Iinternational Journal of Medical Sciences; May 2022;5(2):33-43;
ISSNe 2522-7386 DOI: http://doi.org/10.32441.ijms.5.2.5

# Blood Groups, IL-6, IL-8 and HS-CRP Levels in Non-Pregnant Women With Urinary Tract Infection Caused by *Escherichia coli*

**Sahlah Khorshed Abbas, Department of Biology, Collage of Science ,University of Kirkuk, Iraq**
**Email: sahlalmmuno@uokirkuk.edu.iq**
**Mobile: +9647701340654**
**ORCID: https://orcid.org/0000-0001-8826-581X**
**Najdat B. Mahdi, Department of Biology, Collage of Science ,University of Kirkuk, Iraq**
**Email: drnajdat60@uokirkuk.edu.iq**
**Mobile: +9647702374397**
**ORCID: https://orcid.org/0000-0002-4954-221X**
**Khalaf J. Ahmed, Department of Biology, Collage of Science ,University of Kirkuk, Iraq**
**Email: khlfjrad3@gmail.com**
**Mobile: +9647702184523**
**ORCID: https://orcid.org/0000-0003-1307-0537**

**Correspondence author: Sahlah Khorshed Abbas, Department of Biology, Collage of Science , University of Kirkuk, Iraq. Email: sahlalmmuno@uokirkuk.edu.iq**
**Mobile: +9647701340654, ORCID: https://orcid.org/0000-0001-8826-581X**

**Received:16 /1 /2022 Accepted: 2/4/2022 Published: May 2022**

**Abstract**
**Background:** A urinary tract infection (UTI) is an infectious disease infects the urinary tract which the bladder, urethra, two ureter, and two kidneys and women are more prone to developing UTI.
**Objectives**: This study was aimed to evaluate the relationship between ABO blood groups and UTI, and investigates serum and urine concentration of IL-6, IL-8 and serum HS-CRP in non-pregnant women patients with UTI (*E. coli*).
**Method:** This study was conducted on 400 urine samples collected from patients admitting to Azadi teaching hospital and General Kirkuk hospital labs in Kirkuk city from December 2015 to May 2016. The frequency distribution of ABO in various phenotypes was assessed. **S**erum level of HS-CRP**,** serum and urine levels of Il-6, Il-8 were assessed in 36 UTI patients caused by *E. coli*, as well as 15 apparently healthy women**.**
**Results:** The most common bacteria causing UTI was *Escherichia coli* (78.23%), with the highest frequencies observed in two blood groups of A (53.6%) and O(33%). Serum concentration of HS-CRP, serum and urine concentrations of IL-6 and IL-8 were increased significantly ($p<0.00$) in women infected with UTI caused by *E coli* compared to controls. A positive correlation between the serum IL-6 and HS-CRP was found($r=0.86$).

**Conclusion**: The based on our results showed that the serum and urine IL-6, IL-8 contributes in immune response and inflammation of UTI caused by *E coli*, and the blood group A and O had a higher prevalence rate.

**Keywords: Urinary tract infection, *Escherichia coli*, non pregnant women, IL-6, IL-8, HS-CRP.**

## Introduction

Urinary tract infection (UTI) is the most common bacterial infection in women, especially in those aged 24 years or younger. An estimated 1 out of 3 women have experience with UTI in both serious complications (nephritis) and un-complicated (cystitis)[1,2]. Midstream urine samples obtained from patients indicate infection when bacterial growth exceeds 10,000 organisms per milliliter. *Escherichia coli* (*E. coli*), being the most common cause of UTI [3], has virulence factors such as toxin A moiety of the lipopolysaccharid outer membrane and P fimbria (pili) that are common to all gram negatives [4].

ABO blood group antigens may act as sites for attachment of microbes [5]. Blood group antigens are a group of carbohydrate determinants situated on lymphocytes, phagocytes, erythrocytes and certain epithelial tissues including urothelium [6]. The precursor substance to the ABO blood group antigens existing in populations of all common blood types, is called H antigen. When H antigen is lacking, people lack A or B antigens also; the H antigen acts as a precursor for producing A and B antigens. The H locus is located on chromosome 19 and involves three exons that encode fucosyltransferase which secrets the H antigen on RBCs. The H antigen composes of a chain of β-D-galactose, β-D-N-acetyl glucosamine, β-D-galactose, and 2-linked, α-L-fucose, the chain being connected to the protein. The A allele codes a glycosyltransferase that bonds α-N-acetylgalactosamine to the D galactose end of the H antigen, producing the A antigen. The B allele codes a glycosyltransferase that bonds α-D-galactose to the D- galactose end of the H antigen, creating the B antigen [7,8].

The innate immune mechanism responds quickly to non-specific pathway of infection in the urinary tract, which involves Toll-Like Receptors (TLRs), antimicrobial peptides (AMPs), cytokines and immune cells that respond to flagella, fimbriae and the lipopolysaccharides (LPS) outer membrane of these bacteria[9]. Cytokine response in UTI involves interleukin-6 (IL-6) and interleukin-8 (IL-8). IL-6 is anti-inflammatory cytokine secreted by various cells including macrophages, fibroblasts, endothelial cells, and tubular epithelial cells. It has numerous functions, acting as a pyrogen, stimulating acute phase proteins, activating lymphocytes, playing a role in haemopoieses and increasing immunoglobulin A secretion. IL-8 is produced by macrophages and cortical-epithelial cells when induced by bacterial LPS, IL-1 and tumor necrosis factor alpha (TNF-alpha). IL-8 is a potent chemo- attractant and activator of neutrophils [2]. TLRs act by distinguishing bacterial components and inducing inflammatory response [10].

The aim of this study was to evaluate the association between ABO blood groups and UTI, in addition to investigates the levels ofIL-6,IL-8 and HS-CRP in non-pregnant women with UTI caused by *E. coli.*

## Materials and methods

### Sample collection

Clean – catch midstream urine and venous blood samples were collected from 400 non-pregnant women admitted in Azadi Teaching Hospital and General Kirkuk

Hospital during the period from December 2015 to May 2016, with age ranged between 14 to 60 years, in addition to a fifteen healthy women are also included in the current studies as control group. Informed consent was taken from each women before their enrolment in the study. Urine samples were divided in two parts: first part used for urine culture and the remainder was centrifuged at 3000 rpm for 10 minute and stored at -20 C for cytokines analysis. Venous blood samples were also collected to determine the ABO blood group by hemagglutination methods using BIOTIC-Germany kit, the remaining clotted blood was centrifuged at 3000 rpm for 10 minutes and stored until analysis for serum levels of cytokines and HS-CRP.

**Culturing of Urine Samples**

Blood and MacConkey agar media were used for culturing urine samples by a direct streaking method, using a calibrated bacteriological loop measuring 0.001ml of urine. The plates were incubated overnight aerobically at $37^0$C, and growth examined after incubation for 24 hours. Isolated bacteria colonies on Blood and MacConkey agar plates were identified depending on their diameters, shapes, odor, and other features [11].The biochemical tests were performed for confirmation of bacterial species[12,13]. Additionally, an Api20 system for Enterobacteriaceae (bioMeriex/France) was used for accurate identification of isolates bacteria depending on the procedure by the manufacturing company.

**Cytokines and HS. CRP Assay**

Sera and urine of patients and control were assessed on the level of 1L–6, 1L-8 by (ELISA) method using commercially available kits (KOMA,Korea) and serum level of HS–CRP used (LABOR DIAGNOSTIKA NORD, German).

**Statistical Analysis**

Cytokines and serum levels of HS-CRP data were statistically analyzed using the computer program SPSS (version 13). A t-test was used to measure the differences in means between two groups. Frequencies of UTI and blood groups were presented as a percentage, and significant difference between their distribution in UTI patients and controls were assessed by Fisher exact probability (P). Association between a blood group and disease, relative risk(RP), etiological fraction (EF) and preventive fraction (PF) were also estimated. Then WINPEPI program was used to detect study [14].

**Results and discussion**

From 400 urine sample, 124 (31%) were positive (significant bacteriurea) for bacterial growth and 276 (69%) had no growth. This result was in agreement with a local study conducted in Baghdad, Iraq [15] which found the incidence of UTI in non-pregnant women to be 32%. Another study performed in South Africa [16] showed that prevalence of UTI in non-pregnant women was 42.8%. On the other hand, this discrepancy may be due to samples size, hygiene status or level of education.

Negative urine culture results in this study might be due to other pathogenic agents which include viruses, fungi, gram-positive bacteria and other bacteria that cannot be isolated from routine culture methods, or due to the use of antibiotics prior to urine culture.

Api-20E and biochemical tests of isolated bacteria showed that *E. coli* was the most predominant bacteria among bacterial isolates found in 97 (78.23%) samples (Figure.1). This result agrees with a study from Bangladesh [17] which found the dominant bacterial isolate to be *E. coli* (present in 85%), and in Uganda reached 57.5, 50 and 41.9%, respectively [18-20]. This might be due to *E. coli*, which can colonize the pre-urethral region, and invasive the urinary tract and cause symptomatic disease

[21] or due to producing a hemolysin toxin by *E. coli* that lyses lymphocytes, and inhibits phagocytosis and chemotaxis [22].

Other isolates were included *Klebsiella pneumonia* isolated in 13 samples (10.48%). This result agrees with that of the study from Bangladesh [17] which found the frequency of *Klebsiella pneumonia* was 13.5% and in Uganda11.6% [18]. This bacteria present as normal flora in the intestine, but can colonize the pre-urethral area, enter the urinary system, resist phagocytosis by employing their mucoid macro capsules, as well as adhering to epithelial cells through use of their fimbriae [23,24].

*Proteus species* were isolated from the urine of 8 patients (6.45%). This result was in agreement with a study from Baghdad city, Iraq [25], which found the incidence of *Proteus species* was 5%. *Proteus species* has important virulence factors, including fimbriae (to aid in adherence) and urease enzymes which convert urea to NH3 and CO2. The presence of high amounts of ammonia in the urine, especially in the bladder, leads to urinal phosphate precipitation, resulting in phosphate calculus formation[26].In addition, *Proteus species* have rapid motility secondary to their peritrichous flagella [27].

*Pseudomonas aeruginosa* was isolated from 6 samples (4.84%). This result was agreed with the study from Baghdad city, Iraq [15], who found the frequency of this bacteria to be 8.75%. Infection by this bacteria may be due to low hygienic status in particular groups of individuals, specifically immunocompromised patients. Additionally, virulence factors including proteases, can damage fibrin and immunoglobulin's, and disrupt epithelial tight junctions [28].

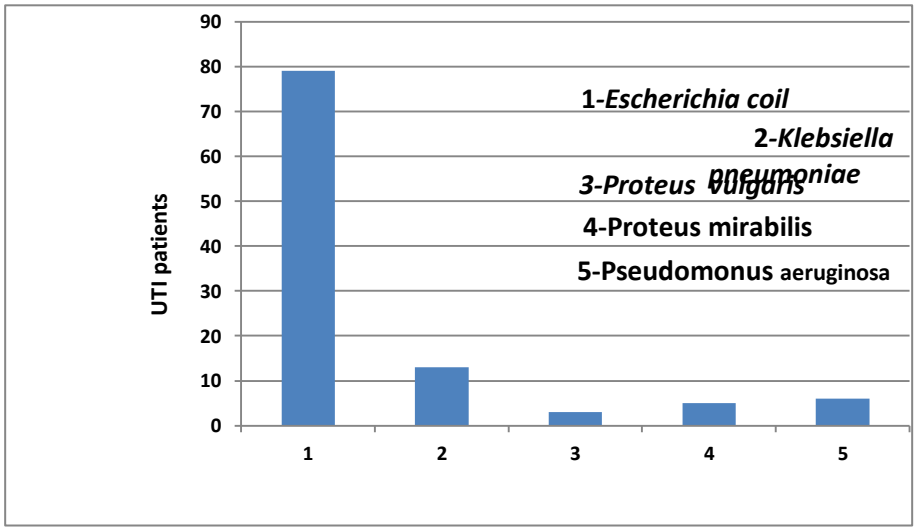


**Fig.1. Number of isolated gram negative bacilli**

**ABO Blood Groups in women patients and Control**

Numerous studies have investigated the association between ABO blood groups and bacterial infection including UTI [29,30], gastric ulcer caused by (*H. pylori*)[31]. As shown in Table 1, the results revealed that 53.6% of patients with *E.coli* related UTI have blood group A and 32.39% in the control group. This data

was associated with RR of 2.41, which contributed etiologically by approximately 30% (EF = 0.31) to the development of disease, and our finding was agreed with previous report [30], which confirmed a positive association between A blood group and UTI. However, Safarkar *et al.,*[32] conclude that A and O blood group had a higher prevalence rate in UTI patients and agreed with Yass *et al.,* [33], who found that A and O blood groups were in high prevalence in female patients with UTI. In contrast low numbers of B blood group was seen in patients compared with control (8.25% vs 25.3%), this association may lead to a protective effect (PF=18%) to UTI development. Additionally, blood group A had a high proportion of UTI caused by *Klebsiella pneumonia*, P*roteus species* and *P. aeruginosa*, however none of these blood groups showed a significant difference between patients and controls. Further studies are required to understand the role of ABO blood groups in UTI.

**Table 1. Frequency of ABO blood groups in women with UTI and controls.**

| | Blood Group | Patients (No.=124) | | Controls (No.=423) | | RR | EF | PF | Fisher exact probability | 95% C.I. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | No | % | No | % | | | | | |
| Infection with *E.coli vs.* control No.=97 | A | 52 | 53.6 | 137 | 32.4 | 2.41 | 0.314 | | $1.6 \times 10^{-4}$ | 1.54-3.77 |
| | B | 8 | 8.2 | 107 | 25.3 | 0.27 | | 0.18 | $1.2 \times 10^{-4}$ | 0.13-0.56 |
| | AB | 5 | 5.2 | 50 | 11.8 | 0.41 | | 0.07 | 0.066 | 0.16-1.04 |
| | O | 32 | 33.0 | 129 | 30.5 | 1.12 | 0.036 | | 0.628 | 0.70-1.79 |
| Infection with *K. Pneumoniae vs.* control No.=13 | A | 6 | 46.2 | 137 | 32.4 | 1.79 | 0.20 | | 0.369 | 0.62-5.20 |
| | B | 2 | 15.4 | 107 | 25.3 | 0.54 | | 0.11 | 0.532 | 0.12-2.32 |
| | AB | 2 | 15.4 | 50 | 11.8 | 1.36 | 0.04 | | 0.660 | 0.31-5.94 |
| | O | 3 | 23.1 | 129 | 30.5 | 0.68 | | 0.09 | 0.762 | 0.19-2.40 |
| Infection with *Proteus species vs.* control No.=8 | A | 3 | 37.5 | 137 | 32.4 | 1.25 | 0.07 | | 0.718 | 0.32-4.85 |
| | B | 2 | 25.0 | 107 | 25.3 | 0.98 | | 0.00 | 1.000 | 0.22-4.47 |
| | AB | 1 | 12.5 | 50 | 11.8 | 1.07 | 0.008 | | 1.000 | 0.15-7.73 |
| | O | 2 | 25.0 | 129 | 30.5 | 0.76 | | 0.07 | 1.000 | 0.17-3.44 |
| Infection with *P. aeurginosa vs.* control No.=6 | A | 4 | 66.6 | 137 | 32.4 | 4.18 | 0.50 | | 0.094 | 0.87-19.92 |
| | AB | 1 | 16.7 | 50 | 11.8 | 1.49 | 0.055 | | 0.534 | 0.21-10.83 |
| | O | 1 | 16.7 | 129 | 30.5 | 0.46 | | 0.16 | 0.673 | 0.06-3.27 |

**RR= Relative Risk, EF= Etiological Fraction, PF=Preventive Fraction, P=p-value, C.I=Confidence Intervals**

In order to determine the relationship between blood groups and UTI, as shown in Table 2, 52.39% of infected samples belonged to A blood group, which contributed 29% as etiology for UTI development. In contrast, the B blood group was with lower frequency (9.67%) in UTI patients compared to controls with a PF value of 17%, which suggest that having B blood group may be a protective factor against the development of UTI.

Bacterial attachment to human epithelial tissues is consider the most important step for infectious to occur [29] may be associated with susceptibility of some blood groups to UTI. Blood group antigens are a group of carbohydrates determinants located on erythrocytes, lymphocytes, phagocytes, certain epithelial cells involving urothelium [6] and Carbohydrates residues situated on the surface of glycoproteins and glycolipids of the uroepithelial cells act as sites of attachment for microbial lectin [34,35]. The specific receptor for type P fimbriae of some strains of *E coli* is the Gal 1- 4 Gal oligosaccharide fractions on both erythrocytes and uroepithelial cells [36]. Chemical structure of ABO blood group can act receptors for

binding with microbial lectin [37]. This suggests relationship between blood phenotying and prevalence of UTI in non-pregnant women.

**Table 2. Observed numbers and percentage frequencies of ABO blood group in infected and non infected women with UTI**

| Blood group | Samples | | | | RR | EF or PF | P | 95% C.I |
|---|---|---|---|---|---|---|---|---|
| | Infected No. = 124 No | % | Not infected No. = 423 No | % | | | | |
| A | 65 | 52.4 | 137 | 32.39 | 2.30 | 0.29 | $8.1x10^{-5}$ | 1.53-3.45 |
| B | 12 | 9.67 | 107 | 25.30 | 0.32 | 0.17 | $1.1x10^{-4}$ | 0.17-0.60 |
| AB | 9 | 7.2 | 50 | 11.82 | 0.58 | 0.049 | 0.188 | 0.28-1.22 |
| O | 38 | 30.6 | 129 | 30.50 | 1.01 | 0.002 | 1.000 | 0.65-1.55 |

**Determination cytokines level and HS-CRP in women with UTI caused by *E. coli***

Table 3 shows that the mean concentration of IL-6 and IL-8 in serum and urine of non-pregnant women with acute UTI caused by *E coli* were highly significant as compared with controls ($P < 0.0001$). This result was in agreement with other [2] who reported a variety of cytokines and chemokines which caused an immune response and inflammation in response to *E.coli* UTI infection (IL-6,IL-8, IL-10,IL-17A,CXCI8 and CCL2). These results were also similar to a study from the Netherlands [38] which found that levels of IL-6 and IL-8 in urine were elevated in patients with UTI. Additionally, Jacobson *et al.,* study [39] from Sweden reported high significant concentrations of IL-6 and IL-8 in serum and urine in patients with acute pyelonephritis. Elevated levels of IL-6 and IL-8 may reflect the increase of TLR4 expression which results in elevated local cytokines. Activation of TLR4 by microbial virulence of UPEC, result in increased transcription of IL-6 and IL-8 via two pathways; by activation of Neuclear factor kappa binding (NF-κβ) or by an increase in intracellular $Ca^{+2}$ [40]. Elevated IL-8 in urine of patients with UTI, may return to normal production by transitional cells (in cystitis), mesangial cells and renal cortical epithelial cells (in cases of kidney infection). This occurs via stimulation of the endotoxins and fimbrieae present in *E. coli* and other gram-negative bacteria.[41-43].

IL-8 acts as a chemo attractant and activator of neutrophils by interacting with the CXCR1 and CXCR2 receptors on the surface of uroepithelial cells and neutrophils, and encourages transepithelial infiltration and chemotaxis of these cells to infected sites [44,45]. Increased urinary IL-6 in patients with UTI may return to normal production by epithelial cells of the bladder and kidney and renal tubular epithelial cells especially after infection by gram-negative bacilli. However, elevated concentrations of IL-6 in serum can contribute to a rise in body temperature and stimulation of production of CRP as a marker of disease in the acute phase, in systemic response [46] and activation of B-cells to produce immunoglobulin A (IgA)[44]. Table 3 shows the mean level of HS-CRP in non-pregnant women with UTI caused by *E coli,* which was highly significant compared with women without UTI ($P <0.001$).

**Table 3.The median values of the parameters in UTI women and the control group.**

| Parameters | Sample | Patients(n=36) | Controls(n=15) | P value < | 95%Confidence Interval |
|---|---|---|---|---|---|
| | | Mean ± SE | | | |
| IL-6 (pg/ml) | Serum | 135.79± 6.03 | 85.9± 8.21 | 0.0001 | 25.66-74.13 |
| | Urine | 176.88±10.12 | 0 | 0.0001 | 156.29-197.47 |
| IL-8 (pg/ml) | Serum | 238± 12.80 | 116.1±10.63 | 0.0001 | 88.83-165.44 |
| | Urine | 463.15±29.08 | 0 | 0.0001 | 403.99-522.31 |
| CRP (mg/l) | Serum | 79.08±42.28 | 8.50±6.05 | 0.001 | 7.84-69.08 |

This result was in agreement with a study from India [47], which found that levels of CRP in serum were elevated in patients with UTI and was higher in upper UTI than in lower UTI. CRP produced by hepatocytes in response to an increase in certain intercellular signaling polypeptides called cytokines, involve interleukin-1β (IL-1β), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α). It binds to phosphocholine, a general component of microbial membranes, and also binds to peptidoglycan, phospholipids and other constituents of bacteria, fungi, and parasites. As the levels rise and then decrease so rapidly, CRP is the most broadly used indicator of acute inflammation. CRP is used to calculate the systemic inflammatory response but there is no usual value for quantifying local host response marker in urine [48]. This study investigated the relationship between IL-6, IL-8 and CRP in serum and urine of patients with UTI caused by *E. coli*. The result showed a positive correlation between high concentration of IL-6 and HS-CRP (r=0.86) in women with UTI (Figure.2). In an acute phase reaction, IL-6 induces the production of acute phase proteins such as CRP in hepatocytes and releases it to the peripheral circulation leading to increasing serum levels [49]. In contrast, there was no significant correlation when serum and urine IL-8 and IL-6 were compared in infected patients.

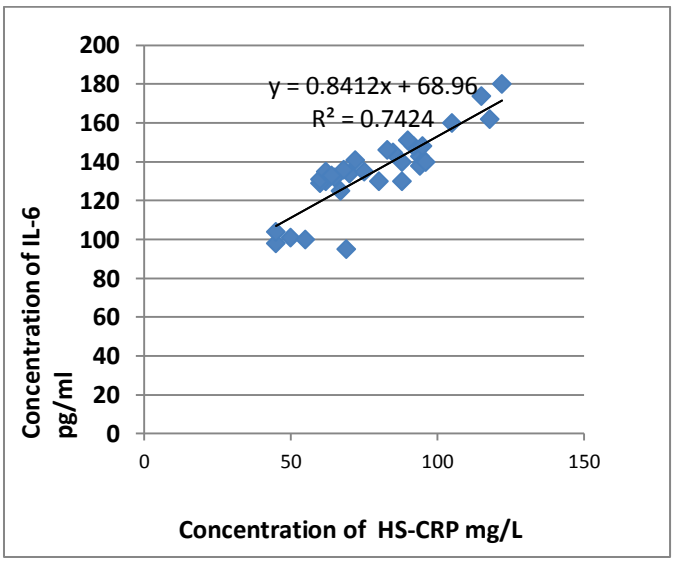


**Figure 2.Correlation between the serum concentration of IL-6 and HS-CRP in non pregnant women with UTI caused by *E. coli* during the acute phase.**

From this study, we concluded that local and systemic IL-6 and IL-8 were detected in serum and urine in UTI caused by *E. coli*. Positive correlation between

high concentration of IL-6 and HS-CRP, which indicated that IL-6 with HS-CRP levels in serum might be a good marker for severe UTI caused by *E. coli.*

**ETHICAL APPROVAL: Kirkuk University College of Science (KUCOS) Ethical Committee**
**CONSENT TO PARTICIPATE: Informed consent was taken from each subject before their enrolment in the study.**
**HUMAN AND ANIMAL RIGHTS: The study conducted in adherence with Helsinki Ethical standards.**
**CONSENT FOR PUBLICATION: Authors transfer the copyright to the International Journal of Medical Sciences.**
**FUNDING: No funding**
**CONFLICT OF INTEREST: The authors declare no conflict of interest, financial or otherwise.**
**DATA AVAILABILITY: The data was available in Kirkuk University College of Science (KUCOS), Kirkuk, Iraq, information set and available on request.**

**References**

1. Kabbara WK, Meski MM, Ramadan WH, Maaliki DS, Salameh P. Adherence to International Guidelines for the Treatment of Uncomplicated Urinary Tract Infections in Lebanon. Cana J Infec Dis and Med Micro 2018; 2018:7404095..
2.Reygaer WC. Innate Immune Response to Urinary Tract Infections Involving *Escherichia coli* J Clin Cell Immunol2014;5 (6): 1-5.
3. Odongo I, Ssemambo R, KunguJM. Prevalence of Escherichia Coli and its Antimicrobial Susceptibility Profiles among Patients with UTI at Mulago Hospital, Kampala, Uganda. Interdisciplinary Perspectives on Infectious Disease 2020; 2020:8042540.
4. Stamm WE, Schaeffer AJ. The state of the Art in the management of Urinary Tract Infections. Am J Med, 2002;133.
5. Usama A, Abdulfattah A, Ibrahim S. Relationship between ABO blood group and end-stage renal disease in Latakia, Syria2017. Saudi Journal of Kidney Diseases and Transplantation2018; 28(2): 445-445.
6. Sakallioglu O, Sakallioglu AE. The effect of ABO ─ Rh blood group determinants on urinary tract infections. Int Uro Neophrol, 2007;39: 577-579.
7. Cooling L. Blood groups in infection and Host susceptibility. Clin Microbiol Rev 2015; 28(3): 801-70.
8.Liu QP, Sulzenbacher G, Yuan H, Bennett EP, Pietz G, Saunders K, et al. Bacterial glycosidases for the production of universal red blood cells Nat Biotechnol 2007; 25 (4): 454-464.
9.Hayes BW, Abraham S. Innate immune response to bladder infection. Microbiol Spectr J, 2016;4(6).
10. Molteni M, Gemma S, Rossetti C. The Role of Toll-Like Receptor 4 in Infectious and Noninfectious Inflammation. Mediat Inflamm J. 2016;.
11.Collee JG, Fraser AG, Marmion BP, Simomons A. Mackie and McCarthy. Practical Medical Microbiology".14th ed. Churchill-Livingstone Inc. USA, 1996; 113-129.
12. McFaddin JF. Biochemical Tests for Identification of Medical Bacteria. 1st Ed. The Williams and Wilkins. Baltimore USA2000.

13. Tille PM. Bailey and Scott's Diagnostic Microbiology13thed.Elsever2014;193-229.
14. Ad'hiah AH. Immunogenetic studies in selected human diseases. Ph D Thesis University of Newcastle upon Tyne England 1990.
15.Kareem IJ, Rasheed IY. Antibiotic Susceptibility of Gram Negative Aerobic Bacteria Isolated from Urinary Tract Infection in Community. Iraqi JM Sci 2011; 9(4): 295- 300.
16. Lewis DA, Gumede LY, Van der hoven LA, De Gita GN, DeKok Ed, De Large T, etal. Antimicrobial susceptibility of organisms causing community-acquired urinary tract infections in Gauten Province, South Africa, SAMJ, 2013; 103(6): 377-381.
17. Chowndhury S, Parial R. Antibiotic Susceptibility of Bacteria among Urinary Tract Infection Patients in Chittagong, Bangladesh. SMU Med J 2015;2 (1):114-125.
18. MwakaA D, Mayanja-Kizza H, Kigonya E, Kaddu-Mulindwa D. Bacteriuria among adult non-pregnant women attending Mulago hospital assessment centre in Uganda. African Health Sciences, 2011;11(2): 182–189.
19. Kabugo D, Kizito S, Ashok DD, Graham KA, Nabimba R, Namunana S, Kabaka MR, Achan B, Najjuka FC. Factors associated with community-acquired urinary tract infections among adults attending assessment centre, Mulago Hospital Uganda. Afr Health Sci. 2016 Dec;16(4):1131-1142
20. Odoki M, Aliero AA, TibyangyeJ, Wampande E, Kato CD, Agwu E, et al. Revalence of bacterial urinary tract infections and associated factors among patients attending hospitals in Bushenyi district, Uganda International Journal of Microbiology2019: 8 .
21. Vagarali MA, Karadesai SG, Patil CG, Metgud SC, Mutnal MB. Haemagglutination and siderophore production as the urovirulence markers of uropathogenic *Escherichia coli*. Indian J Med Microbiaol, 2008;2: 68-70.
22. Raksha H, Srinivasa H, Macaden RS. Occurrence and characterization of uropathogenic *Escherichia coli* in urinary tract infection. Indian J Med Microbiol, 2003;21(2): 102-107.
23. Donta ST, Peduzz P, Cross AS, Saddof J. Immunoprophylaxis against *Klebsiella* and *Pseudomonasaer uginosa* infection. J Infect Dis, 1996; 174 (3): 537-543.
24. Richard MH. Microbiology and Immunology. 4th ed. Heidellberg press, New York. 1995; 432- 531.
25. Mahmood MA. Prevalence and Antimicrobial Susceptibility of Pathogen in Urinary Tract Infections . Al-Nahrain University J Sci, 2011;14 (4):146-152.
26. Sujoy B, Aparna A. Enzymology, Immobilization and Application of Urease Enzyme. International Research Journal of Biology Science.2013;2(6): 51-56.
27. Tuson HH, Copeland MF, Carey S, Sacotte R, Weibel DB. Flagellum Density Regulates *Proteus mirabilis* swarmed Cell Motility in Viscous Environments, Journal of bacteriology.2013;195(2): 368-77.
28.Kipnis E, Sawa T, Wiener-Kronish J. Targeting mechanism of *Pseudomonas aeruginosa* pathogenesis. Med Mol Infect, 2006;36: 78-91.
29. Mandato VD, Torricelli F, Mastrofilppo V, Ciarlini G, Pirillo D, Farnetti E, et al. Prognostic Impact of ABO Blood Group on Type I Endometrial Cancer Patients-Result from our own and other Studies. J Cancer 2017; 8(14):2828-2835.

30. Rocha D, Silva A, Pereira A, Borges A, Rodrigues E, Mendonca V, Osorio N, et al. Urinary tract infections and blood groups: Do they relate?. International Invention Journal of Medicine and Medical Science. 2015; 2(5):66-72.
31. Abbas S Kh, Hamdi B, ALbarzanji ZNM. Increased Serum Interleukin-10 Could lead to Allergic Disease Development in Helicobacter pylori Infected Adult. Energy Procedia 2019; 157:193-198
32. Safarkar R, Bonabi R, Massiha A, Dejagah S, Khani S, Abafat N, Pahlaviani MMK. Relationship between Urinary Tract Bacterial Infections and Blood Groups in Patients Referring to Alavi Hospital in Ardabil, Iran in 1395. Scientific Journal of Nursing, Midwifery and Paramedical Faculty, 2018; 4(1):41-51.
33. Yass MA, Ali SS, Ahmed AA. ABO-Rh blood groups and type of Food amongst urinary tract infection causative. Znco J Med Sci, 2012;16 (1): 71-77.
34. Blackwaell. The role of ABO blood groups and secretor status in host defenses. FEMS. Microbial Immunol1989;47: 341-350.
35. Weir DM. Carbohydrates as a recognition molecules in infection and immunity FEMS Microbial. Immunol1989; 47:331-340.
36. Rehman RE, Kliegman RM, Jenson HB. Nelson Textbook of pediatrics.16th ed. Elsevier, USA.2000; 1623.
37. Wu AM, Wu JH, Singh T, Liu JH, Tsai MS, Gilboa-Garber N. Interactions of the fucose-specific *Pseudomonas aeruginosa* Lectin, PA-IIIL, with mammalian glycoconjugates bearing polyvalent Lewis(a) and ABH blood group glycotopes. Biochimie.2006;88:1479-92.
38. Willize EVS, Cees VN, Uginia T, Marleen SMZ, Jan PRK, Tanny JKV, Jaap TV, Esther V.D. Treatment duration and prognostics in febrile urinary tract infection. PLOs One, 2015;10(3): 112-133.
39. Jacobson SH, Hylander B, Wretlind B, Brauner A. Interleukin-6 and Interleukin-8 in Serum and Urine Patients with Acute Pyelonephritis in relation to Bacterial-virulence- Associated Traits and Renal Function. Nephron1994; 67(2): 172-179.
40. Song J, Duncan ML, Li G, Chan C, Grady R, Stapleton A, Abraham SNA. Novel TLR4-mediated signaling pathway leading to IL-6 responses in human bladder epithelial cellsPloSPathog2007;3(4): 541-52.
41. Brown Z, Strieter RM, Chensue SW, Ceska M, Lindley I, Nilid GH, Kunkel AL, Westwick J. Cytokine activated human mesngail cells generate the neutrophil chemoattractant, interleukin 8. Kidney Int1991; 40(1):86-90.
42. Kunser D J, Luebbers EL, Nowinski RJ, Konieczkowski M, King CH, Sedor JR. Cytokinee and LPS-induced synthesis of interleukin-8 from human mesngail cells. Kidney Int, 1991; 39:1240-1248.
43. Schmouder R L, Strieter RMC, Wiggings RG, Chensue SW, Kunkel SL. In vitro and in vivo interleukin-8 production in human renal cortical epithelia. Kidney Int 1991;41:191-198.
44. Ragnrsdottir B, Svanborg C. Susceptibility to acute pyelonephritis or asymptomatic bacteriuria: host-pathogen interaction in urinary tract infections. PediatrNephrol2012;27(11): 2017-29.
45. Cheng CH, Lee YK, Tsau YK, Lin TY. Genetic polymorphisms and susceptibility to parenchymal renal infection among pediatric patients. Pediat Infect Dis J2011;30: 309-314.
46. Gabay C. Interleukin-6 and chronic inflammation. Arth Res Ther2006;8(2):1-6.
47. Agrawal P, Pandey A, Sompura S, Pursnani ML. Role of Blood C-Reactive Protein Levels in Upper Urinary Tract Infection and Lower Urinary Tract Infection

in Adult Patients (> 16 years).Journal of the Association of Physicians of India.2013; 61: 26-27.

48. Otto G, Burdick M, Strieter R, Godaly G. Chemokine response to febrile urinary tract infection. Kidney Int2005; 68: 62-70.
49. Azab S, Zakaria M, Raafat M, Seief S. The combination of urinary IL - 6 and renal biometry as useful diagnostic tools to differentiate acute pyelonephritis from lower urinary tract infection**.** Int Braz J Urol, 2016 **;**42(4): 810–816.