C2 clone attributes impart all the excess resistance and virulence to ST131 *E. coli* in comparison to its non-ST131 counterpart. The SEA-C2 strains displayed local circulation and dominance in Southeast Asia, putting an end to the myth that frequent migration and travel would also impart opportunities to other strains for establishing themselves[43].

A study highlighting resistance to carbapenem and colistin among Enterobacteriaceae isolates from Southeast Asia found that carbapenem-resistant Enterobacteriaceae infections have been reported on a global scale while emerging colistin resistance has further aggravated this threat in South-East Asia since 3 out of eleven countries have filed their reports on carbapenem and colistin resistance to World Health Organization[44]. The study found that lower resistance prevailed among *E. coli* isolates, except in two of seven countries, Indonesia and Myanmar, where more than a hundred *E. coli* strains displayed high resistance. The most commonly found carbapenems were New Delhi metallo-β-lactamase and Oxacillin-hydrolyzing β-lactamase. Furthermore, polymyxin resistance was

observed among eight out of eleven countries, showing *mcr–1* as a predominant genotype. This broadened spectrum of carbapenem and polymyxin resistance posed a significant risk, intensifying concerns about development of antibiotic resistance towards these two classes in generally occurring *E. coli* infections[44].

During a study conducted regarding the etiology and antibiotic resistance pattern among community-acquired UTI cases at JNMC Hospital, Aligarh, India, it was found that majority of the community-acquired UTI cases were caused by *E. coli*, showing an increased resistance towards co-trimoxazole, along with the development of extended spectrum β-lactamase activity in UTI pathogens of the community[45]. Similarly, another report by Kothari *et al*. on antibiotic resistance pattern observed among pathogens causing community-acquired UTIs in India; it was found that out of 531 samples, *E. coli* accounted for the highest percentage (68.0%) among all UTI isolates and showed antibiotic susceptibility to amikacin at a rate of 67.0%, amoxicillin at 14.7%, amoxicillin/clavulanate at 40.8%, ciprofloxacin at 28.0%, cotrimoxazole at 26.0%, nitrofurantoin at 75.6%, piperacillin-tazobactum at 90.3%, and meropenem at 100%[46].

During a retrospective study on antibiotic resistance pattern among UTI causing microbes at a Tertiary Care Hospital in South India, out of the 896 urine samples, it was found that *E. coli* (52.6%) was the predominant microorganism, showing the least resistance to imipenem (8.0%) and amikacin (16.0%), while showing higher resistance to co-trimoxazole (69.0%) and ampicillin (86.0%). Thus, it is crucial to recognize the regional trend in antibiotic resistance pattern, enabling the physicians to provide effective treatment for UTI cases[47].

5. Strategies for eliminating antimicrobial resistance during UTI treatment

5.1. Natural and alternative therapies

Since *E. coli* isolates from UTI patients have developed resistance to various antibiotics, it is essential to develop some novel strategies for combating UTIs. Some novel approaches have been enacted to overcome this problem, including the use of cranberry juice which has a higher acid content that causes acidification of the urine and thereby protects against UTIs[48]. Cranberry juice was first administered during 1994 to elderly women residing of nursing homes showing incidence of asymptomatic bacteriuria with pyuria. The cranberry juice significantly reduced the aforesaid symptoms among patients around four weeks of its administration[48]. Here, *E. coli* accounted for 43% of all positive cultures, which significantly reduced due to ingestion of cranberry juice[49]. Similarly, UTIs can be prevented through protective lactobacilli by interfering with bacterial attachment, thereby restoring normal vaginal flora[50–56]. Reid *et al*. was the first to observe the phenomena of decrease in UTI episodes through administration of lactobacillus strain L rhamnosus GR-1 intravaginally[57,58].

Development of vaccines can also be an effective novel approach to reduce antibiotic resistance among *E. coli* isolates causing UTIs. SolcoUrovac, which is a mixture of ten heat-killed bacteria namely six uropathogenic *E. coli* with one *Proteus mirabilis*, one *Proteus morganii*, one *Enterococcus faecalis* and one *Klebsiella pneumoniae*, has been found to act as whole-cell vaccine[59]. It has been reported that women receiving all 6 doses and boosters of this vaccine for a period of around four months did not develop UTIs, along with no side-effects also reported for around 160 days[60]. Another vaccine named Uro-Vaxom, which comprises 18 serotypes of immune stimulating fractions from *E. coli*, has been successfully tested in Europe[61]. Similarly, a parenteral vaccine-based approach for preventing *E. coli* associated UTIs based on interaction of type 1 fimbriae with host uroepithelial cells, is currently being developed[62].

5.2. Novel therapies and biomakers

The technique of bacterial interference, which involves deliberate colonization of human urinary bladder with benign *E. coli* isolate in persons suffering from spinal cord injury, has been proven to be a quite useful technique in drastically decreasing the recurrence of UTIs. This technique works using competitive inhibition of colonization of the urinary bladder with other virulent strains causing UTIs[63–66]. Recently, the prevention of UTIs, along with treatment of established infections, has been achieved by using oligosaccharide-based competitive inhibitors targeting bacterial adherence, colonization and infection at mucosal sites[67,68].

A biomarker is a naturally occurring molecule or gene that helps in the identification of a particular disease or any pathological or physiological process. Serum and urine biomarkers for predicting recurrent UTIs have become quite popular nowadays. The first possible biomarker found in recurrent UTIs are serum antibodies, while for lower urinary tract diseases, urinary biomarkers has been widely used for years[69].

6. Conclusions

UTIs are generally caused by *E. coli*, and different studies have shown an alarming increase in antibiotic resistance in this bacterium towards commonly prescribed antibiotics[70–75]. Previous studies have demonstrated the drug-resistance pattern among UPEC isolates, which are considered extracellular pathogens, as they can survive and replicate intracellularly within epithelial cells and macrophages. The novel strategies to combat antibiotic resistant UPEC include cranberry juice, vaccinet, probiotics, phage therapy and biomakers. In addition, Artificial Intelligence and nanotechnology in the medical field are consistently and prominently encouraged to combat UTIs

without the risk of antibiotic resistance.

Despite the fact that *E. coli* strains are useful to us as they live as commensals in our intestine and synthesise many vital products such as Vitamin-K2, which helps in blood clotting[76], the increasing antimicrobial resistance has weakened or suppressed the immunity of most persons, resulting in harmless *E. coli* strains nowadays also causing infections. That's why, better and safer alternatives for UTI treatment should be implemented as much as possible to control the risks of antibiotic resistance development. These new methods for countering antibiotic resistance among UPEC isolates will emerge as the most effective tool for treating UTI cases in the near future and replace the antibiotic therapy. In this way, the severity of antibiotic resistance in UPEC isolates can be completely controlled and suppressed.

Conflict of interest statement

The authors declare that there is no conflict of interest.

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Data availability statement

The data supporting the findings of this study are available from the corresponding author upon request.

Authors' contributions

Biswas S and Rana R contributed to the conception and design of the manuscript. Ranjit M and Bal M revised the manuscript and supplemented materials for preparing the manuscript thoroughly. Pati S and Suar M completed the manuscript. All the authors agree with the content of the manuscript.

References

[1] Foxman B. Epidemiology of urinary tract infections: Incidence, morbidity, and economic costs. *Am J Med* 2002; **113**: 5-13.

[2] Shalini MA, Prabhakar K, Lakshmi SY. A Study on prevalence and evaluation of clinical isolates from community acquired infections using different media in semiurban areas. *World J Med Sci* 2010; **5**: 49-53.

[3] Dias Neto JA, Martins AC, Silva LD, Tiraboschi RB, Domingos AL, Cologna AJ, et al. Community acquired urinary tract infection: Etiology and bacterial susceptibility. *Acta Cir Bras* 2003; **18**: 33-36.

[4] Barros IC, Ribeiro AD, Costa AC, Nunes DC, Neres KD, Carneiro DS, et al. Microorganisms prevalent in urinary tract infections and antimicrobial sensitivity profile: Analysis of patients attended at the Military Police Hospital of the State of Goiás, Brazil, in the period from 1998 to 2008. *J Health Sci Inst* 2011; **29**(4): 243-247.

[5] Nerurkar A, Solanky P, Naik SS. Bacterial pathogens in urinary tract infection and antibiotic susceptibility pattern. *J pharm biomed sci* 2012; **21**(12): 1-3.

[6] Gyansa-Lutterodt M, Afriyie D, Asare G, Amponsah S, Abutiate H, Darko D. Antimicrobial use and susceptibility pattern of uropathogens associated with urinary tract infections at the Ghana Police Hospital. *Glob J Pharmacol* 2014; **8**: 306-315.

[7] Afriyie DK, Gyansa-Lutterodt M, Amponsah SK, Asare G, Wiredu V, Wormenor E, et al. Susceptibility pattern of uropathogens to ciprofloxacin at the Ghana Police Hospital. *Pan Afr Med J* 2015; **22**(87): 6037 .

[8] Bahadin J, Teo SS, Mathew S. Aetiology of community-acquired urinary tract infection and antimicrobial susceptibility patterns of uropathogens isolated. *Singapore Med J* 2011; **52**(6): 415.

[9] Mittal R, Aggarwal S, Sharma S, Chhibber S, Harjai K. Urinary tract infections caused by *Pseudomonas aeruginosa*: A mini review. *J Infect Public Health* 2009; **2**(3): 101-111.

[10] Ronald A. The etiology of urinary tract infection: Traditional and emerging pathogens. *Am J Med 2002*; **113**(1): 14-19.

[11]Totsika M, Gomes Moriel D, Idris A, A Rogers B, J Wurpel D, Phan MD, et al. Uropathogenic *Escherichia col*i mediated urinary tract infection. *Curr Drug Targets* 2012; **13**(11): 1386-1399.

[12]Kot B. Antibiotic resistance among uropathogenic. *Pol J Microbiol* 2019; **68**(4): 403-415.

[13]Haghighatpanah M, Mojtahedi A. Characterization of antibiotic resistance and virulence factors of *Escherichia coli* strains isolated from Iranian inpatients with urinary tract infections. *Infect Drug Resist* 2019; **12**: 2747-2754.

[14]Tenaillon O, Skurnik D, Picard B, Denamur E. The population genetics of commensal *Escherichia coli*. *Nat Rev Microbiol* 2010; **8**(3): 207-217.

[15]Singleton P. *Bacteria in biology, biotechnology and medicine*. Hoboken: John Wiley & Sons; **2004**.

[16]Berg HC. *E. coli in motion*. New York: SpringerLink; **2004**.

[17]Drasar BS, Hill MJ. *Human intestinal flora*. New York: Academic Press; **1974**.

[18]Centers for Disease Control and Prevention. *National center for emerging and zoonotic infectious diseases: National enteric disease surveillance*. [Online] Available from: https://www.cdc.gov/ncezid/index.html. [Accessed on 29 July 2024].

[19]Vogt RL, Dippold L. *Escherichia coli* O157: H7 outbreak associated with consumption of ground beef, June–July 2002. *Public Health Rep* 2005; **120**(2): 174-178.

[20]Peng Z, Wang X, Huang J, Li B. *Pathogenic Escherichia coli* in molecular medical microbiology. New York: Academic Press; 2024.

[21]Hu J, Afayibo DJ, Zhang B, Zhu H, Yao L, Guo W, et al. Characteristics, pathogenic mechanism, zoonotic potential, drug resistance, and prevention of avian pathogenic *Escherichia coli* (APEC). *Front Microbiol* 2022; 13: 1049391.

[22]Gaitonde S, Malik RD, Zimmern PE. Financial burden of recurrent urinary tract infections in women: A time-driven activity-based cost analysis. *Urology* 2019; **128**: 47-54.

[23]]Lukjancenko O, Wassenaar TM, Ussery DW. Comparison of 61 sequenced *Escherichia coli* genomes. *Microb Ecol* 2010; **60**: 708-720.

[24]Okeke IN, de Kraker ME, Van Boeckel TP, Kumar CK, Schmitt H, Gales AC, et al. The scope of the antimicrobial resistance challenge. *Lancet* 2024; **403**(10442): 2426-2438.

[25]Manges AR, Johnson JR, Foxman B, O'Bryan TT, Fullerton KE, Riley LW. Widespread distribution of urinary tract infections caused by a multidrug-resistant *Escherichia coli* clonal group. *N Engl J Med* 2001; **345**(14): 1007-1013.

[26]Bingen-Bidois M, Clermont O, Bonacorsi S, Terki M, Brahimi N, Loukil C, et al. Phylogenetic analysis and prevalence of urosepsis strains of *Escherichia coli* bearing pathogenicity island-like domains. *Infect Immun* 2002; **70**(6): 3216-3226.

[27]Bonacorsi S, Clermont O, Houdouin V, Cordevant C, Brahimi N, Marecat A, et al. Molecular analysis and experimental virulence of French and North American *Escherichia coli* neonatal meningitis isolates: Identification of a new virulent clone. *J Infect Dis* 2003; **187**(12): 1895-1906.

[28]Johnson JR, Stell AL. Extended virulence genotypes of *Escherichia coli* strains from patients with urosepsis in relation to phylogeny and host compromise. *J Infect Dis* 2000; **181**(1): 261-272.

[29]Johnson JR, O'Bryan TT, Delavari P, Kuskowski M, Stapleton A, Carlino U, et al. Clonal relationships and extended virulence genotypes among *Escherichia coli* isolates from women with a first or recurrent episode of cystitis. *J Infect Dis* 2001; **183**(10): 1508-1517.

[30]Duriez P, Clermont O, Bonacorsi S, Bingen E, Chaventré A, Elion J, et al. Commensal *Escherichia coli* isolates are phylogenetically distributed among geographically distinct human populations. *Microbiology* 2001; **147**(6): 1671-1676.

[31]Manges AR, Geum HM, Guo A, Edens TJ, Fibke CD, Pitout JD. Global extraintestinal pathogenic *Escherichia coli* (ExPEC) lineages. *Clin Microbiol Rev* 2019; **32**(3): e00135-18.

[32]Johnson JR, Stell AL, O'Bryan TT, Kuskowski M, Nowicki B, Johnson C, et al. Global molecular epidemiology of the O15:K52:H1 extraintestinal pathogenic *Escherichia coli* clonal group: Evidence of distribution beyond Europe. *J Clin Microbiol* 2002; **40**(6): 1913-1923.

[33]Pitout JD. Extraintestinal pathogenic *Escherichia coli*: A combination of virulence with antibiotic resistance. *Front Microbiol* 2012; **3**: 9.

[34]World Health Organization. *Antimicrobial resistance: Global report on surveillance*. [Online] Available from: https://www.who.int/publications/i/item/9789241564748. [Accessed on 29 July 2024].

[35]Naas T, Oueslati S, Bonnin RA, Dabos ML, Zavala A, Dortet L, et al. Beta-lactamase database (BLDB)-structure and function. *J Enzym Inhib Med Ch* 2017; **32**(1): 917-919.

[36]Karim A, Poirel L, Nagarajan S, Nordmann P. Plasmid-mediated extended-spectrum β-lactamase (CTX-M-3 like) from India and gene association with insertion sequence ISEcp1. *FEMS Microbiol Lett* 2001; **201**(2): 237-241.

[37]Pai H, Choi EH, Lee HJ, Hong JY, Jacoby GA. Identification of CTX-M-14 extended-spectrum β-lactamase in clinical isolates of *Shigella sonnei*, *Escherichia coli*, and *Klebsiella pneumoniae* in Korea. *J Clin Microbiol* 2001; **39**(10): 3747-3749.

[38]Bevan ER, Jones AM, Hawkey PM. Global epidemiology of CTX-M β-lactamases: Temporal and geographical shifts in genotype. *J Antimicrob Chemother* 2017; **72**(8): 2145-2155.

[39]Sehgal R, Kumar Y, Kumar S. Prevalence and geographical distribution of *Escherichia coli* O157 in India: A 10-year survey. *Trans R Soc Trop Med Hyg* 2008; **102**(4): 380-383.

[40]Hoban DJ, Nicolle LE, Hawser S, Bouchillon S, Badal R. Antimicrobial susceptibility of global inpatient urinary tract isolates of *Escherichia coli*: Results from the study for monitoring antimicrobial resistance trends (SMART) program: 2009–2010. *Diagn Microbiol Infect Dis* 2011; **70**(4): 507-511.

[41]Bryce A, Hay AD, Lane IF, Thornton HV, Wootton M, Costelloe C. Global prevalence of antibiotic resistance in paediatric urinary tract infections caused by *Escherichia coli* and association with routine use of antibiotics in primary care: Systematic review and meta-analysis. *BMJ* 2016; **352**: i939.

[42]Hara H, Yusaimi YA, Zulkeflle SN, Sugiura N, Iwamoto K, Goto M, et al. Molecular characterization of multi-drug resistant *Escherichia coli* isolates from tropical environments in Southeast Asia. *J Gen Appl Microbiol* 2018; **64**(6): 284-292.

[43]Chen SL, Ding Y, Apisarnthanarak A, Kalimuddin S, Archuleta S, Omar SF, et al. The higher prevalence of extended spectrum beta-lactamases among *Escherichia coli* ST131 in Southeast Asia is driven by expansion of a single, locally prevalent subclone. *Sci Rep* 2019; **9**(1): 13245.

[44]Malchione MD, Torres LM, Hartley DM, Koch M, Goodman JL. Carbapenem and colistin resistance in Enterobacteriaceae in Southeast Asia: Review and mapping of emerging and overlapping challenges. *Int J Antimicrob Agents* 2019; **54**(4): 381-399.

[45]Akram M, Shahid M, Khan AU. Etiology and antibiotic resistance patterns of community-acquired urinary tract infections in JNMC Hospital Aligarh, India. *Ann Clin Microbiol Antimicrob* 2007; **6**(1): 1-7.

[46]Kothari A, Sagar V. Antibiotic resistance in pathogens causing community-acquired urinary tract infections in India: a multicenter study. *J Infect Dev Ctries* 2008; **2**(05): 354-358.

[47]Somashekara SC, Deepalaxmi S, Jagannath N, Ramesh B, Laveesh MR, Govindadas D. Retrospective analysis of antibiotic resistance pattern to urinary pathogens in a Tertiary Care Hospital in South India. *J Basic Clin Pharm* 2014; **5**(4): 105-108.

[48]Howell AB. Cranberry proanthocyanidins and the maintenance of urinary tract health. *Crit Rev Food Sci Nutr* 2002; **42**(S3): 273-278.

[49]]Xia JY, Yang C, Xu DF, Xia H, Yang LG, Sun GJ. Consumption of cranberry as adjuvant therapy for urinary tract infections in susceptible populations: A systematic review and meta-analysis with trial sequential analysis. *PLoS One* 2021; **16**(9): e0256992.

[50]Dempsey E, Corr SC. *Lactobacillus* spp. for gastrointestinal health: Current and future perspectives. *Front Immunol*. 2022; **13**: 840245.

[51]Mazhar SF, Afzal M, Almatroudi A, Munir S, Ashfaq UA, Rasool M, et al. The prospects for the therapeutic implications of genetically engineered probiotics. *J Food Qual* 2020; **2020**(1): 1-11.

[52]Mashatan N, Heidari R, Altafi M, Amiri A, Ommati MM, Hashemzaei M. Probiotics in vaginal health. *Pathog Dis* 2023; **81**: ftad012.

[53]Velraeds MM, van de Belt-Gritter B, Busscher HJ, Reid G, van der Mei HC. Inhibition of uropathogenic biofilm growth on silicone rubber in human urine by lactobacilli–A teleologic approach. *World J Urol* 2000; **18**: 422-426.

[54]Reid G, Bruce AW. Selection of *Lactobacillus* strains for urogenital probiotic applications. *J Infect Dis* 2001; **183**(Supplement_1): S77-80.

[55]Saimmai A, Riansa-ngawong W, Maneerat S, Dilit P. Application of biosurfactants in the medical field. *Walailak J Sci Technol* 2020; **17**(2): 154-166.

[56]Carvalho FM, Teixeira-Santos R, Mergulhao FJ, Gomes LC. Effect of *Lactobacillus* plantarum biofilms on the adhesion of *Escherichia coli* to urinary tract devices. *Antibiotics* 2021; **10**(8): 966.

[57]Hudson PL, Hung KJ, Bergerat A, Mitchell C. Effect of vaginal *Lactobacillus* species on *Escherichia col*i growth. *Urogynecology* 2020; **26**(2): 146-151.

[58]Sadahira T, Wada K, Araki M, Mitsuhata R, Yamamoto M, Maruyama Y, Iwata T, Watanabe M, Watanabe T, Kariyama R, Nasu Y. Efficacy of *Lactobacillus* vaginal suppositories for the prevention of recurrent cystitis: A phase II clinical trial. *Int J Urol* 2021; **28**(10): 1026-1031.

[59]Reddy S, Chitturi C, Yee J. Vaccination in chronic kidney disease. *Adv Chronic Kidney Dis* 2019; **26**(1): 72-78.

[60]Stephenson J. Experimental vaccine for recurrent UTIs. *JAMA* 2002; **287**(6): 702-703.

[61]Loubet P, Ranfaing J, Dinh A, Dunyach-Remy C, Bernard L, Bruyère F, et al. Alternative therapeutic options to antibiotics for the treatment of urinary tract infections. *Front Microbiol* 2020; **11**: 1509.

[62]Stapleton A. Novel approaches to prevention of urinary tract infections. *Infectious Disease Clinic*s 2003; **17**(2): 457-471.

[63]Nielsen KL, Stegger M, Kiil K, Lilje B, Ejrnæs K, Leihof RF, et al. *Escherichia coli* causing recurrent urinary tract infections: Comparison to non-recurrent isolates and genomic adaptation in recurrent infections. *Microorganisms* 2021; **9**(7): 1416.

[64]Darouiche RO, Donovan WH, Del Terzo M, Thornby JI, Rudy DC, Hull RA. Pilot trial of bacterial interference for preventing urinary tract infection. *Urology* 2001; **58**(3): 339-344.

[65]Darouiche RO, Hull RA. Bacterial interference for prevention of urinary tract infection: An overview. *J Spinal Cord Med* 2000; **23**(2):136-141.

[66]Roth JD, Pariser JJ, Stoffel JT, Lenherr SM, Myers JB, Welk B, et al. Patient subjective assessment of urinary tract infection frequency and severity is associated with bladder management method in spinal cord injury. *Spinal cord* 2019; **57**(8): 700-707.

[67]Hoyos P, Perona A, Juanes O, Rumbero Á, Hernáiz MJ. Synthesis of glycodendrimers with antiviral and antibacterial activity. *Chem Eur J* 2021; **27**(28): 7593-7624.

[68]Cummings RD. Stuck on sugars–how carbohydrates regulate cell adhesion, recognition, and signaling. *Glycoconj J* 2019; **36**: 241-257.

[69]Jhang JF, Kuo HC. Recent advances in recurrent urinary tract infection from pathogenesis and biomarkers to prevention. *Tzu Chi Med J* 2017; **29**(3): 131.

[70]Duława J. Urinary tract infection-2003. *Rocz Akad Med Bialymst* 2004; **49**: 182-184.

[71]Obiogbolu CH, Okonko IO, Anyamere CO, Adedeji AO, Akanbi AO, Ogun AA, et al. Incidence of urinary tract infections (UTIs) among pregnant women in Akwa metropolis, Southeastern Nigeria. *Sci Res Essays* 2009; **4**(8): 820-824.

[72]Behzadi P, Behzadi E. The microbial agents of urinary tract infections at central laboratory of Dr. Shariati Hospital, Tehran, Iran. *Turk Klin Tip Bilim* 2008; **28**(4): 445.

[73]Mohamed AH, Yusuf Mohamud MF, Mohamud HA. Epidemiology and antimicrobial susceptibility pattern of uropathogens in patients with the community-and hospital-acquired urinary tract infections at a tertiary hospital in Somalia. *Jundishapur J Microbiol* 2020; **13**(9): 1-7.

[74]Ebie MY, Kandakai-Olukemi YT, Ayanbadejo J, Tanyigna KB. Urinary tract infections in a Nigerian military hospital. *Niger J Microbiol* 2001; **15**(1): 31-37.

[75]Garofalo CK, Hooton TM, Martin SM, Stamm WE, Palermo JJ, Gordon JI, et al. *Escherichia coli* from urine of female patients with urinary tract infections is competent for intracellular bacterial community formation. *Infect Immun* 2007; **75**(1): 52-60.

[76]Abby SS, Kazemzadeh K, Vragniau C, Pelosi L, Pierrel F. Advances in bacterial pathways for the biosynthesis of ubiquinone. *Biochim Biophys Acta Bioenerg* 2020; **1861**(11): 148259.

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