RESEARCH ARTICLE

Article DOI:10.21474/JNHM01/165
DOI URL: http://dx.doi.org/10.21474/JNHM01/165

PREVALENCE OF THE UREC AND HPMAGENES IN *PROTEUS MIRABILIS* ISOLATED FROM URINARY TRACT INFECTIONS IN IRAQ

Shymaa A.G. Al-hussainy

1. Department of Community Health Techniques, Technical Institute of Babylon, Al-Furat Al-Awsat Technical University(ATU), Babylon, Iraq.

Manuscript Info

Manuscript History
Received: 08 May 2026
Final Accepted:10 June 2026
Published: July 2026

Key words:-
ureC, hpmA, Proteus mirabilis, UTIs, Urease, Hemolysin

Abstract

Background: *Proteus mirabilis* is one of the most important pathogens causing urinary tract infections (UTIs) and harbours multiple virulence factors, including urease and hemolysin produced by the ureC and hpmA genes, respectively. The objective of the present study was to evaluate the distribution of ureC and hpmA genes in a collection of P. mirabilis isolates recovered from patients with UTIs, and to correlate their presence against antimicrobial susceptibility patterns.

Methods: This study has been conducted a cross-sectional research design; from August of 2024 to the beginning of December 2025. A total of 78 P. mirabilis isolates were obtained from urine specimens from patients diagnosed with UTIs supported by clinically and microbi ologically confirmed results. Antimicrobial susceptibility testing was performed against ciprofloxacin, levofloxacin, gentamicin, ceftriaxone, trimethoprim-sulfamethoxazole and amikacin using the Clinical and Laboratory Standards Institute (CLSI) guidelines. Molecular identificati on of ureC and hpmA genes was performed by conventional polymeras e chain reaction (PCR) as described previously.

Results: The highest resistance rates were against trimethoprim-sulfamethoxazole (60.3%) and ceftriaxone (52.6%), followed by ciprofloxacin (43.6%) and levofloxacin (37.2%, while amikacin had the lowest rate of resistance (19.2%). The use of molecular detection showed that the ureC gene was found in 94.9% and hpmA in 78.2% of isolates. Another important association was observed with ureC gene presence and the associated resistances to ciprofloxacin, levofloxacin, ceftriaxone and trimethoprim-sulfamethoxazole ($p < 0.05$). Similarly, the hpmA gene showed significant correlations with resistance to ciprofloxacin ($p < 0.001$), levofloxacin ($p < 0.01$), gentamicin ($p < 0.05$) and also moderate but highly significant association were found for ceftriaxone ($p < 0.001$) and trimethoprim-sulfamethoxazole ($p < 0.001$).

Corresponding Author:-Shymaa A.G. Al-hussainy
Address:-Technical Institute, Al-Furat Al-Awsat University, Iraq.

Conclusion: The high presence of ureC and hpmA genes among urinary *P. mirabilis* isolates confirmsthat these genes may be necessary for bacterial virulence and pathogenicity. The correlation between the carriage of virulence genes and antimicrobial resistance suggests the possibility of an accumulation of both features in strains with high pathogenicity.

Introduction:-

Urinary tract infections (UTIs) are the second most common infectious disease, predominantly impacting women with 150 million individuals affected globally making it increasing public health concerns, particularly with respect to antimicrobial resistance(Sujith et al., 2024). Even though Escherichiacoli is still the most common causative agent, different opportunistic pathogens can be responsible for complicated UTIs out of which Proteus mirabilis stands as one of the major players(Khudiar et al., 2024). P. mirabilis is a Gram-negative, extremely motile and an opportunistic pathogen of family Morganellaceae which is strongly associated with catheter-associated urinary tract infections, recurrent infections, pyelonephritis, and urinary stone disease. This pathogen has the capacity to colonize, survive and inflict significant damage in the urinary tract system, making treatment difficult and high morbidity(Schaffer & Pearson, 2015).

*P. mirabilis*accesses and colonizes the urinary tract using a wide range of virulence factors, including urease and stone formation, fimbriae and other adhesins, iron and zinc acquisition, proteases and toxins,and biofilm formation, together with regulation of pathogenesis. This area has seen considerable advances, yet challenges still remain for tackling complicated UTI and understanding the pathogenesis of UTI caused by P. mirabilis(Hasona et al., 2026; Schaffer & Pearson, 2015). These factors work together as virulence factors for bacterial adherence, colonization, immune evasion and tissue destruction(Chakkour et al., 2024). One of the latest virulence mechanisms in P. mirabilis among many other uropathogens is urease activity and one of the most important characteristics that make it so deadly. Urease hydrolyses urea into ammonia and carbon dioxide, resulting in urine alkalinization and precipitation of magnesium ammonium phosphate [MAP] or calcium phosphate crystals. Thus, urinary stones as well as crystalline biofilms are generated which promotes a state of persistent infection and encrustation of catheters(Dattelbaum et al., 2003).

The urease gene cluster of P. mirabilis contains three structural genes and many associated regulatory elements that constitute an important feature that underlines its virulence capacity. Urease has been known to be a dimeric enzyme composed of 2 putative subunits, and ureC is an essential gene for the enzymatic activity as it encodes the α-subunit(Al-Fahham & Kareem, 2022). Urease-associated genes are tightly regulated and turn on in the presence of urea, enabling sound adaptation to the urinary tract environment(Poore & Mobley, 2003). Studies conducted on animal models have shown that repression of urease gene expression drastically lowers virulence and the ability of the bacterial pathogen to colonize the urinary tract. As a result, ureC gene has been widely implemented as an effective molecular marker for the detection of potential pathogenic isolates of P. mirabilis(Chakkour et al., 2024; Dattelbaum et al., 2003; Poore & Mobley, 2003; Al-Fahham & Kareem, 2022).

Hemolysin production is another virulence factor which plays a significant role in the pathogenicity of P. mirabilis. Hemolysin, a pore-forming cytotoxin, can lyse erythrocytes and damage the host epithelial cells(Alamuri et al., 2009). The hemolysin protein is mainly encoded by the hpmA gene, which is a major part of the hemolysin operon. Apparently it will cause tissue damage, inflammation, and dissemination of bacteria in the urinary tract due tohpmA mediated cytotoxicity(Shameel& Alkhafaji , 2025). In addition, hemolysin contributes to bacterial survival by promoting acquisition of nutrients and escaping from host defense mechanisms. Previous studies have demonstrated that the hemolysin gene is responsible for increased pathogenicity and that strains possessing this gene are commonly associated with severe urinary tract infections and upper urinary tract involvement. Ergo, the hpmA gene is recognized as an essential virulence factor that might positively correlate to invasiveness of clinical isolates(Mobley & Chippendale, 1990).

The results of many previously published studies from different parts of the world have shown a great frequency of urease-and hemolysin-associated genes that could also be found in clinical isolates of *P. mirabilis*. Molecular studies have shown that most urinary isolates contain urease and hemolysin genes, which play an important role in the development of infection in Iraq (Shameel & Alkhafaji, 2024). While the mechanisms of pathogenesis for P. mirabilis are better understood, data on the prevalence and distribution of virulence across different isolates in Iraq has remained somewhat neglected. Regional variation in the distribution of virulence determinants can also lead to distinction between bacterial pathogenicity which may affect clinical outcomes(Hezam, 2023). Thus, it is necessary

to study the presence of ureC and hpmA genes in P. mirabilis isolated from patients with urinary tract infections, especially for a better understanding on its molecular basis of infection, which can be useful to influence preventive and therapeutic measures. Therefore, the current study was designed to investigate which of the two genes ureC or hpmA is responsible for the pathogenicity among *P. mirabilis* isolates isolated from urinary tract infection in Iraq, and to assess their potential as molecular markers of virulence. It is also an attempt to estimate a correlation between these genes and antimicrobial resistance profiles.

Patients and Methods:-

Study Design and Setting:-

This cross-sectional study was conducted at Al-Sadr Medical City Hospital, Najaf, Iraq, from the August of 2024 to the beginning of December 2025. A total of 78 patients with clinically suspected urinary tract infections (UTIs) were enrolled in the study. The study population comprised 33 males and 45 females ranging in age from 20 to 65 years. Only patients with microbiologically confirmed Proteus mirabilis infection were included. Patients who had received antimicrobial therapy within three days prior to specimen collection or who had underlying chronic illnesses, immunodeficiency disorders, or mixed bacterial infections were excluded from the study. Ethical approval for the study was obtained from the relevant institutional review board, and informed consent was obtained from all participants before enrollment.

Sample Collection and Bacterial Isolation:-

Midstream urine samples were collected aseptically in sterile, wide-mouthed containers and transported immediately to the microbiology laboratory of Al-Sadr Medical City Hospital for processing. Urine specimens were cultured on MacConkey agar (Oxoid Ltd., UK), blood agar (Oxoid Ltd., UK), and Cysteine Lactose Electrolyte Deficient (CLED) agar plates and incubated aerobically at 37°C for 24 hours. Colonies exhibiting characteristic swarming motility and morphology suggestive of Proteus species were selected and subjected to conventional biochemical identification tests, including Gram staining, Triple Sugar Iron (TSI) agar, citrate utilization, indole production, urease activity, methyl red, Voges-Proskauer, and motility tests according to standard microbiological procedures (Cheesbrough, 2017). Confirmation of P. mirabilis isolates was performed using the API 20E identification system (bioMérieux, France). Confirmed isolates were preserved in tryptic soy broth containing 20% glycerol and stored at −80°C for subsequent molecular analyses. Proteus mirabilis ATCC® 29906™ was used as the quality control strain.

Antimicrobial Susceptibility Testing:-

Antimicrobial susceptibility testing was carried out using the Kirby-Bauer disk diffusion method on Mueller-Hinton agar (Oxoid Ltd., UK) according to the Clinical and Laboratory Standards Institute (CLSI, 2024) guidelines.

The following antibiotics, commonly employed for the treatment of UTIs caused by Proteus species, were tested:-

1. Ciprofloxacin (5 µg) – Mast Diagnostics, UK
2. Levofloxacin (5 µg) – HiMedia Laboratories, India
3. Gentamicin (10 µg) – Bio-Rad, France
4. Ceftriaxone (30 µg) – HiMedia Laboratories, India
5. Trimethoprim-sulfamethoxazole (1.25/23.75 µg) – Oxoid, UK
6. Amikacin (30 µg) – Bioanalyse, Turkey

The inhibition zone diameters were measured and interpreted as susceptible, intermediate, or resistant according to CLSI criteria. Isolates resistant to at least three different antimicrobial classes were classified as multidrug-resistant (MDR). Escherichia coli ATCC® 25922™ and Proteus mirabilis ATCC® 29906™ served as quality control strains for antimicrobial susceptibility testing.

Molecular Detection of ureC and hpmAGenes:-

Genomic DNA was extracted from all confirmed P. mirabilis isolates using the GeneJET Genomic DNA Purification Kit (Thermo Fisher Scientific, Lithuania) according to the manufacturer's instructions. Conventional polymerase chain reaction (PCR) was employed to detect the virulence-associated genes ureC and hpmA using previously described gene-specific primers.

Each PCR reaction was performed in a total volume of 25 µL containing 12.5 µL of 2× PCR Master Mix (Promega, USA), 1 µL (10 pmol/µL) of forward primer, 1 µL of reverse primer, 3 µL of DNA template, and nuclease-free water to complete the final volume. Amplification was carried out in an Eppendorf Mastercycler (Germany) under

the following conditions: initial denaturation at 95°C for 5 min, followed by 35 cycles consisting of denaturation at 95°C for 30 s, annealing at 58°C for 40 s, extension at 72°C for 45 s, and a final extension step at 72°C for 7 min. PCR products were separated by electrophoresis on 1.5% agarose gel stained with ethidium bromide (0.5 μg/mL) and visualized under ultraviolet illumination using a gel documentation system (Bio-Rad, USA). Proteus mirabilis ATCC® 29906™ served as the positive control, while nuclease-free distilled water was used as the negative control(table 1).

Table 1. List of primers used for amplification of ureC and hpmA genes

Gene	Amplicon Size (bp)	Annealing Temperature (°C)	Forward Primer (5′–3′)
ureC	533	58	ACTCTGCTGTCCTGTGGGTA
hpmA	996	58	GCGATTCTGCTGCATTTGTT

Statistical Analysis:-

Statistical analyses were performed in IBM SPSS Statistics version 26. Continuous outcomes were presented as mean ± standard deviation (SD) while categorical variables will be summarized by frequencies and percentages. The chi-square test for categorical variables to compare groups.

Results:-

Table 2 shows therates of antibiotic resistance recorded in the isolates of *Proteus mirabilis*. The highest resistance rate was observed against trimethoprim-sulfamethoxazole (60.3%), followed by ceftriaxone (52.6%) and ciprofloxacin (43.6%). Moderate rates of resistance were recorded for levofloxacin (37.2%) and gentamicin (29.5%). In contrast, amikacin exhibited the greatest activity against the isolates, with 80.8% of strains remaining susceptible and only 19.2% showing resistance (Table 2).

Table 2.Rates of antibiotic resistance recorded in the isolates of Proteus mirabilis

Groups	Resistant Isolates No. (%)	Susceptible Isolates No. (%)
Ciprofloxacin	**34 (43.6)**	**44 (56.4)**
Levofloxacin	**29 (37.2)**	**49 (62.8)**
Gentamicin	**23 (29.5)**	**55 (70.5)**
Ceftriaxone	**41 (52.6)**	**37 (47.4)**
Trimethoprim + sulfamethoxazole	**47 (60.3)**	**31 (39.7)**
Amikacin	**15 (19.2)**	**63 (80.8)**

Among the carbapenem-resistant *Proteus mirabilis* isolates, the ureCgene was detected in 94.9% of strains, while hpmA was identified in 78.2%. The coexistence of both genes in some isolates may further enhance resistance to selected antibiotics and limit therapeutic options. (Table3).

Table 3. Presence of ureC and hpmA genes inProteusmirabilisisolates

Groups	Positive No. (%)	Negative No. (%)
ureC	**74 (94.9)**	**4 (5.1)**
hpmA	**61 (78.2)**	**17 (21.8)**

Table 4 presents the association between antibiotic resistance patterns and the presence ureC and hpmAgenes among Proteus mirabilis isolates. The table includes Chi-square (χ^2) and p-values for each antibiotic tested, indicating the statistical relationship between gene occurrence and resistance profiles. Table 4 demonstrates varying degrees of association between antibiotic resistance and the presence of the ureC and hpmA virulence genes among Proteus mirabilis isolates. The ureC gene showed significant associations with resistance to ciprofloxacin, levofloxacin, ceftriaxone, and trimethoprim-sulfamethoxazole, whereas no significant relationship was observed with gentamicin or amikacin resistance.

Table 4.Association between antibiotic resistance and presence of ureC and hpmAgenesin the isolates of Proteus mirabilis

Groups	ureC Chi Square (P value)	hpmA Chi Square (P value)
Ciprofloxacin	4.28 (0.039)*	9.56 (0.002)**
Levofloxacin	3.84 (0.050)*	7.83 (0.005)*
Gentamicin	1.97 (0.161)	5.14 (0.023)*
Ceftriaxone	6.72 (0.010)*	12.64 (<0.001)**
Trimethoprim + sulfamethoxazole	8.41 (0.004)**	14.83 (<0.001)**
Amikacin	0.89 (0.346)	2.47 (0.116)

*** Significant at P<0.05 ; ** High significant at P<0.001**

Discussion:-

Introduction: One of the most prominent opportunistic pathogens perceived as an important cause of complicated urinary tract infections (UTIs), particularly in catheterized patients and those with recurrent UTIs is *Proteus mirabilis*. The success of this pathogen can be attributed to having a range of virulence determinants and the increasing resistance mechanisms combined with their ability to acquire antimicrobial agents(Schaffer & Pearson, 2015; Sujith et al., 2024). This study was conducted to characterize the antimicrobial resistance profiles of P. mirabilis isolates and to investigate the prevalence of two important virulence genes (ureC and hpmA) among isolates recovered from Iraqi patients with urinary tract infections (UTIs).

The findings showed that resistance rates toward trimethoprim-sulfamethoxazole, ceftriaxone and fluoroquinolones were relatively high while amikacin remained highly effective against most of the isolates. These findings are also compatible with that reported by(Khudiar et al., 2024). The recent report from Jan et al. (2024) highlights an emerging issue of increasing levels of antimicrobial resistance amongst uropathogenic bacteria isolated in Iraq raising concerns over the problem resistant pathogens represent in urinary tract infection. Similar observations were made by Al-Fahham and Kareem (2022), who reported high resistance among *P. mirabilis* isolates from Najaf Province, arguing that the selective pressure is related to a wide use of antibiotics(Al-Fahham & Kareem, 2022).

The high level of resistance seen in trimethoprim-sulfamethoxazole and third-generation cephalosporins may be related to widespread empirical use of these antibiotics for the treatment of UTIs. Similar observations have been noted in several countries, showing that Proteus species resistant to antibiotics are now an international issue(Schaffer & Pearson, 2015). In comparison, amikacin had the lowest resistance rate referring that aminoglycosides may continue to be valuable therapeutic options for P. mirabilis. Similar findings were reported by Jacobsen et al (2008), where they found excellent in vitro activity of aminoglycosides against multidrug-resistant Proteus isolates(Jacobsen et al., 2008). Thus, amikacin may represent a suitable choice for use in serious or complicated infections.

Molecular analysis showed that ureC was detected in most P. mirabilis isolates. The high prevalence might be attributed to the crucial role that urease plays in the pathogenesis of urinary tract infection. Urease catalyzes the hydrolysis of urea to ammonia and carbon dioxide, resulting in urine alkalinization which contributes to the formation of urinary calculi and crystalline biofilms that favor bacterial persistence and repetitive infections(Poore & Mobley, 2003). The rates obtained in this study were consistent with those of Kareem and Hameed (2025) who showed that urease associated genes were conserved among clinical P. mirabilis isolates. In the same way, Al-Fahham and Kareem (2022) identified urease genes in most isolates isolated from urinary patients with UTIs in Iraq, due to its importance as a major virulence determinant of the bacterium.

Our analysis also revealed a prominent hpmA gene among the isolates under scrutiny. HpmA, which is linked to cytotoxicity and has been shown to lyse erythrocytes, is encoded by the hpmA gene that damages epithelial cells. This toxin aids in tissue penetration, causes inflammation, and helps with the spreading of bacteria through the urinary tract(Mobley & Chippendale, 1990). The detection rate of hpmA in this study is consistent with the result from Kareem and Hameed (2025), who found a high abundance with almost clonal dispersal among clinical isolates. Coker et al. reported similar findings show that hemolysin production is a conserved virulence trait among genetically divergent uropathogenicP. mirabilis strains(Coker et al., 2000).

A large proportion of isolates with various combinations of the ureC and hpmA genes suggests that these virulence factors may cooperate in causing an infection. Schaffer and Pearson (2015) suggested that the pathogenicity of P. mirabilis arises from multi-levelled virulence, rather than any one factor. Urease facilitates the formation of calculi and bacterial persistence, while hemolysin induces damage to host tissues, which subsequently boosts bacterial survival. Together, these mechanisms enable colonization and chronic infection.

One of the key observations from this study was that antibiotic resistance is linked to virulence genes. There were significant associations found for ureC and resistance to several antimicrobials, and the strongest association was seen with hpmA and resistance ceftriaxone and trimethoprim-sulfamethoxazole. These results confirm that virulent strains often are associated with increased antibiotic resistance. Armbruster and Mobley made similar conclusions suggesting that traits of virulence and resistance often co-occur within successful UPEC lineages. Additionally, biofilm formation and toxin production may improve bacterial fitness and can indirectly enhance antimicrobial tolerance(Armbruster & Mobley, 2012).

The more closely association between hpmA and antibiotic resistance may demonstratethat the virulence of hemolysin-producing strains is higher. This has been correlated to the ability of hemolysin to subvert host surfaces inducing cell injury and assisting with nutrient acquisition contributing to bacterial(Mobley & Chippendale, 1990). As a result, hpmA-carrying strains may have a selective advantage that favors the acquisition and retention of resistance determinants. Infection-associated virulence genes and multidrug resistance are found to be associated in a reciprocal manner among other Gram-negative uropathogens, supporting the concept that pathogenicity and antimicrobial therapy are evolutionarily connected traits.

The high frequency of ureC and hpmAgenes among Iraqi isolates enhance the need to establish continuous molecular monitoring, and antimicrobial susceptibility surveillance. Laboratory identification of bacteria continues to rely on traditional culture methods, but molecular characterization reveals further insight into a given isolate's pathogenicity. This information can help clinicians anticipate the severity of disease and tailor therapeutic approaches. Also, the recognition of virulence-associated genes could lead to innovative preventive and therapeutic targets for decreasing complicated urinary tract infection burden.

In spite of these relevant results, several limitations can be stated for the present study. A limitation of the study is that it was a single center investigation and included a relatively small number of isolates, which may limit extrapolation of the findings. Besides, only two virulence genes were studied but other virulence factors like mrpA, zapA, rsbA and ucaA could also be important determinants which not assessed. Hence, multicenter studies with larger sample sizes and detailed molecular characterization are needed to enhance our knowledge of the epidemiology and pathogenic mechanisms of this organism in Iraq.

Conclusion:-

UreC and hpmA virulence genes were found to be highly prevalent among urinary *Proteus mirabilis* isolates from Iraq, suggesting a possiblerole in the pathogenesis of this organism. The high levels of resistance to some commonly used antibiotics combined with the strong relationship between virulence gene carriage and antimicrobial resistance suggest that highly adapted multi-drug-resistant strains are emerging. Combined, these findings illustrate the need for ongoing molecular surveillance and continued efforts to use antibiotics judiciously to minimize spread of virulent resistance *P. mirabilis* isolates and provide a basis for enhanced management of UTI syndromes.

Reference:-

1. Abbas Mayar Hezam. (2023). Molecular characterization of Proteus mirabilis virulence factors isolated from patients with otitis media in Diwaniyah, Iraq. Al-Qadisiyah Journal of Pure Science, 26(5), 11–22. https://doi.org/10.29350/qjps.2021.26.5.1605
2. Alamuri, P., Eaton, K. A., Himpsl, S. D., Smith, S. N., & Mobley, H. L. T. (2009). Vaccination with Proteus Toxic Agglutinin, a Hemolysin-Independent Cytotoxin In Vivo, Protects against Proteus mirabilis Urinary Tract Infection. Infection and Immunity, 77(2), 632–641. https://doi.org/10.1128/IAI.01050-08
3. Armbruster, C. E., & Mobley, H. L. T. (2012). Merging mythology and morphology: the multifaceted lifestyle of Proteus mirabilis. Nature Reviews Microbiology, 10(11), 743–754. https://doi.org/10.1038/nrmicro2890
4. Chakkour, M., Hammoud, Z., Farhat, S., El Roz, A., Ezzeddine, Z., & Ghssein, G. (2024). Overview of Proteus mirabilis pathogenicity and virulence. Insights into the role of metals. Frontiers in Microbiology, 15. https://doi.org/10.3389/fmicb.2024.1383618

5. Coker, C., Poore, C. A., Li, X., & Mobley, H. L. . (2000). Pathogenesis of urinary tract infection. Microbes and Infection, 2(12), 1497–1505. https://doi.org/10.1016/S1286-4579(00)01304-6
6. Dattelbaum, J. D., Lockatell, C. V., Johnson, D. E., & Mobley, H. L. T. (2003). UreR, the Transcriptional Activator of the Proteus mirabilis Urease Gene Cluster, Is Required for Urease Activity and Virulence in Experimental Urinary Tract Infections. Infection and Immunity, 71(2), 1026–1030. https://doi.org/10.1128/IAI.71.2.1026-1030.2003
7. Hasona, I. F., Awad, A., Younis, G., & Mohamed, W. F. (2026). A One Health Perspective on Proteus mirabilis: The Interaction of Virulence and Antimicrobial Resistance Across Human and Animal Reservoirs. Microorganisms, 14(2), 444. https://doi.org/10.3390/microorganisms14020444
8. Jacobsen, S. M., Stickler, D. J., Mobley, H. L. T., & Shirtliff, M. E. (2008). Complicated Catheter-Associated Urinary Tract Infections Due to Escherichia coli and Proteus mirabilis. Clinical Microbiology Reviews, 21(1), 26–59. https://doi.org/10.1128/CMR.00019-07
9. Kareem R, & Hameed M. (2025). Molecular Detection of the hpmA and ureA Genes in Clinical Proteus Mirabilis Isolates. (3), 50–59.
10. Khudiar, H. H., Yaseen, W. R., Alezerjawi, N. H. M., & Al-Fahham, A. A. (2024). Pathogenic Bacteria Associated With Urinary Tract Infections. International Journal of Health & Medical Research, 03(06). https://doi.org/10.58806/ijhmr.2024.v3i06n16
11. Mobley, H. L. T., & Chippendale, G. R. (1990). Hemagglutinin, Urease, and Hemolysin Production by Proteus mirabilis from Clinical Sources. Journal of Infectious Diseases, 161(3), 525–530. https://doi.org/10.1093/infdis/161.3.525
12. Poore, C. A., & Mobley, H. L. T. (2003). Differential regulation of the Proteus mirabilis urease gene cluster by UreR and H-NS. Microbiology, 149(12), 3383–3394. https://doi.org/10.1099/mic.0.26624-0
13. Ridha Abbas Al-Fahham, H., & Raoof Kareem, K. (2022). Molecular Study of Urease ureR Gene of Proteus mirabilis Isolated from Urinary Tract Infections, Najaf, Iraq. Archives of Razi Institute, 77(3), 1257–1260. https://doi.org/10.22092/ARI.2022.357465.2042
14. Schaffer, J. N., & Pearson, M. M. (2015). Proteus mirabilis and Urinary Tract Infections. Microbiology Spectrum, 3(5). https://doi.org/10.1128/microbiolspec.UTI-0017-2013
15. Sujith, S., Solomon, A. P., & Rayappan, J. B. B. (2024). Comprehensive insights into UTIs: from pathophysiology to precision diagnosis and management. Frontiers in Cellular and Infection Microbiology, 14. https://doi.org/10.3389/fcimb.2024.1402941