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Review article

The Role of Immune Response in Bacterial Urinary Tract Infections in Iraq: A Review

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Abstract
Urinary tract infections (UTIs) are among the most prevalent bacterial infections, frequently caused by uropathogenic Escherichia coli (UPEC), the primary causative agent in humans. The immune system plays a critical role in defending against bacterial UTIs by preventing pathogen attachment and colonization in the urinary epithelium, while also mitigating infection severity through innate and adaptive immune responses. This review examines previous studies conducted in Iraq on the immunological aspects of bacterial UTIs. It explores the mechanisms of antibiotic resistance in UTI-causing bacteria and synthesizes findings from 42 studies that investigate the role of innate and adaptive immunity in UTI-related inflammation. Iraqi research has consistently demonstrated a high prevalence of multidrug-resistant (MDR) UPEC strains, underscoring the urgent need for immune-based interventions, such as vaccines and immunomodulatory therapies, to enhance host defenses and reduce dependence on antibiotics. Key findings highlight the involvement of neutrophils, cytokines, and antimicrobial peptides in immune defense against UPEC, while bacterial immune evasion strategies contribute to recurrent infections. This review also emphasizes the necessity for personalized medicine approaches that integrate genetic and immunological insights to improve UTI prediction, prevention, and treatment in the Iraqi population. Numerous Iraqi studies have reinforced the crucial role of innate immunity in determining UTI susceptibility. A deeper understanding of immune mechanisms in UTI pathogenesis is essential for developing more effective treatment strategies and alleviating the burden of recurrent infections.
Keywords: *Urinary Tract Infections, Bacterial UTIs, Antibiotic Resistance, Immune System, Iraq.*

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Introduction

The invasion of the urinary tract by pathogenic bacteria, mainly from the gastrointestinal tract, can cause bacterial urinary tract infections (UTIs), with *Escherichia coli (E. coli)*, which accounts for 70–90% of uncomplicated UTIs [1]. *E. coli, Klebsiella pneumoniae, Proteus mirabilis, Enterococcus faecalis, and Staphylococcus saprophyticus* are the public causative pathogens [2]. Two types of Bacterial UTIs include lower UTIs, which include cystitis (bladder infection) and urethritis (infection of the urethra). Symptoms include dysuria, urgency, frequency, and suprapubic pain. Upper UTIs: Includes pyelonephritis (kidney infection), characterized by flank pain, fever, and systemic symptoms [3]. The primary mechanism of Bacterial UTIs includes bacterial adhesion to the uroepithelium. Uropathogenic E. coli (UPEC) express adhesins like type 1 and P fimbriae, which bind to urothelial receptors and this triggers immune responses and bacterial invasion [4].

Bacterial UTIs mainly result from the colonization and invasion of the urinary tract by UPEC. The pathogenesis of UPEC involves Adherence to urothelial cells by type 1 fimbriae. The FimH adhesin at the tip of these fimbriae binds to mannose residues on the host cell surface, facilitating initial colonization [5]. Following adherence, UPEC invades the umbrella cells, where it can evade host immune responses. Inside these cells, UPEC forms biofilm-like intracellular bacterial communities (IBCs), allowing for clonal expansion and protection from host defenses [5]. Some UPEC can transition into a quiescent state within deeper layers of the bladder epithelium, forming intracellular reservoirs. These reservoirs can persist undetected by the immune system and may serve as a source for recurrent infections [6].

UTIs affect millions worldwide, with a higher incidence in women due to anatomical predispositions. Factors such as sexual activity, certain contraceptive methods, and postmenopausal changes increase susceptibility. In men, UTIs are less common but can occur secondary to urological abnormalities or procedures. The global burden of UTIs is substantial, contributing to significant healthcare costs and morbidity [7].

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Antibiotic resistance in UTIs

Bacterial UTIs can be resistant to antibiotics, and this is acquired by developing mechanisms that allow them to survive in the presence of antibiotics, as shown in Figure 1 and Table 1.

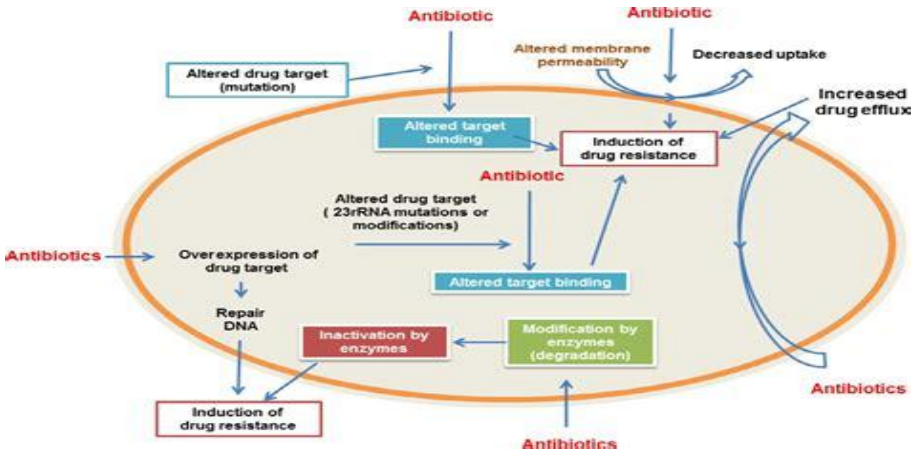

Figure 1: Mechanism of Antibiotic resistance in bacterial UTIs [8]

Table 1: Mechanism of Antibiotic resistance in bacterial-caused UTIs

Mechanism	Description	Examples	Clinical Relevance	Reference
Enzymatic Degradation	Bacteria produce enzymes that degrade or inactivate antibiotics.	Beta-lactamases (e.g., ESBLs) break down penicillins and cephalosporins.	ESBL-producing E. coli and Klebsiella pneumoniae cause resistant UTIs. 1	[9]
Efflux Pumps	Active expulsion of antibiotics from bacterial cells reduces intracellular drug concentration.	AcrAB-TolC efflux system (e.g., in E. coli) for fluoroquinolones.	Major resistance mechanism for fluoroquinolones and other drugs in multidrug-resistant (MDR) uropathogens.	[10]
Target Site Modifications	Mutations or alterations in bacterial target sites reduce antibiotic binding and effectiveness.	DNA gyrase/topoisomerase IV mutations for fluoroquinolone resistance.	Common in quinolone-resistant E. coli and beta-lactam-resistant Proteus mirabilis.	[11]
Reduced Membrane Permeability	Decreased expression or loss of porin proteins in the bacterial outer membrane, reducing antibiotic entry.	OmpF loss in E. coli reduces uptake of beta-lactams and aminoglycosides.	Observed in Klebsiella pneumoniae and Pseudomonas aeruginosa UTIs.	[12]
Acquisition of Resistance Genes	Resistance by horizontal transfer of genes can be transmitted through plasmids, transposons, or bacteriophages.	Sulfonamide resistance via sul genes.	Widespread among uropathogens due to plasmid-mediated resistance.	[13]
Biofilm Formation	Bacteria form structured communities (biofilms) on surfaces like urinary catheters, protecting antibiotics and immune responses.	Proteus mirabilis and E. coli biofilms in catheter-associated UTIs (CAUTIs).	Biofilms significantly increase antibiotic tolerance, complicating treatment of CAUTIs.	[14]

Immune Response in UTIs

The host's innate immune response is the first barrier of defense. It involves the activation of Toll-like receptors (TLRs), which recognize bacterial components. Cytokines like interleukin-8 (IL-8) recruit neutrophils to the infection site [15].

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The immune system employs a multifaceted approach to defend against bacterial urinary tract infections (UTIs), as shown in Table 2.

Table 2. Defense Mechanism against Bacterial UTI

Defense Mechanism	Description	Reference
Mechanical Barriers	The urinary epithelium contains epithelial cells with various immune cells as a defense line against infections.	[16]
Innate Immune Response	Versatile innate immune defenses play a crucial role in protecting the urinary lining from the invading uropathogens.	[17]
Pattern Recognition Receptors (PRRs)	Innate immune response can be induced or mediated by certain pattern recognition receptors, neutrophils, and antimicrobial peptides.	[18]
Neutrophil Recruitment	Neutrophils are immunologically considered as primary cells that eliminate pathogens and bacteria as soon as the inflammatory response is triggers. They are recruited to infection as phagocytose and kill pathogenic bacteria.	[19]
Adaptive Immune Response	The adaptive immune system generates specific responses against pathogens, including the production of antibodies and the activation of T cells, providing long-term immunity and memory against recurrent infections.	[20]
Antimicrobial Peptides	Epithelial cells produce antimicrobial peptides that have bactericidal properties, directly targeting and neutralizing pathogens to prevent their colonization and invasion of the urinary tract.	[21]
Cytokine Production	As a response to UTI, the immune system releases cellular cytokines that modulate the immune response, enhancing the recruitment and activation of additional immune cells to effectively combat the invading bacteria.	[22]
Complement System Activation	The complement system is activated during UTIs, leading to opsonization of bacteria, recruitment of inflammatory cells, and direct lysis of pathogens, thereby enhancing the overall immune response against the infection.	[23]

Role of the immune system in Bacterial UTIs

A recent study titled "A Dynamic Interplay of Innate Immune Responses During Urinary Tract Infection" delves into the critical role of innate immunity in defending against uropathogenic bacteria. The research highlights the role of innate immune cells, such as neutrophils and macrophages, in various mechanisms, including recognizing and eliminating the invading pathogens in the urinary tract. The study emphasizes the importance of understanding this dynamic interplay to develop effective therapeutic strategies against urinary tract infections (UTIs) [24]. Additionally, advancements in vaccine development are being explored to enhance immune defenses against UTIs. For instance, Australian researchers are developing the world's first mRNA vaccine targeting the bacteria responsible for up to 90% of UTIs. This vaccine aims to prepare the immune system to recognize and combat UPEC, potentially reducing the incidence of these infections and addressing the growing issue of antibiotic resistance [25]. Many previous studies in Iraq are also shown in Table 3.

Table 3: Previous studies on the role of the immune system in Bacterial UTIs

Parameters	Location	Population	Key findings	Year	Reference
Meta-analysis (2000–2023)	Various cities in Iraq	Pregnant women	High prevalence of UTIs, particularly among women aged 16–35. Escherichia coli is identified as the most common pathogen.	2009	[26]
IL-6	Baghdad	132 bladder cancer and UTI patients	Patients with bladder cancer can be affected by bacterial UTIs strains, display a significant	2010	[27]

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			increase in serum IL-6 levels (by ELISA technique)		
IgG, IgM, and IgA	Al-Diwanyia	392 UTI	Study findings mentioned that the serum levels of IgG, IgA were increase, while IgM levels were decreased in UTI patients	2012	[28]
IL-2, IL-4 and IL-17A	Baghdad	151 autoimmunity patients	IL-2 levels were notably higher in all patients, including those with RA, AS, and SLE, compared to the control group. A similar elevation was observed in UTI-positive cases compared to UTI-negative ones among the total patient group, as well as in AS and SLE, but not in RA or the controls. In contrast, IL-4 exhibited less distinct differences, while IL-17A showed no significant variation.	2013	[29]
IgG, IgM, IgA , C3	Erbil	570 pregnant women	The findings indicate significant variations in the mean ± SD levels of IgG, IgM, and IgA among patients with UTIs. Additionally, the mean ± SD concentration of complement component C3 was significantly higher in the sera of UTI patients compared to women without UTIs (P-value < 0.000).	2017	[30]
IL-17A	Karbala	110UTIpatients	Detected upec in 44.64% of significant bacteriuria cases; identified virulence genes (papc, cnfa, fimh, fyua) in 60–68% of isolates; elevated urinary il-17a levels in upec-infected patients, suggesting its role in immune response.	2019	[31]
IL-23	Baghdad	94 urine samples	There were significant variations in urine IL-23 concentrations among different bacterialUTItypes. The IL-23 levels in urine appear to be influenced by UTIs, particularly in patients with indwelling urinary catheters.	. 2020	[32]
TLR4 Polymorphisms	Babylon	39 pregnant women	Serum IL-17 levels demonstrated a significant difference ($p = 0.016$) between patients and controls. Similarly, serum TNF-α levels showed a significant difference ($p = 0.021$) between the two groups.	2020	[33]
IL-8 and TLR-4	AL-Diwaniyah	800 uti patients	The data indicated a coordinated role of IL-8 and TLR-4 in the response to recurrent urinary tract infections, suggesting their potential as markers for distinguishing between pyelonephritis and cystitis.	2020	[34]
IL-6, IL-8 and TNF-α	Diwaniyah	100 blood and urine samples	The levels of IL-6, IL-8, and TNF-α were significantly elevated in patients. These key cytokines play a crucial role in UTIs, particularly during the acute phase.	2020	[35]
IL-6	Erbil	125UTIpatients	The fact that PCT and IL-6 levels in UTI patients are noticeably greater than in healthy people raises the possibility that these tests could be employed as UTI diagnostic indicators.	2021	[36]
IL-6 ,IL-8	Baghdad	150 Serum and urine samples	Children with UTIs had greater levels of IL-8 in their urine and serum, but there was no discernible change in their levels of IL-6 in their urine and serum.	2021	[37]
IL-6 and IL-18	Baghdad	159 blood and urine specimens	Groups (PCOS with and without UTI) had higher levels of both IL-6 and il-18 than the UTI group.	2022	[38]

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IL-6, IL-8 and hs-crp	Kirkuk	400 urine samples	Serum levels of hs-crp, as well as serum and urine levels of il-6 and il-8, were considerably (p<0.00) higher in women with E. coli-caused UTIs than in controls.	2022	[39]
IL-6	Kirkuk	90 blood samples	Children with positive urine cultures have higher levels of IL-6 than those with negative urine cultures.	2022	[40]
IL-4 gene polymorphisms	Kerbela	135 samples	The distribution of interleukin 4 genotypes ($p < 0.001$) and alleles ($p < 0.001$) varies significantly between study groups in comparison to the control group.	2022	[41]
IL-18, INF-β	Babylon	223 urine and blood samples	While there was no significant change in the mean IL-18 serum concentration in patients (12.01±9.8 pg/ml), the mean IL-18 urine concentration in patients (13.39±3.3 pg/ml) was substantially elevated at a p-value ≤ 0.05.	2022	[42]
TLR-2	Babylon	100 urine samples	When the p-value is less than 0.045, there is a significant difference in the urine TLR-2 concentration between the control group and UTI patients. Toll-like receptor 2 levels were noticeably greater in febrile urinary tract infection patients than in the control group.	2022	[43]
IL-6	Salah al-Din	118 urine samples	Patients with urinary tract infections who did not have diabetes had a significantly higher level of interleukin-6 (0.01>p). Patients with urinary tract infections who do not have diabetes had a significant (P>0.01) rise in IL-6.	2022	[44]
IL-6 and IL-18	Baghdad	159 blood and urine samples	The PCOS with and without UTI groups had greater levels of both IL-6 and IL-18 than the UTI group.	2022	[45]
IL-22	Mosul	140 serum and urine samples	Higher serum IL-22 levels in patients with UTIs due to E. coli compared to the control group.	2022	[46]
IL-22	Al-Najaf	60UTIpatients	When compared to healthy controls, the study found that UTI patients had significantly higher levels of IL-22. IL-22 levels were higher in female patients than in male individuals.	2022	[47]
IL-6,IL-8	Baghdad	200 women	Identified immune-related markers, such as elevated cytokines (IL-6, IL-8), correlating with severe UTI cases. Resistance to quinolones and cephalosporins was high.	2023	[48]
Innate immunity	Baghdad	237 patients	Uti prevalence of 26.58%; higher incidence in females (58.9%); identified risk factors including anatomical differences and potential innate immunity defects contributing to recurrence.	2023	[49]
IL-18	Baghdad	60 rheumatoid arthritis patients	Found a higher prevalence of UTIs among rheumatoid arthritis patients, with elevated levels of interleukin-18, suggesting a link between immune dysregulation and increased susceptibility to UTIs	2023	[50]
IL-8,IL-6	Baghdad	139UTIpatients	IL-8 and IL-6 levels in patients are considered high as they play a role in the inflammatory response.	2023	[51]
IL-35	Not specified	60 children with t1dm	Elevated IL-35 and MIF levels in diabetic children with UTIs suggest a link between cytokine secretion and susceptibility.	2023	[52]

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IL-6, IL-33	AL-Najaf City	102UTIs and UTIs-KF	IL-6 and IL-33 are significantly higher than control group in both UTIs and UTIs-KF patients.	2023	[53]
SDF1, IgA	Babylon	`180 Patients (UTI, BV)	A significant rise in SDF1 and IgA in UTI patients was documented.	2023	[54]
IL-15	Baghdad	587 patients UTIs	Levels of IL-15 are the highest in the sera of UTI patients with K-ESBL strains	2023	[55]
IL-1	Tikrit	200 urines	IL-17 level was significantly high at $P < 0.05$ in UTI patients of both *E. coli* and *S. aureus*, rather than controls.	2023	[56]
IL-23	Baghdad	200 urine and vaginal	The IL-23 mean of UT-infected patients with *E. faecalis* was higher than that of controls ($P<0.05$).	2023	[57]
C3 , C4	Diyala	205 UTI samples	Means of both C3 and C4 were the lowest in UTI patients	2023	[58]
CD17 , TLR-4	Diyala	205 UTI samples	An increase in CD17 and TLR-4 levels was detected in UTI patients, while they were lower in control individuals	2023	[59]
IL-9	Salah el-din	50 urine samples	Many important changes resulting from infection of bacterial UTIs, especially the interleukin-9 levels.	2024	[60]
IL-12, IL-17, TNF-α	AL-Hilla	150 patients UTIs	Observed elevation of both IL-12 and IL-17 documented in *P. aeruginosa*-infected UTI patient compared to TNF-α levels	2024	[61]
TLR-4, TLR-2	Babylon	90 serum specimens, 45 patients with UTIs	The immunological findings showed that both TLR2 and TLR4 were raised, as they may contribute to susceptibility to UTIs	2024	[62]
IL-1 Beta	Karbala	70 UTIs patients	An increase in IL-1 Beta was detected in bacterial UTIs, with a negative correlation in LBP in sera.	2024	[63]
IL-6	Anbar	500 urines	UTI patients had a mean IL-6 was significantly higher than that in controls ($p < 0.0001$)	2024	[64]
IL-8	*Babylon*	120 UTI patients	Findings showed elevated levels of IL-8 in sera of the patients compared with controls	2024	[65]
TLR-4, TLR-2	Babylon	45 patients with UTIs	There were increased TLR2 levels in patients with UT in comparison control mean (by using the ELISA technique)	2024	[66]
IL-6, IL-1 β	Salah al-Din	155 patients UTIs	Regarding the immunological parameters, the serum levels of both IL-6 and IL-1β in patients' sera were higher than in healthy people. Moreover, it was found that some differences in IL-6 values among UTIs-causing bacterial strains were statistically significant at a p-value (less than 0.05).	2025	[67]

In short, the immunological changes in human tissues and sera could be affected by the action of invasive bacteria as they have been developed genetically by both vertical and horizontal gene transfer [68]. Many detection methods were also modified to be for the bacterial mutations and immunogenetic modulations [69,70].

Conclusion

Many Iraqi studies have highlighted the significant and crucial role of innate immunity in determining susceptibility to urinary tract infections (UTIs). Genetic variations in immune response genes may increase the risk of infection by altering immune signaling. Research also confirms UPEC as the dominant bacterial cause of UTIs, with immune responses involving cytokine production and neutrophil activation playing a key role in infection control. However, bacterial immune evasion mechanisms, such as biofilm formation, contribute to

recurrent infections. These findings suggest the need for immune-based interventions, including vaccines and immunomodulatory therapies, to enhance resistance against UTIs. Future research should focus on personalized medicine approaches that consider genetic and immunological factors to predict and prevent recurrent infections.

Conflict of interest

There is no conflict of interest.

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