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**Faculty of Natural and Life Sciences**

**Department of Microbiology**

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End-of-cycle dissertation

Fort the attainment of

a **Master's degree**

Topic:

# Research on Isolated BMR from Community-Acquired Urinary Tract Infections at the Private Analysis Laboratory LALAOUI

**Presented by Miss:** Nesrine HACHEMAOUI

Defended on June 30, 2024

Before the jury composed of:

| | | |
|---|---|---|
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**To the members of the jury.**

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I dedicate this thesis to:

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To you who have been my greatest support and my inexhaustible source of encouragement. Your sacrifices and unwavering love have been the pillars of my success. Without you, this dream could never have become a reality. I am deeply grateful to have you as my parents and I hope to continue to make you proud.

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I also dedicate this work to my grandmother Taleb Ounissa and my aunt Kheira.

**To my friends** Yasmine, Katia, and Melissa**,**

Thank you for your unwavering support and friendship. Your encouragement and presence have been invaluable to me throughout this journey. I am truly grateful to have you in my life.

Nesrine

# List of abbreviations

**AMK :** Amikacin

**AMR :** Antimicrobial Resistance

**AMX/CLAV :** Amoxicillin/Clavulanate

**CAZ :** Ceftazidime

**CDC :** Centers for Disease Control and Prevention

**CFU :** Colony-Forming Units

**CFZ/TAZ :** Cefazolin/Tazobactam

**CHL :** Chloramphenicol

**CIP :** Ciprofloxacin

**CTX :** Cefotaxime

**DPA :** Dipicolinic Acid

**DNA :** Deoxyribonucleic Acid

**EDTA :** Ethylenediaminetetraacetic Acid

**ERT :** Ertapenem

**ESBL :** Extended-Spectrum Beta-Lactamase

**ESBLCARBA :** Carbapenem-resistant ESBLs

**FDA :** Food and Drug Administration

**FOS :** Fosfomycin

**FOX :** Cefoxitin

**GEN :** Gentamicin

**MBL :** Metallo-β-lactamase

**MDR :** Multidrug-Resistant

**MRSA :** Methicillin-Resistant Staphylococcus Aureus

**NF :** Nitrofurantoin

**OXA :** Oxacillin

**PBP :** Penicillin-Binding Protein

**PBPs :** Penicillin-Binding Proteins

**Qnr :** Quinolone Resistance

**RNA :** Ribonucleic Acid

**rRNA :** Ribosomal RNA

**TMP/SMX :** Trimethoprim/Sulfamethoxazole

**UTI :** Urinary Tract Infection

**WHO :** World Health Organization

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# Introduction

# Introduction

The discovery of antibiotics in the early 20th century marked a revolutionary medical advancement, transforming the treatment of bacterial infections and saving millions of lives ( Fleming et *al.*,1949) The introduction of penicillin in the 1920s inaugurated a golden age in which new classes of antibiotics were regularly discovered, primarily from soil organisms (Woodruf et *al.*, 2014 ). This period of progress enabled the effective treatment of various infections, including urinary tract infections, which are among the most common affecting the population (Kunin et *al.*, 1994 )

However, the widespread use of antibiotics quickly revealed a major issue : bacterial resistance. As antibiotics were extensively prescribed, bacteria began to evolve to survive these treatments, rendering some medications ineffective (Davies et *al.*, 2010). Antibiotic resistance has become a global medical challenge, particularly concerning in the context of urinary tract infections. These infections, though often benign when properly treated, can become complex and difficult to manage in the presence of antibiotic-resistant bacteria (Gupta et *al.*, 2011).

In Algeria, antibiotic resistance has become a major public health concern. Selfmedication with antibiotics accounts for 50% of general sales, significantly exacerbating this problem. A study reported in 2021 clearly indicates a troubling progression in the rates of resistant strains. Additionally, in 2018, Algeria ranked as the fifth-largest consumer of antibiotics worldwide (Attaba & Echikr (2021))

In the current context marked by a growing concern over the increasing antibiotic resistance, it is crucial to enhance our comprehension of this phenomenon. Confronted with a notable deficiency in surveillance system in Algeria, as well as a scarcity of studies dedicated to exploring resistance in community urinary tract infections, consequently, we have undertaken h research aimed at characterizing multidrug resistant bacteria associated with this infection at the Dr Laloui’s private laboratory analysis.

# Bibliographic synthesis

**Urinary tract infection (UTI)** is a common microbial infection found in all ages and sexes which involves inflammation of the urinary tract (Kaur et *al.,* 2020). They occur when uropathogens colonize the urinary tract, facilitated by their production of toxins, siderophores, and adhesins, which aid in colonization and invasion (Ait mimoun et *al.,* 2022) . UTIs can be categorized based on the source of infection, such as hospital-acquired and community-acquired infections. Community-acquired UTIs develop before a patient is admitted to a healthcare facility and not within 10 days after discharge (silago et *al.,* 2022). UTIs are caused by a high concentration of specific bacteria, and symptomatic patients generally present values $\geq 10^5$ CFU of bacteria per mL in their urine samples (Santos et *al.*, 2022). They are caused by both Gram-negative and Gram-positive bacteria, as well as by certain fungi (Flores-Mireles et *al.*, 2015) .

Urinary tract are classified as uncomplicated complicated (Geerlings et *al.,* 2012). Complicated UTI is less common and is associated with a structural or functional abnormality (e.g., urinary obstruction, neurologic disease, immunosuppression, renal dysfunction, or catheterization) as well as those that occur in women during pregnancy ( Melekos et *al.,* 2000). Uncomplicated UTI where there are no relevant functional or anatomical abnormalities in the urinary tract, no relevant kidney function impairment, and no relevant concomitant diseases promoting the UTI or risk of developing serious complications (Medina et *al.,* 2019) ; these infections are differentiated into lower UTIs (cystitis) and upper UTIs (pyelonephritis) (Flores-Mireles et *al.*, 2015) .

Cystitis (infection of the bladder or lower UTI) has the following symptoms: dysuria with or without frequency, urgency, suprapubic pain, or hematuria. Clinical manifestations suggestive of pyelonephritis (infections of the kidney or upper UTI) are fever (temperature >38°C) and chills, mental confusion as a sign of delirium, flank pain, costovertebral-angle tenderness, and nausea or vomiting (Geerlings et *al.,* 2012).

The distinction between complicated and uncomplicated infections is important because when complicating factors are present, antimicrobial resistance is more common and the response to therapy is often disappointing even with agents active against the pathogen. Furthermore, severe complications are associated with complicated UTIs which may lead to urosepsis, renal scarring or even to end-stage disease ( Melekos et *al.*, 2000).

**Enterobacteriaceae** is family within Gammaproteobacteria, encompasses a broad spectrum of Gram-negative bacteria, facultatively anaerobic, nonspore-forming, and rodshaped bacteria (Batisti Biffignandi et *al.*, 2021) usually inhabit the digestive tract of humans and mammals (Mamar et *al.*, 2019) This family, such as *Klebsiella, Enterobacter, Salmonella, Escherichia coli, Shigella, Providencia, Proteus, Serratia, Morganella, and Citrobacter*. Among the bacterial speciesbelonging to the Enterobacteriaceae family, we identify human pathogens responsible for a variety of infections :urinary tract infections (cystitis, pyelonephritis),septicemia, pneumonia, hepato-digestive infections (peritonitis, cholangitis), meningitis... Enterobacteriaceae are thus considered the main source of community and hospital-acquired infections, with Escherichia coli, by far the pathogen responsible for the greatest number of human infections (Dortet et *al.*, 2013 .The vast majority of established genera and species presently included in the family Enterobacteriaceae, order "Enterobacterales," have been recognized for over 50 years . From a biochemical standpoint, members of this family are in general catalase positive and oxidase negative, with the ability to reduct the nitrate to nitrite, and product acid starting from glucose fermentation (Batisti Biffignandi et *al.*, 2021) .

In the late 1970s and early 1980s, several antibiotics introduced to the market were effective against Enterobacter infections. However, over the past 25 years, these bacteria have developed resistance to these antibiotics, spreading globally and leaving carbapenems (imipenem, ertapenem, meropenem, etc.) as the only treatment option .Unfortunately, in the past decade, Enterobacter species have also developed resistance to this class of antibiotics, which was previously considered the last resort (Bovin et *al.*, 2016).

**Antibiotic target**

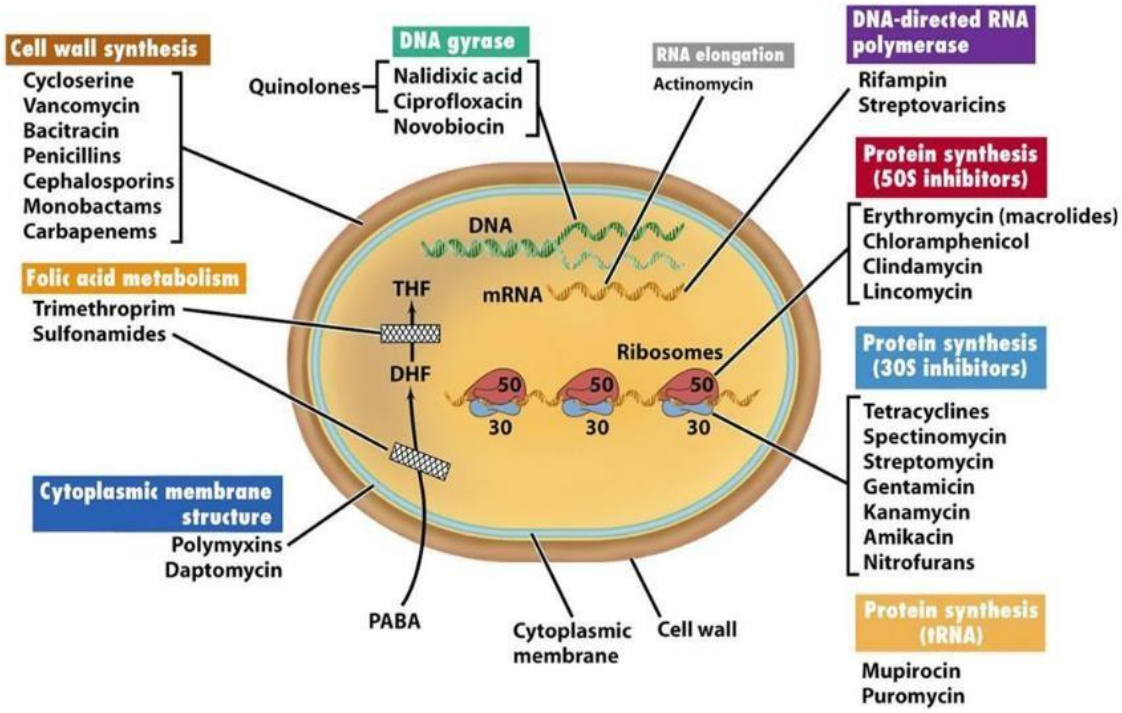


*Figure 1:Antibiotic target sites*

**Antibiotic resistance** is defined as the ability of microorganisms to counteract the action of antimicrobial agents and this phenomenon occurs when an antibiotic loses its efficiency to inhibit the bacteria ( Pulingam et *al.*, 2022) The introduction of antibiotics has revolutionized medicine, providing effective treatment for infectious diseases that were once fatal and enabling modern medicine, such as surgery and organ transplantation (Shukla et *al.*, 2023) Widespread resistance development thwarts the effectiveness and lifespan of antibiotics, calling for the discovery of new drugs in the perpetual standoff against human pathogens (Shukla et *al.*, 2023).

**The site of resistance** vary between bacterial species, and are classified into several pathways. In some cases, within the same bacterial strain, several different resistance mechanisms (Bouyahya et *al.*, 2017).

Bacterial resistance to antibiotics arises through several mechanisms. One common strategy is the modification of the antibiotic molecule itself, achieved by producing enzymes that either add specific chemical groups to the compound or destroy the molecule entirely, rendering it ineffective. Another approach involves altering the target sites of antibiotics within bacterial cells, either by protecting the target or modifying it to reduce the antibiotic's affinity. Additionally, bacteria can develop resistance through broader cellular adaptations, such as mechanisms to maintain cell wall synthesis and membrane integrity. Furthermore, the

acquisition of various efflux pump genes, both chromosomal and plasmid-related, enables bacteria to expel antimicrobial agents from the cell, leading to resistance against previously effective antibiotics. Examples include the multidrug-resistant efflux pumps found in bacteria like Pseudomonas aeruginosa and Escherichia coli (Munita et *al.*, 2015 ; Tanwar et *al.*,2014).

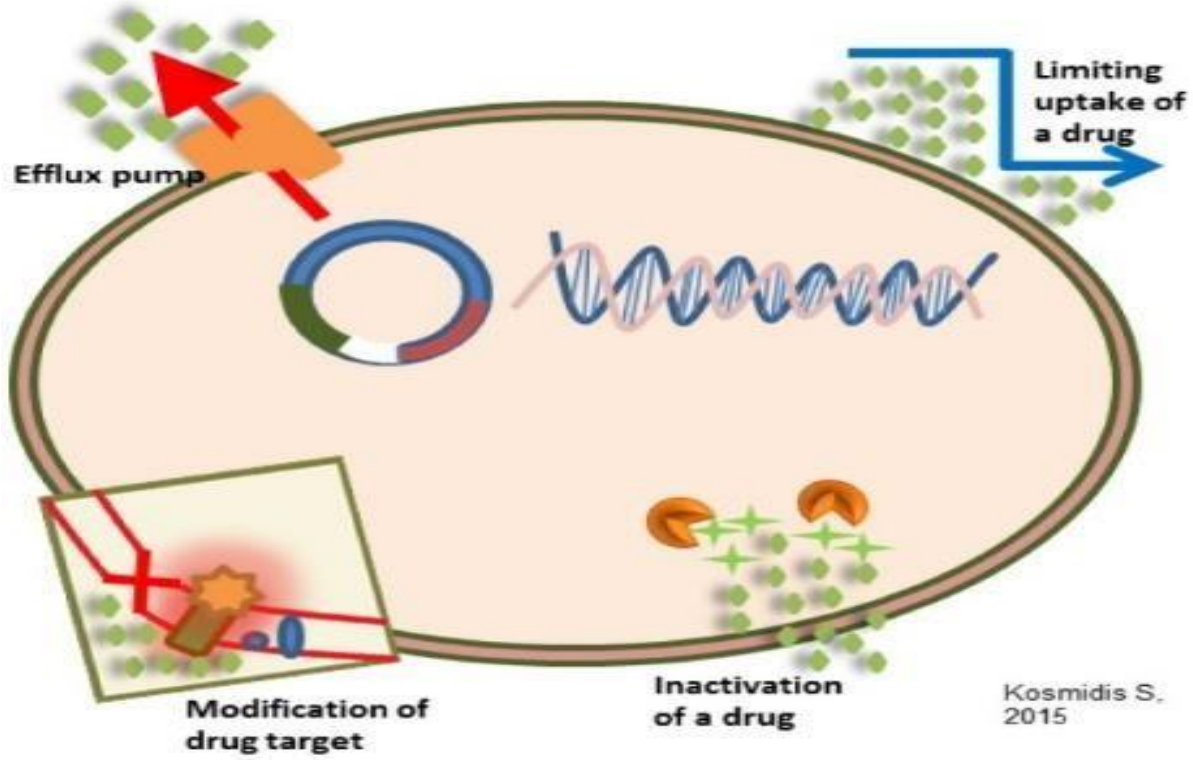


*Figure 2: General antimicrobial resistance mechanisms.*

**The resistance of the cephalosprine** is due to Three primary mechanisms cause antibiotic resistance against cephalosporins: (i) bacteria producing enzymes that inactivate the antibiotic, such as beta-lactamases, (ii) alterations in the target protein (PBPs), and (iii) reduced amounts of antibiotics reaching their target. In Gram-negative bacteria, resistance may occur due to low membrane permeability of antibiotics, structural or quantitative changes in porin, and the efflux pump. Resistance can be intrinsic or acquired through mobile genetic elements from other bacteria, and can be horizontally transferred between bacteria through conjugation, transformation, and bacteriophage transduction. Recently, new and stronger beta-lactamases have received attention, such as extended-spectrum beta-lactamases (ESBL), which are plasmid-encoded and able to hydrolyze third-generation cephalosporins. Complex resistances can cause complications in clinical diagnosis and limit options for efficient therapeutic antibiotics. Combinations of an antibiotic and a beta-lactamase inhibitor have been suggested to enhance

bactericidal action, even in the presence of a certain level of resistance to the antibiotic alone. (Lin et *al.*, 2022).

**ESBLs**, or Extended-Spectrum Beta-Lactamases, represent a diverse group of enzymes that have undergone structural and functional mutations from their ancestral β-lactamase counterparts.

Initially, β-lactamases were categorized using the Ambler classification system based on their molecular structure and the Bush–Jacoby–Medeiros classification system based on their functional characteristics. ESBLs primarily fall into classes A and D of the Ambler classification, where serine serves as the enzyme's active center. In the Bush–Jacoby–Medeiros system, ESBLs are grouped under group 2, based on their ability to hydrolyze β-lactam substrates and their response to inhibitors.

Further refinements in classification have led to the recognition of three main groups of ESBLs: Ambler class A ESBLs (ESBLA), miscellaneous ESBLs (ESBLM), and ESBLs capable of degrading carbapenems (ESBLCARBA). ESBLA, the most common group, includes various types of β-lactamases such as SHV, TEM, and CTX-M, with notable resistance profiles against different β-lactam antibiotics.

CTX-M-type ESBLs have gained prominence in recent outbreaks, displaying a preference for hydrolyzing cefotaxime and originating from non-pathogenic Enterobacteriaceae. They are divided into major types based on amino acid sequence variance and are frequently detected in various environments and organisms (Lin et *al.*, 2022).

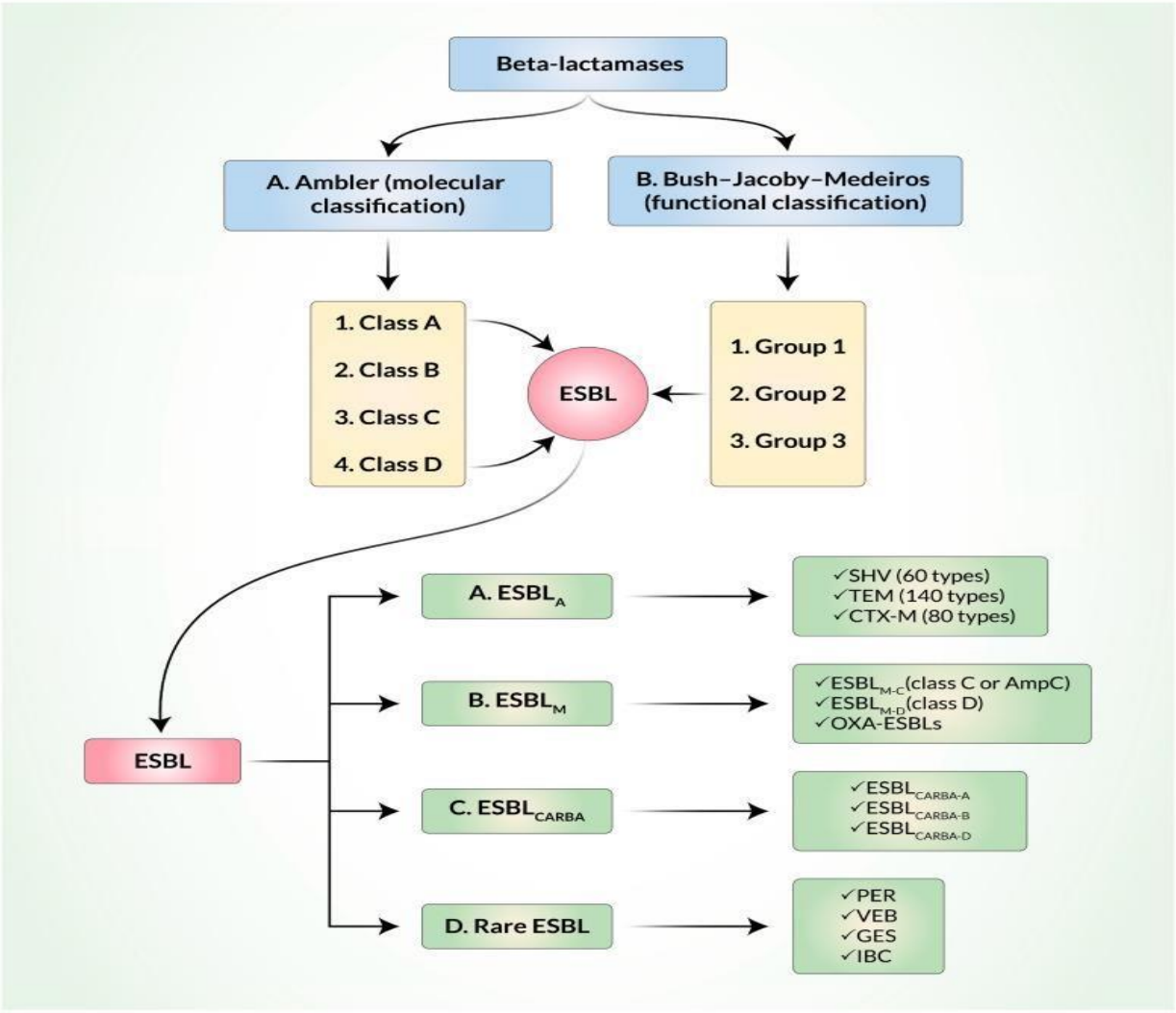


*Figure 3 : ESBL Classification: Ambler vs. Bush–Jacoby–Medeiros*

**Carbapenem** As the crisis of antimicrobial resistance escalates, dealing with carbapenem resistance in gram-negative pathogens presents a unique clinical hurdle. Carbapenems have traditionally been viewed as highly effective and powerful agents against multidrug-resistant (MDR) gram-negative pathogens (doi et *al.*, 2019) .Bacteria develop resistance to carbapenems through the action of carbapenemase enzymes, which break down carbapenem molecules. These enzymes are often carried on plasmids and can be easily transferred between bacteria. The Ambler classification system divides β-lactamase enzymes into four groups (A, B, C, D) based on their core catalytic region and preference for breaking down. Class A, B, and D contain carbapenemases, while class C enzymes primarily target cephalosporins. Class B enzymes are metallo-β-lactamases (MBLs) that use zinc in their active site. Avibactam, a newer drug, can block the action of class A, B, and D serine-β-lactamases, but not class B MBLs. Class C includes AmpC β-lactamase enzymes, which can contribute to

carbapenem resistance when combined with other factors like permeability issues. Class D enzymes, also known as oxacillin carbapenemases (OXA enzymes), are a diverse group of β-lactamases with significant carbapenemase activity. Stenotrophomonas maltophilia is naturally resistant to carbapenems due to the presence of a chromosomally encoded MBL called L1. Non-enzymatic mechanisms of carbapenem resistance involve various changes, including the downregulation of genes responsible for porin expression, mutations occurring in chromosomally encoded porin genes (like OprD), and the upregulation of genes encoding efflux pumps (such as MexAB-OprM, MexXY-OprM, or MexCD-OprJ), particularly noticeable in *P. aeruginosa*. Porins act as general channels in the outer membrane of gram-negative bacteria, facilitating the passive movement of hydrophilic molecules, nutrients, and even certain antibiotics across the otherwise impermeable membrane. The reduction in porin levels and the increased expression of efflux pumps linked with carbapenem resistance might also contribute to resistance against other βlactams and various antibiotic classes (Nordman et *al.,* 2019) .

**Aminoglycosides**, such as streptomycin, neomycin, and gentamicin, hinder protein synthesis by tightly binding to the A-site on the 16S ribosomal RNA (rRNA) of the 30S ribosomal subunit. Consequently, these antibiotics promote the misinterpretation of codons when aminoacyltransfer RNA is delivered. This results in flawed protein synthesis, where incorrect amino acids accumulate into a polypeptide that is subsequently released, causing damage to the cell membrane (Uddin et al., 2021) minoglycoside entry into bacterial cells involves three stages: electrostatic binding, energy-dependent entry, and rapid uptake. This leads to increased protein synthesis inhibition, mistranslation, and accelerated cell (Krause et *al.*, 2016)

Aminoglycoside resistance takes many different forms including enzymatic modification, target site modification via an enzyme or chromosomal mutation, and efflux. Each of these mechanisms has varying effects on different members of the class and often multiple mechanisms are involved in any given resistant isolate. Resistance to aminoglycosides via target site mutations has not been observed because nearly all prokaryotes, with the exception of Mycobacterium spp. and Borrelia spp , encode multiple copies of Rrna (Krause et *al.,* 2016)

**Quinolones** are bactericidal agents that inhibit the replication and transcription of bacterial DNA, leading to rapid cell death. They target two key antibacterial enzymes, DNA

gyrase (topoisomerase II) and DNA topoisomerase IV. DNA gyrase is made up of two subunits, GyrA and GyrB, and it introduces negative supercoils into DNA, facilitating the separation of daughter chromosomes. DNA topoisomerase IV consists of four subunits, two ParC and two ParE subunits, and is responsible for decatenating DNA, allowing it to segregate into two daughter cells. Quinolones interact with the enzyme-DNA complex, forming a drug-enzymeDNA complex that inhibits progression and the replication process (Uivarosi et *al.*, 2013)

Quinolone resistance is a growing clinical issue that threatens drug use due to specific mutations in gyrase and/or topoisomerase IV. Mutations at the serine and acidic residues disrupt the water-metal ion bridge, leading to high levels of resistance. These mutations may represent a "resistance mutation" that provides protection against naturally occurring antibiotics. Plasmid-mediated quinolone resistance is another mechanism, caused by mutations in gyrase and topoisomerase IV. This resistance can be transmitted horizontally or vertically and affects quinolone sensitivity. Three families of genes are associated with plasmid-mediated quinolone resistance: Qnr genes, aac(6′)-Ib-cr, and efflux pumps. These proteins confer quinolone resistance by decreasing the binding of gyrase and topoisomerase IV to DNA, lowering the number of available enzyme targets, and inhibiting quinolones from entering cleavage complexes. Quinolone resistance in Gram-negative bacteria is regulated by diffusion-mediated drug uptake and pump-mediated efflux. Changes in quinolone uptake and retention cause lowlevel resistance, but lowering cellular concentration can create a conducive environment for other forms of resistance (Aldred et *al.*, 2014).

# Materials and methods

## 1.1 Collecting samples

In this study conducted at Dr. Laloui's private laboratory, urine samples were collected from patients with community-acquired urinary tract infections (UTIs) over the period from 18/02/2024 to 05/05/2024.

## 1.2 Bacterial strain identification

The cytobacteriological examination of urine is a urine sampling procedure used to diagnose urinary tract infections. The sampling is performed on the first morning urine to obtain a more concentrated sample, which improves the reliability of the results. Patients were instructed to collect the midstream urine to reduce the risk of contamination. Prior to sampling, a thorough cleaning of the hands and genital area with antiseptic soap and water was performed.

The urine was collected in sterile containers, ensuring that there was no contact between the inside of the container and external parts of the body.

### 1.2.1 Sample Processing

During collection, urine samples undergo an initial macroscopic analysis to assess color, typically pale yellow, the presence or absence of turbidity, odor, etc. Subsequently, they undergo infection screening using the automated system 'Urised 3 Pro.' Positive cultures indicating bacterial growth are then selected for further analysis. "UriSed 3 PRO" **(figure 4)** represents a cutting-edge automated urine sediment analyzer. It features an innovative optical system that integrates bright-field and phase contrast microscopy. With its advanced capabilities, the Urised 3 Pro analyzer allows for the precise detection and quantification of a wide range of components found in urinary sediment. This comprehensive analysis extends beyond the identification of White Blood Cells (WBCs), Red Blood Cells (RBCs), epithelial cells, casts, crystals, bacteria, yeasts, mucus, Trichomonas, and renal tubular epithelial cells (RTE cells). It

provides valuable insights into the overall health status of the urinary tract, helping clinicians make informed diagnoses and treatment decisions.


*Figure 4: Urised 3 Pro Automated Analyzer*

### 1.2.2 Bacterial Species Identification

Positive cultures were inoculated onto chromogenic agar plates to facilitate bacterial identification. The plates were then incubated at 37°C for 24 hours to allow for bacterial growth. Species identification was based on the color and morphology of the colonies observed on the plates. The colors corresponding to each bacterial identification are listed in (**table 4)** in the appendix.

### 1.2.3 Confirmation of Identifications

Following the initial identification on chromogenic agar, bacterial identification was confirmed using the automated "Vitek 2 Compact" system, which allows for multiple biochemical tests **(table 5)** in the appendix. Subsequently, the vitek card **(figure 5)** in the appendix gallery was used to reconfirm the identification.

## 1.3 Antibiotics Susceptibility Test

After initial identification with the “Vitek 2 Compact system”, bacterial isolates were tested for antibiotic susceptibility. Bacterial suspensions were prepared and inoculated onto specialized test cards containing various antibiotics (**table 4** ) in the appendix . These cards were incubated in the “Vitek 2 compact”, which automatically monitored bacterial growth and interpreted results. The system generated a report indicating the susceptibility profile of the bacterial isolates to different antibiotics, aiding in the selection of appropriate antibiotic therapy.


*Figure 5: Automate Vitek Compact 2*

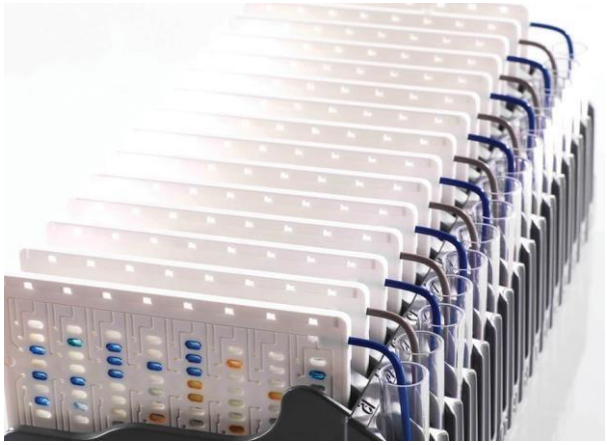

*Figure 6 : Vitek Card Gallery and Antibiogram*

## 1.4 Statistical analysis of the data

In this study, statistical analysis was conducted using Microsoft Excel software. Data pertaining to multi-resistant urinary tract infections, including microbiological profiles of urine samples and demographic characteristics of patients, were extracted from electronic medical records. Once collected, these data were imported into Excel spreadsheets for in-depth statistical analysis. Excel's built-in functions were utilized to calculate measures of central tendency, such as mean and odds ratio. Additionally, graphs were created to visually represent the distribution of patient ages, frequency of isolated bacteria, as well as gender distribution and specific bacteria identified. This approach facilitated a comprehensive analysis of the data, providing crucial insights into the prevalence of multi-resistant urinary tract infections and their clinical implications.

# Result

## 1.1 Urinary samples

During our study, we collected 171 urine samples from a diverse population, covering a wide age range from 1 month to 95 years, a including both sexes. This diversity enabled us to explore the distribution of bacterial strains in different demographic groups, and to examine potential variations in bacterial composition as a function of age and gender. In addition, we assessed resistance to various antibiotics to better understand resistance patterns within this population.

| | |
|---|---|
| Bacterial strains | 171 |
| Sex | F and M |
| Age range | 1 months to 95 years |
| Average age | 47.88 years |
| Sex-ratio | 2.98 |

## 1.2 Strains Frequency

This table presents the distribution of isolated bacterial strains, classified by species and gender (F for female, M for male), along with the overall total for each category.

**Table 1:** The distribution of isolated bacterial strains, classified by species and gender.

| Strains | F | F% | M | M% | Total | Total % |
|---|---|---|---|---|---|---|
| ***E.coli*** | 94 | 74.60 | 26 | 60.7 | 120 | 70.18 |
| ***Klebsiella pneumoniae*** | 20 | 15.8 | 9 | 20.93 | 29 | 16.96 |
| ***Proteus mirabilis*** | 7 | 5.5 | 1 | 2.33 | 8 | 4.68 |
| ***Pseudomonas aeroginosa*** | 2 | 1.59 | 2 | 4.65 | 4 | 2.34 |
| ***Serratia marcescens*** | 0 | 0.00 | 2 | 4.65 | 2 | 1.17 |
| ***Enterobacter aerogenes*** | 2 | 1.59 | 0 | 0.00 | 2 | 1.17 |

| | | | | | | |
|---|---|---|---|---|---|---|
| ***Providencia rettgri*** | 0 | 0.00 | 1 | 2.33 | 1 | 0.58 |
| ***Morganella morganii*** | 0 | 0.00 | 1 | 2.33 | 1 | 0.58 |
| ***Enterobacter coalcae*** | 0 | 0.00 | 1 | 2.33 | 1 | 0.58 |
| ***Enterobacter cloacae complex*** | 1 | 0.79 | 0 | 0.00 | 1 | 0.58 |
| ***Citrobacter koseri*** | 1 | 0.79 | 0 | 0.00 | 1 | 0.58 |
| ***Citrobacter freundi*** | 1 | 0.79 | 0 | 0.00 | 1 | 0.58 |
| ***Total général*** | **126** | **100.00** | **43** | **100.00** | **171** | **100.00** |

## 1.3 Distribution of Bacterial Strains in Positive Urine Culture Results

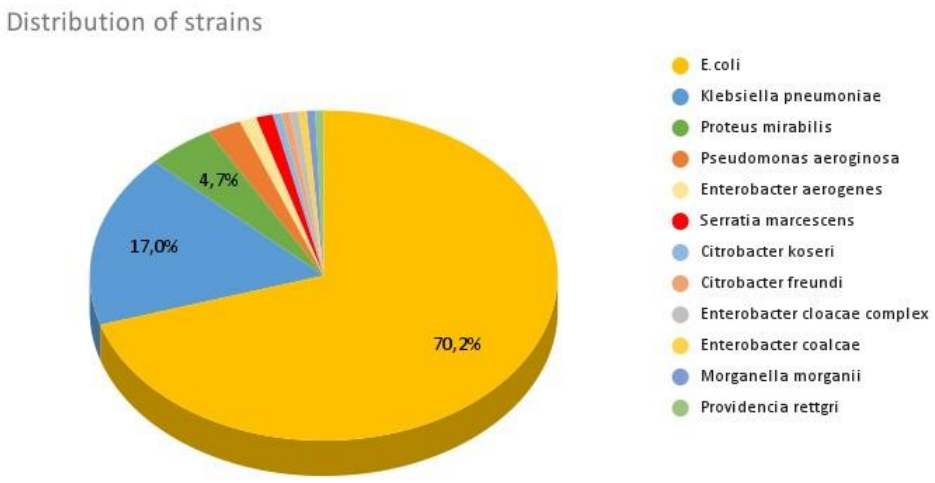


*Figure 7: Distribution of Bacterial Strains in Positive Urine Cultures*

The distribution of bacterial strains identified in the positive urine culture (ECBU) **(figure 8)** results shows an overwhelming predominance of *Escherichia coli* (*E. coli*), accounting for 70.2% of the cases. The second most common strain is *Klebsiella pneumoniae*, present in 17.0% of the cases. The other strains are much less frequent: *Proteus mirabilis* (4.7%), *Pseudomonas aeruginosa, Enterobacter aerogenes, Serratia marcescens, Citrobacter koseri,*

*Citrobacter freundii, Enterobacter cloacae complex, Enterobacter coalcae, Morganella morganii, and Providencia rettgeri* are all present in negligible proportions. These results highlight the predominance of *E. coli* in urinary tract infections, followed by *Klebsiella pneumoniae*, with other pathogens occurring relatively rarely.

## 1.4 Distribution of Positive Urine Culture Results  Gender

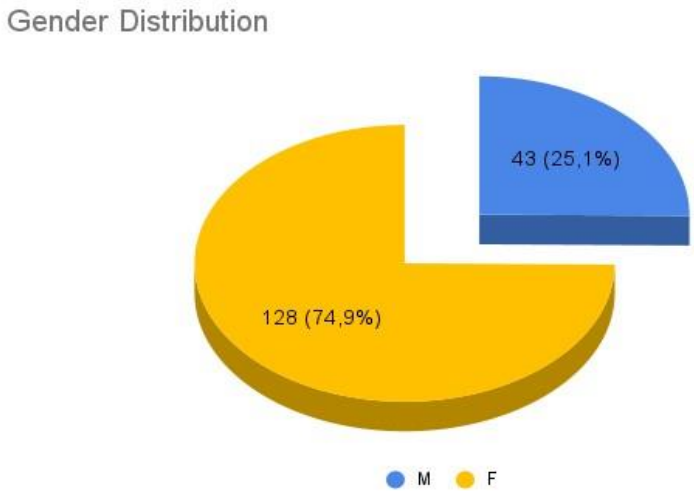


*Figure 8: Gender Distribution of Positive Urine Culture  Results*

The distribution of positive urine culture **(figure 8)** results by gender reveals a clear female predominance. Out of 171 cases, 128 are women, representing 74.9% of the cases, while 43 are men, representing 25.1% of the cases. This significant difference suggests that urinary tract infections detected by ECBU are much more frequent in women than in men in this studied population.

### 1.4.1 Odds ratio

OR=43/128 ≈ 2.98

The odds ratio (OR) is approximately 2.98. This means the odds of a UTI being diagnosed in females are about 2.98 times higher than in males in the studied population

## 1.5 Distribution cases by Age categories

***Table 2*:** Disrtibution cases by Age categories and sex

| | 0-1 year | 2-15years | 16-64 years | <65 years | Total |
|---|---|---|---|---|---|
| **F** | 2 | 18 | 70 | 38 | 128 |
| **M** | 3 | 5 | 19 | 16 | 43 |
| **Total** | **5** | **23** | **89** | **54** | **171** |

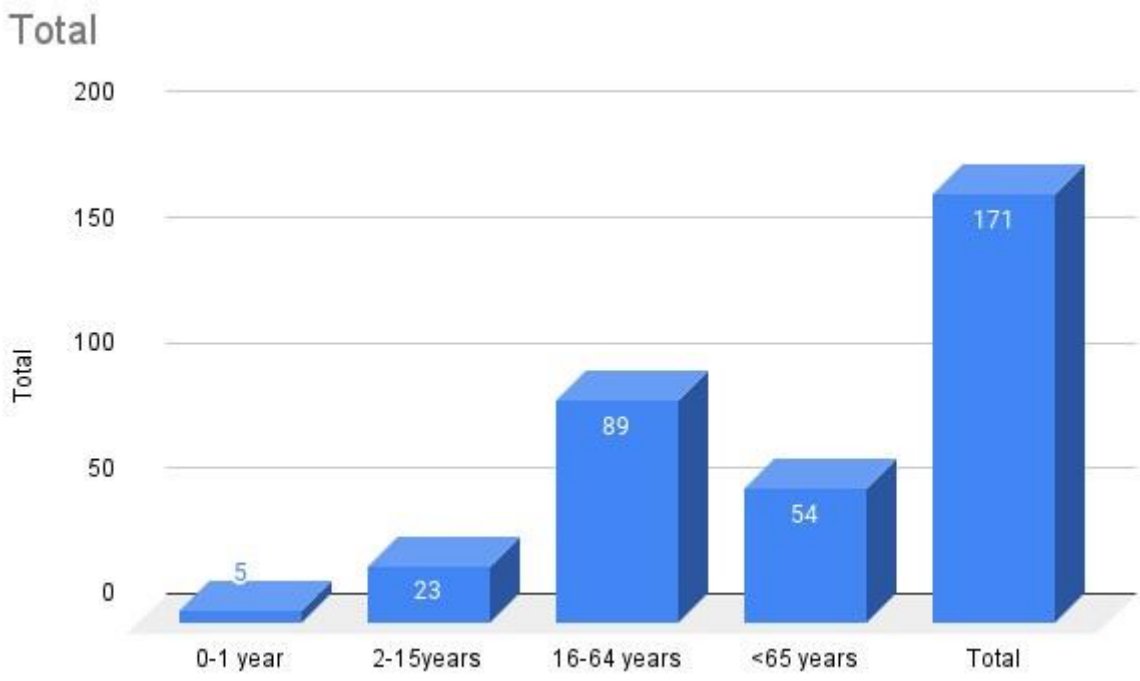


*Figure 9: Distribution of Bacterial Strains by Age Categories*

This graph **(figure 9)** illustrates the distribution of isolates by age categories:

In the 0-1 year age group, the number of diagnosed cases is relatively low, with 5 cases. For children aged 2 to 15 years, a notable increase in diagnosed cases is observed, reaching approximately 23 cases. In the 16-64 year age group, the highest number of diagnosed cases is observed, with approximately 89 cases. For individuals aged 65 years and older, there is a decrease compared to the previous age group, with approximately 54 diagnosed cases. Although the number of cases is lower than that of younger adults.

### 1.5.1 Distribution of age categories by sex

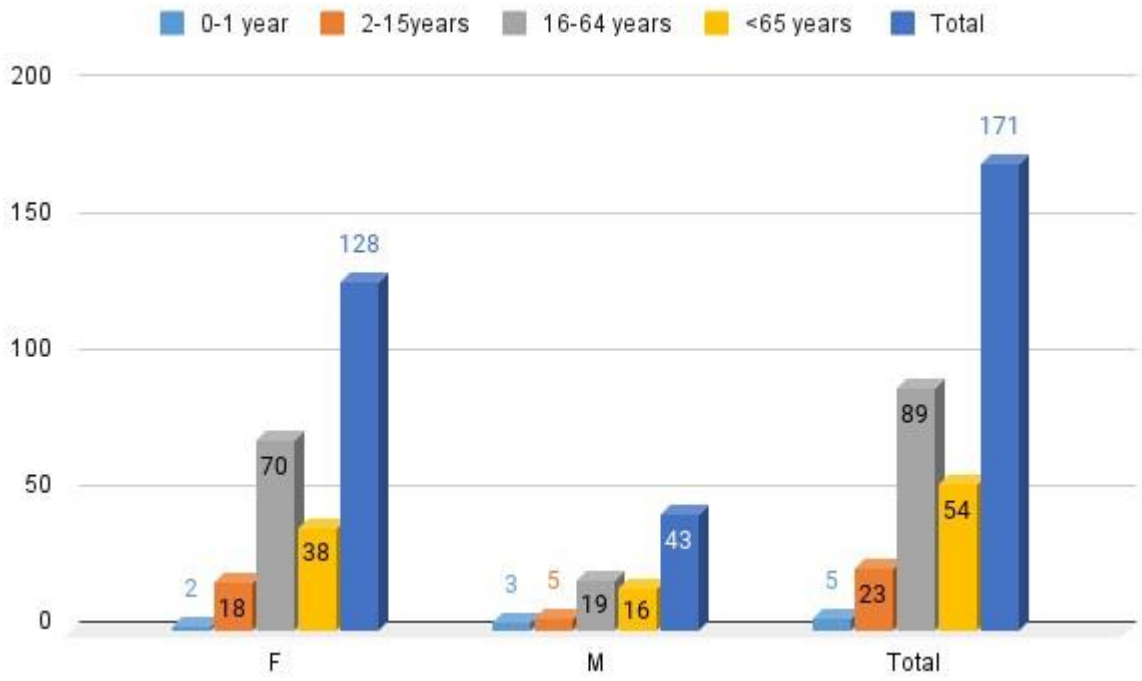


*Figure 10 :Bar Chart of Distribution of sex by Age Categories*

The bar chart **(figure 10 )** provides a breakdown of diagnosed bacterial infection cases by gender and age categories: 0-1 year, 2-15 years, 16-64 years, and 65 years and older. The categories are represented by different colored bars.

- Female (F) Distribution:
  - 0-1year: The number of cases is very low.
  - 2-15 years: There is a slight increase compared to the 0-1 year category.
  - 16-64 years: This category shows a significant increase, with the highest number of cases among females, nearly reaching 100 cases.
  - 65 years and older: There is a decrease from the 16-64 year category, but the number remains substantial.
- Male (M) Distribution:
  - year: The number of cases is very low, similar to females.
  - 2-15 years: There is an increase, but it remains relatively low compared to females.
  - 16-64 years: There is a noticeable increase, but it is still much lower than the number of cases in females of the same age category.
  - 65 years and older: The number of cases decreases compared to the 16-64 year category but is comparable to females in the same age group.

### 1.5.2 The average age

During the study on community-acquired urinary tract infections, the average age of participants was calculated to be **47.88 years**. This average represents the mean age of individuals included in the studied sample, providing a general indication of the age distribution within the studied population

## 1.6 Antibiotic test

**Table 3:** The prevalence of antibiotic resistance

| Antibiotic | Prevalence % | n = |
|---|---|---|
| AMP | 77.78% | |
| AMX/CLAV | 34.50% | |
| PIP/TAZ | 17.50% | |
| CFZ | 39.18% | |
| FOX | 10.53% | |
| CTX | 16.37% | |
| CAZ | 14.62% | |
| ERT | 2.34% | |
| IMP | 7.60% | |
| AMK | 7.60% | |
| GEN | 8.77% | |
| CIP | 16.37% | |
| FOS | 18.13% | |
| NF | 12.87% | |
| CHL | 9.36% | |
| TMP/SMX | 42.11% | |

### 1.6.1 Antibiotic Resistance Rates of 171 Bacterial Strains

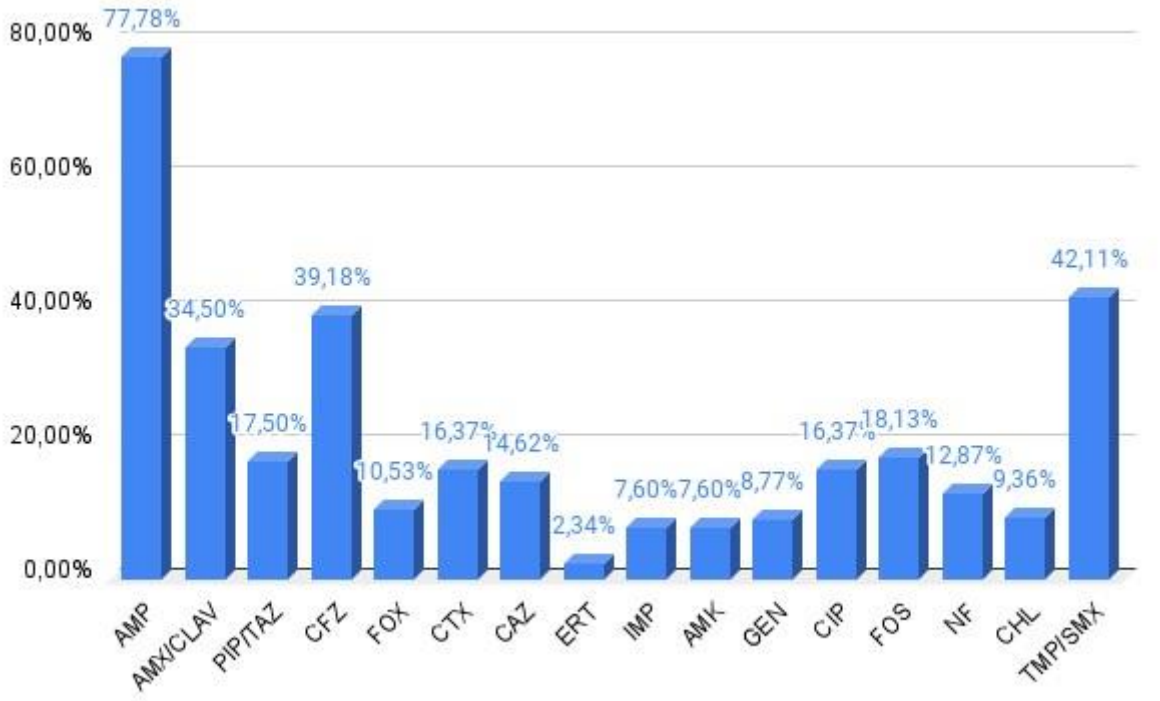


*Figure 11: Histogram of Antibiotic Resistance Prevalence*

These results **(figure 11)** reveal a high prevalence of resistance to several commonly used antibiotics, particularly ampicillin (77.78%) and trimethoprim/sulfamethoxazole (42.11%). Carbapenems (ertapenem (2.34%) and imipenem (7.60%)) and, to a lesser extent, cefoxitin (10.53%) and aminoglycosides (amikacin (7.60%) and gentamicin (8.77%)) show better efficacy against these strains. This underscores the importance of continuous surveillance of antibiotic resistance and prudent antibiotic use to prevent exacerbation of this public health issue.

### 1.6.2 Prevalence of strains resistant to certain antibiotic

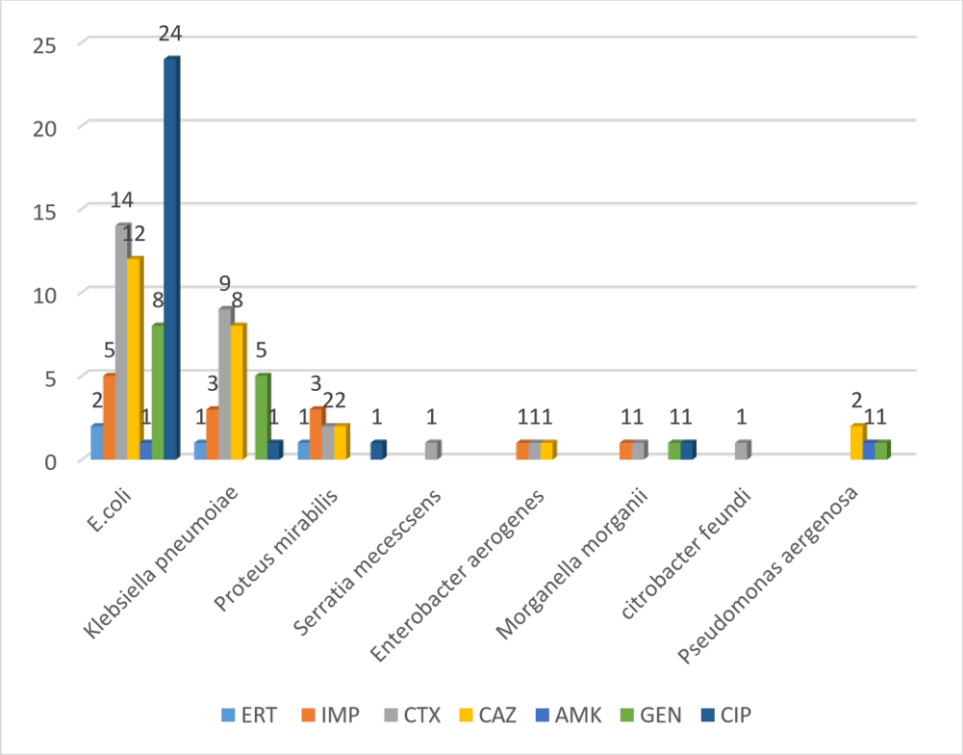


*Figure 12: Histogram of Prevalence of Strains Resistant to Certain Antibiotics*

The provided bar chart **(figure 12)** illustrates the resistance patterns of various bacterial strains to different antibiotics :

- *E. coli* shows notable resistance to CIP (24 resistant isolates), followed by CTX (14 isolates) and CAZ (12 isolates). Resistance to AMK and ERT is much lower, with 1 and 2 resistant isolates, respectively.
- *Klebsiella pneumoniae* exhibits moderate resistance with a maximum of 9 resistant isolates to CTX. Resistance to other antibiotics is less pronounced, with no notable resistance to AMK.
- *Proteus mirabilis* displays overall lower resistance, with a maximum of 3 resistant isolates to IMP. No resistance was observed for AMK.
- *Serratia marcescens* shows virtually no significant resistance, except for one isolate resistant to CTX.
- *Enterobacter aerogenes* has low resistance, with one isolate resistant to IMP, CTX, and CAZ.
- *Morganella morganii* shows low resistance, with one isolate resistant to IMP, CTX, GEN, and CIP.

# Discussion

The analysis of the distribution of strains isolated from urinary tract infections reveals a notable predominance of *Escherichia coli*, representing 70.18% (n=120) of all isolates. This observation is consistent with the literature, which identifies Escherichia coli as the main pathogen in community-acquired urinary tract infections. In Algeria, numerous studies[34] [35] [36] [37] have reported similar results, confirming the high prevalence of *Escherichia coli* in this type of infection. *Klebsiella pneumoniae* follows with a prevalence of 16.96% (n=29) , aligning with trends reported in other studies, where this species is often cited as the second most common pathogen in urinary tract infections. Similar results have also been reported in Italy[38] and Poland[39]. These bacteria reside in the lower intestinal tract of warm-blooded vertebrates, where they lead a seemingly harmless existence until they gain access to a niche, such as the urinary tract, where they can cause disease [40]. Other strains, such as *Proteus mirabilis* (4.68% n=8) and *Pseudomonas aeruginosa* (2.34% n=4), though less frequent, still play a significant role.

The examination of the distribution of community-acquired urinary tract infections by gender, as indicated in the attached pie chart (figire 8),

Reveals a clear predominance of females over males. Out of the 171 cases analyzed, 128 (74.9%) are female, compared to 43 (25.1%) male cases, with a female-to-male ratio (F/M) of 2.98. This distribution is not surprising and aligns with literature data demonstrating a predisposition of females to urinary tract infections nducted in Guelma, Algeria[37], as well as the one carried out in Italy[38], reported similar results.

. This difference is primarily explained by anatomical factors. Specifically, the shorter length of the female urethra can facilitate the passage of bacteria from the urethral opening to the bladder. Additionally, colonization of the vaginal introitus by gastrointestinal pathogens can increase the risk of urinary tract infection. Factors such as urinary tract obstruction, incomplete voiding, and anatomical anomalies also contribute to the predisposition to urinary tract infections. Among other risk factors are a history of urinary tract infections, sexual intercourse, and the use of contraceptives containing spermicides. Although several comorbidities increase vulnerability to urinary tract infections, the majority of cases occur in otherwise healthy women[40]

The results show a significant variation in antibiotic resistance among the different bacterial strains responsible for community-acquired urinary tract infections. The high

resistance of *E. coli* to Ciprofloxacin (20%) and Cefotaxime (11.67%) is concerning. However, Amikacin shows only a 1% resistance rate, providing a valuable therapeutic option. These findings are consistent with trends observed in other similar studies.

It is important to note, however, that resistance to Imipenem is observed at a rate of 4.17%, which is slightly higher than those reported in other studies, such as the one conducted by Ait Mimoun in Tizi Ouzou, Algeria[35], and another study in Guelma, Algeria[37]. In these studies, no cases of Imipenem resistance were reported. Similarly, a study in Tunisia[41] also revealed no resistance to Imipenem in *E. coli*.

In our study, *Klebsiella pneumoniae* exhibits high resistance to Cefotaxime (31.03%) and Ceftazidime (27.58%), as well as notable resistance to other antibiotics. These results are consistent with several previous studies [41]that have also reported increased resistance of *K. pneumoniae* to third-generation cephalosporins. Additionally, in our study, we observed a resistance rate of 3.44% to Ertapenem, in contrast to the study reported in Tunisia [41]where the resistance to Ertapenem was 0%.

An IMP-resistant strain of *Morganella morganii* was recorded during this study. On PubMed and Google Scholar, no study in Algeria has reported carbapenem resistance for this organism.

This resistance phenomenon is due to several factors, such as the sub-optimal use of antibiotics during treatment Its emergence is also due to prolonged hospitalization, long length of stay and co-morbidities, failure to observe hygienic practices and the transfer of patients between hospitals .[22]

Standardized definitions of resistance phenotypes have historically been a problem for the field of antimicrobial resistance research. . Magiorakos and colleagues suggested consensus definitions, Experts representing the Centers for Disease Control and Prevention (CDC) generated consensus definitions for multidrug-resistant (MDR), extensively-drug-resistant (XDR), and pandrug-resistant (PDR) [42].

MDR was defined as acquired resistance to at least one agent from three or more antimicrobial categories [20] .Bacteria categorized as XDR are epidemiologically significant not only because they resist multiple antimicrobial agents, but also due to the alarming possibility of being resistant to all, or nearly all, approved antimicrobial agents. In medical literature, XDR has been utilized as an acronym for various terms including 'extreme drug

resistance', 'extensive drug resistance', 'extre mely drug resistant', and 'extensively drug resistant'[26]. Pandrug resistant (PDR) is a term meaning a species or bacterial isolate is resistant to all antimicrobial agents. Definitions vary, but current examples include resistance to almost all commercially available antimicrobials, routinely tested antimicrobials, and all antibiotic classes available for empirical treatment [26].

Different molecular typing methods are being used to determine the spread of resistance and resistant microbes. Older methods like PFGE, AFLP, MST, and MLVA are being replaced by WGS, which provides a comprehensive view of the bacterial core and accessory genome, allowing discriminatory clonal relatedness and data on resistance genes, plasmids, and virulence-potential.[43] .

Antimicrobial resistance (MDR) leads to high mortality rates and medical costs, affecting the effectiveness of antimicrobial agents. It increases treatment costs and prolongs infection duration. Current medical applications, resistance profiles, and public hygiene quality also impact MDR effectiveness. Global trade and tourism expansion increase MDR's potential, affecting developing countries' economies and affecting exports and imports.[24]

High rates of recurrent UTIs suggest antibiotics are not effective for all UTIs. Translational research has been conducted to identify essential mechanisms of virulence and guide the development of UTI treatments and prophylactics that are optimized against uropathogens without altering the normal micro flora. Targeted therapies have been developed to neutralize pathogenic bacteria and prevent disease in animal models. However, more work is needed to develop new strategies for UTI treatment and prevention. The FimH vaccine is in Phase I clinical trials, but other potential therapies are still in the preclinical stages and have only been tested in animal models. Future clinical trials are essential for translating these antivirulence therapies into new treatments.[11]

# Conlusion

This study highlights the significant prevalence of *Escherichia coli* as the primary pathogen in community-acquired urinary tract infections (UTIs), corroborating existing literature and regional studies in Algeria. *Klebsiella pneumoniae* follows as the second most common pathogen, with its prevalence and resistance patterns aligning with international trends. The study also underscores the higher susceptibility of females to UTIs due to anatomical and physiological factors.

The findings reveal a concerning level of antibiotic resistance, particularly among *E. coli* and *Klebsiella pneumoniae* strains, with notable resistance to commonly used antibiotics such as ciprofloxacin and cefotaxime. Although amikacin remains an effective therapeutic option, the emergence of imipenem-resistant strains, including an IMP-resistant strain of *Morganella morganii*, poses a significant challenge:

- Continuous Surveillance of Antibiotic Resistance Profiles : Swift detection of emerging resistances for treatment adjustment.
- Optimization of Antibiotic Use: Promotion of judicious and rational antibiotic usage to reduce selective pressure.
- Development of New Treatments and Vaccines : Investment in targeted therapies against urinary pathogens.
- Enhancement of Hygiene Practices : Strengthening preventive measures to limit infection transmission**.**

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**Appendix:**

**Table 4**:Microorganisms' colors on Ureselect.

| Microorganisms | Appearance and color on Chromagar |
|---|---|
| *Escherichai Cloli* | Pink to move colored colonies |
| *Klebsiella, Enterobacter, Serratia, Citrobacter (KESC)* | Metallic blue colonies with a possible reddish halo |
| *Pseudomonas aerogenes* | Translucent colonies with a natural pigmentation ranging from cream to green |
| *Proteus, Morganella, Proviencia* | White to beige colonies with a swarming pattern |

**Table 5:** Vitek 2 compact biochemical tests.

| | | | | | | |
|---|---|---|---|---|---|---|
| APPA | O129R | ADO | BNAG | dMAL | LIP | dTAG |
| H2S | AGLU | ODC | GGAA | PyrA | AGLTp | dMAN |
| BGLU | PLE | dTRE | SUCT | LDC | IMLTa | IARL |
| ProA | dGLU | dMNE | TyrA | CIT | NAGA | IHISa |
| SAC | ELLM | dCEL | GGT | BXYL | URE | MNT |
| ILATk | AGAL | CMT | ILATa | BGAL | OFF | BAlap |
| GlyA | dSOR | 5KG | PHOS | BGUR | | |

**Table 6**: Antibiotics' MICs table.

| Antibiotic | Abbreviation | Famille |
|---|---|---|
| Ampicillin | AMP | Beta-lactams, Penicillins |
| Amoxicillin / Clavulanic acid | AMX , CLAV | Beta-lactams, Penicillins combined with a beta-lactamase inhibitor |

| | | |
|---|---|---|
| Piperacillin / Tazobactam | CFZ , TAZ | Beta-lactams, Penicillins combined with a beta-lactamase inhibitor |
| Cefazoline | CFZ | Beta-lactams, First-generation Cephalosporins |
| Cefoxitin | FOX | Beta-lactams, Second-generation Cephalosporins |
| Cefotaxme | CTX | Beta-lactams, Third-generation Cephalosporins |
| Ceftazidime | CAZ | Beta-lactams, Third-generation Cephalosporins |
| Erthapenem | ERT | Beta-lactams, Carbapenems |
| Amikacin | AMK | Aminoglycosides |
| Gentamicin | GEN | Aminoglycosides |
| Ciprofloxacin | CIP | Quinolones, Fluoroquinolones |
| Fostomycin | FOS | Phosphonic acid derivatives |
| Nitrofusranton | NF | Nitrofurans |
| chloramphenicol | CHL | Phenicols |
| Trimethoprim / sulfamethoxazole | TMP , SMX | ntimetabolites, Sulfonamides |

**Table 1**: Data Collected During the Study

| Strain | Code | Age | Sex | AMP | AMX/CLAV | PIP | CFZ | F |
|---|---|---|---|---|---|---|---|---|
| *E.coli* | 8383 | 1 year | M | | R | S | R | R |
| *Klebsiella pneumoniae* | 8945 | 82 years | F | S | S | S | S | S |
| *Proteus mirabilis* | 8802 | 85 years | F | R | I | S | S | S |
| *Pseudomonas aeroginosa* | 9348 | 10 years | F | R | I | S | S | S |
| *E.coli* | 8964 | 24 years | F | S | S | S | S | S |
| *Enterobacter aerogenes* | 10294 | 73 years | F | R | R | R | R | I |
| *Serratia marcescens* | 10163 | 68 years | M | R | I | S | S | S |
| *E.coli* | 10365 | 52 years | F | R | I | S | S | I |
| *E.coli* | 10059 | 91 years | F | R | R | R | R | S |
| *E.coli* | 10714 | 59 years | F | R | R | S | R | S |
| *E.coli* | 10887 | 14 years | F | R | I | S | S | S |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| *E.coli* | 10931 | 29 years | F | R | I | S | S | S |
| *E.coli* | 10953 | 7 years and 8months | F | R | S | S | S | S |
| *E.coli* | 10796 | 69 years | F | | S | S | S | S |
| *E.coli* | 11325 | 4 years | M | R | R | R | R | S |
| *E.coli* | 11087 | 72 years | M | | | S | R | |
| *E.coli* | 11292 | 7 years | M | R | R | I | R | S |
| *Citrobacter koseri* | 11224 | 1 year | F | S | S | S | S | I |
| *E.coli* | 11050 | 75 years | F | R | R | I | R | S |
| *E.coli* | 12385 | 41 years | F | R | I | S | S | S |
| *E.coli* | 12463 | 61 years | F | R | S | S | S | S |
| *E.coli* | 12385 | 41 years | F | R | I | S | S | S |
| *E.coli* | 12433 | 59 years | F | R | S | S | S | S |
| *Klebsiella pneumoniae* | 13039 | 65 years | F | R | S | S | S | S |
| *E.coli* | 13317 | 64 years | F | R | I | I | I | S |
| *E.coli* | 13233 | 60 years | F | R | I | S | R | S |
| *Klebsiella pneumoniae* | 13191 | 59 years | M | R | R | R | R | R |
| *E.coli* | 12636 | 72 years | F | R | R | S | R | R |
| *Klebsiella pneumoniae* | 280 | 72 years | M | R | R | S | R | S |
| *Klebsiella pneumoniae* | 297 | 78 years | F | R | I | I | R | S |
| *E.coli* | 337 | 71 years | F | S | S | S | S | S |
| *E.coli* | 470 | 64 years | M | R | R | R | R | S |
| *E.coli* | 472 | 63 years | M | R | R | I | R | R |
| *E.coli* | 1624 | 4 months | M | R | I | I | R | S |
| *Klebsiella pneumoniae* | 1463 | 21 years | F | R | | S | R | S |
| *E.coli* | 8083 | 84 years | M | S | S | S | S | S |
| *E.coli* | 7960 | 4 years and 6 months | F | R | R | I | S | S |
| *E.coli* | 7848 | 72 years | M | | R | S | R | R |
| *Citrobacter freundi* | 7762 | 69 years | F | R | R | I | S | S |
| *E.coli* | 7516 | 35 years | F | R | I | I | R | S |
| *E.coli* | 9163 | 79 years | F | R | S | S | S | S |
| *Klebsiella pneumoniae* | 9202 | 58 years | F | R | I | I | I | S |
| *E.coli* | 9720 | 2 years | M | R | | S | R | S |
| *Proteus mirabilis* | 8225 | 78 years | F | R | I | S | S | S |
| *E.coli* | 9074 | 59 years | F | R | R | R | R | S |
| *E.coli* | 8918 | 65 years | M | S | S | S | S | S |
| *E.coli* | 8392 | 65 years | M | | | S | R | |
| *Pseudomonas aeroginosa* | 7954 | 64 years | M | | R | S | R | R |
| *Enterobacter cloacae complex* | 8981 | 73 years | F | S | I | S | S | S |

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|---|---|---|---|---|---|---|---|---|
| *E.coli* | 8972 | 68 years | F | S | I | S | S | S |
| *E.coli* | 9720 | 2 years | F | R | | S | R | S |
| *Proteus mirabilis* | 9748 | 25 years | F | R | S | S | S | S |
| *Klebsiella pneumoniae* | 9971 | 36 years | F | R | R | S | S | S |
| *E.coli* | 8432 | 24 years | F | R | S | S | S | S |
| *E.coli* | 8587 | 1 month | M | R | S | S | R | S |
| *Klebsiella pneumoniae* | 8254 | 81 years | M | R | R | R | I | S |
| *E.coli* | 5607 | 42 years | F | R | R | R | R | S |
| *E.coli* | 5425 | 39 years | F | R | R | S | R | S |
| *Proteus mirabilis* | 5349 | 44 years | M | R | I | I | R | I |
| *E.coli* | 5304 | 2 years | F | R | R | I | I | S |
| *E.coli* | 5272 | 72 years | F | R | R | I | I | S |
| *E.coli* | 5170 | 3 months | F | R | I | S | S | S |
| *E.coli* | 4808 | 63 years | F | R | R | R | R | R |
| *Klebsiella pneumoniae* | 4241 | 79 years | F | R | I | S | R | S |
| *E.coli* | 4746 | 24 years | F | R | S | S | S | S |
| *E.coli* | 4261 | 50 years | F | R | S | S | S | S |
| *E.coli* | 4378 | 42 years | F | R | I | S | I | S |
| *E.coli* | 4694 | 63 years | F | R | R | R | R | S |
| *Klebsiella pneumoniae* | 4014 | 62 years | M | S | S | S | S | S |
| *E.coli* | 5072 | 39 years | F | R | S | S | S | S |
| *E.coli* | 3751 | 77 years | F | R | I | I | R | S |
| *E.coli* | 2609 | 68 years | F | R | R | R | S | S |
| *E.coli* | 3199 | 63 years | F | R | R | I | I | S |
| *E.coli* | 3198 | 95 years | F | R | R | I | R | S |
| *E.coli* | 3190 | 74 years | F | I | R | S | S | I |
| *E.coli* | 3060 | 40 years | F | R | R | R | R | S |
| *E.coli* | 3016 | 25 years | F | R | S | S | S | S |
| *E.coli* | 2717 | 53 years | F | R | I | S | R | S |
| *E.coli* | 2606 | 23 years | M | R | R | R | I | S |
| *E.coli* | 3113 | 27 years | F | R | I | S | I | R |
| *Proteus mirabilis* | 3349 | 42 year s | F | R | R | R | R | R |
| *Klebsiella pneumoniae* | 5807 | 53 year s | M | | R | | R | R |
| *Serratia marcescens* | 5890 | 33 years | M | | R | | R | R |
| *E.coli* | 5910 | 70 years | F | R | I | S | I | S |
| *E.coli* | 5937 | 62 years | M | R | I | S | S | I |
| *Pseudomonas aeroginosa* | 6664 | 78 years | M | | | R | R | |

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|---|---|---|---|---|---|---|---|---|
| *E.coli* | 6559 | 11 years | F | R | R | R | R | I |
| *E.coli* | 5974 | 12 years | F | R | R | R | I | S |
| *E.coli* | 6060 | 62 years | F | R | R | R | R | R |
| *E.coli* | 1979 | 76 years | F | R | S | S | R | S |
| *Enterobacter coalcae* | 770 | 53 years | M | | R | S | R | R |
| *E.coli* | 784 | 68 years | F | R | S | S | R | S |
| *E.coli* | 800 | 77 years | F | R | R | R | R | S |
| *E.coli* | 945 | 73 years | M | R | I | S | S | S |
| *E.coli* | 1502 | 43 years | F | R | S | S | S | S |
| *E.coli* | 1499 | 73 years | F | R | R | S | S | S |
| *Klebsiella pneumoniae* | 1030 | 32 years | F | R | S | S | S | S |
| *E.coli* | 3542 | 10 years | F | R | R | I | R | S |
| *E.coli* | 3372 | 67 years | F | S | S | S | S | S |
| *E.coli* | 3286 | 4 years et 4 months | F | R | I | S | S | S |
| *Klebsiella pneumoniae* | 3247 | 78 years | F | R | S | S | S | S |
| *E.coli* | 2919 | 60 years | F | R | R | R | R | S |
| *Klebsiella pneumoniae* | 2720 | 50 years | F | R | R | S | R | R |
| *Klebsiella pneumoniae* | 2617 | 71 years | F | R | I | S | R | S |
| *E.coli* | 1884 | 9 years and 9 months | F | S | S | S | S | S |
| *E.coli* | 1843 | 58 years | M | R | R | S | S | S |
| *Enterobacter aerogenes* | 1627 | 55 years | F | | R | S | R | R |
| *Klebsiella pneumoniae* | 2089 | 26 years | F | R | S | S | S | S |
| *E.coli* | 2035 | 3 years and 3 months | M | R | I | I | S | S |
| *Klebsiella pneumoniae* | 11047 | 87 years | F | R | R | R | R | R |
| *E.coli* | 10602 | 38 years | F | R | S | S | S | S |
| *E.coli* | 10229 | 55 years | F | R | S | S | S | S |
| *E.coli* | 10642 | 41 years | F | R | I | I | R | S |
| *E.coli* | 11269 | 74 years | F | S | S | S | S | S |
| *E.coli* | 52 | 19 years | F | S | S | S | S | S |
| *Klebsiella pneumoniae* | 195 | 92 years | M | R | R | R | S | R |
| *E.coli* | 10324 | 79 years | M | R | S | S | S | S |
| *E.coli* | 10217 | 5 years and 4 months | F | R | R | R | S | S |
| *E.coli* | 10825 | 6 years and 1 month | F | R | I | S | S | S |
| *E.coli* | 10326 | 86 years | F | S | S | S | S | S |
| *E.coli* | 227 | 44 years | M | R | I | R | R | S |
| *E.coli* | 272 | 39 years | F | R | I | S | S | S |
| *E.coli* | 461 | 59 years | F | S | S | S | S | S |

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|---|---|---|---|---|---|---|---|---|
| *E.coli* | 4109 | 21 years | M | R | S | S | S | S |
| *E.coli* | 3712 | 26 years | F | I | S | | S | S |
| *E.coli* | 4121 | 25 years | F | R | R | I | S | S |
| *E.coli* | 4079 | 3 years et 8 months | F | R | R | R | R | S |
| *Klebsiella pneumoniae* | 4616 | 37 years | F | R | I | S | S | S |
| *E.coli* | 4877 | 50 years | F | R | R | R | I | S |
| *E.coli* | 4842 | 5 years and 11 months | F | R | R | I | R | S |
| *Klebsiella pneumoniae* | 4449 | 46 years | F | R | I | I | R | S |
| *E.coli* | 4907 | 72 years | F | R | R | R | I | S |
| *E.coli* | 5052 | 7 years and 3 months | F | R | R | R | R | S |
| *E.coli* | 5939 | 63 years | F | S | S | S | S | S |
| *E.coli* | 6111 | 16 years | F | R | S | S | S | S |
| *E.coli* | 6649 | 21 years | F | R | R | I | S | S |
| *Klebsiella pneumoniae* | 5794 | 78 years | F | R | S | S | S | S |
| *E.coli* | 6631 | 34 years | F | R | I | S | S | S |
| *E.coli* | 5832 | 54 years | M | R | R | S | I | I |
| *Klebsiella pneumoniae* | 6163 | 89 years | M | R | I | I | R | S |
| *Klebsiella pneumoniae* | 6931 | 40 years | M | R | I | I | R | S |
| *E.coli* | 6985 | 55 years | F | S | S | S | S | S |
| *E.coli* | 7503 | 3 years et 8 months | M | R | I | S | S | S |
| *E.coli* | 6547 | 19 years | F | R | I | S | S | S |
| *Klebsiella pneumoniae* | 7275 | 68 years | M | R | I | S | R | S |
| *Pseudomonas aeroginosa* | 7365 | 29 years | F | | | | R | |
| *E.coli* | 8131 | 79 years | F | R | I | S | R | S |
| *Klebsiella pneumoniae* | 9715 | 57 years | F | R | I | S | R | S |
| *Morganella morganii* | 9789 | 64 years | M | R | R | R | R | R |
| *E.coli* | 7777 | 76 years | F | I | I | S | S | I |
| *E.coli* | 8596 | 30 years | F | R | S | S | S | S |
| *E.coli* | 8463 | 9 years and 9 months | F | R | I | S | S | S |
| *E.coli* | 8446 | 40 years | F | R | S | S | S | S |
| *E.coli* | 8253 | 76 years | F | S | S | SS | S | S |
| *E.coli* | 9312 | 76 years | F | R | I | I | S | S |
| *E.coli* | 9358 | 81 years | M | R | S | S | R | S |
| *E.coli* | 9800 | 64 years | F | I | S | I | S | S |

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|---|---|---|---|---|---|---|---|---|
| *E.coli* | 9549 | 40 years | F | S | S | S | S | S |
| *Klebsiella pneumoniae* | 9710 | 33 years | F | R | S | S | S | S |
| *Proteus mirabilis* | 9801 | 31 years | F | R | S | I | S | S |
| *E.coli* | 6111 | 16 years | F | R | S | S | S | S |
| *Klebsiella pneumoniae* | 10261 | 56 years | F | R | S | S | R | S |
| *E.coli* | 9866 | 50 years | M | S | S | S | S | S |
| *Proteus mirabilis* | 9892 | 4 years and 11 months | F | S | S | S | S | S |
| *Providencia rettgri* | 252 | 52 years | M | R | S | R | S | S |
| *E.coli* | 650 | 51 years | F | R | I | S | R | S |
| *E.coli* | 574 | 55 years | F | R | R | S | S | S |
| *E.coli* | 677 | 64 years | F | R | R | S | S | S |
| *Klebsiella pneumoniae* | 8451 | 52 years | F | R | S | S | S | S |
| *E.coli* | 549 | 81 years | M | S | S | S | S | S |
| *Proteus mirabilis* | 10006 | 27years | F | R | | S | I | S |

**General composition of CHROMagar agar**

**Peptone Mixture**: 15 g/L

**Chromogenic Mix:** 1 g/L (varies by manufacturer and specific CHROMagar type)

**Agar:** 15 g/L

**NaCl (Sodium Chloride):** 5 g/L

**Growth Factors and Supplements:** (quantities vary, proprietary mix)

**Selective Agents:** (quantities and types vary depending on target organisms)

**Résumé :**

**Introduction**: Les infections urinaires acquises en communauté représentent un problème de santé publique majeur en raison de l'émergence des résistances bactériennes. Cette étude examine les caractéristiques des bactéries multirésistantes dans les infections urinaires communautaires en Algérie**.**

**Matériel et Méthode:**Les échantillons d'urine ont été collectés entre le 18/02/2024 et le 05/05/2024. L'examen cytobactériologique des urines a été réalisé sur les premières urines du matin. Les cultures positives ont été identifiées à l'aide des systèmes automatisés "Urised 3 Pro" et "Vitek 2 Compact". Les tests de sensibilité aux antibiotiques ont été effectués sur les isolats bactériens.

**Résultats:** Haute résistance à Ciprofloxacine (20%) et Cefotaxime (11,67%) ,Faible résistance à Amikacine (1%) et Résistance à Imipenem (4,17%) chez *E.coli* . *Klebsiella pneumoniae* a marqué une Haute résistance à Cefotaxime (31,03%) et Ceftazidime (27,58%) et une Résistance à l'Ertapenem (3,44%), , *Morganella morganii* une souche résistante aux carbapénèmes (IMP) enregistrée, sans précédent rapporté en Algérie

**Discussion :** La prédominance d'Escherichia coli et la résistance élevée aux antibiotiques courants soulignent la nécessité d'une surveillance continue et d'une utilisation judicieuse des antibiotiques. Les facteurs anatomiques expliquent la plus grande susceptibilité des femmes aux infections urinaires

**Conclusion :** Cette étude met en évidence la prévalence élevée d'Enterobacteries dans les infections urinaires communautaires et la résistance importante aux antibiotiques en Algérie. La surveillance continue, l'optimisation de l'utilisation des antibiotiques, et le développement de nouveaux traitements sont cruciaux.

**Absract :**

**Introduction:**Community-acquired urinary tract infections (UTIs) are a major public health concern due to the emergence of bacterial resistance. This study examines the characteristics of multidrug-resistant bacteria in community-acquired UTIs in Algeria.

**Material and Methods:** Urine samples were collected between 18/02/2024 and 05/05/2024. Cytobacteriological examination of the urine was performed on the first morning urine. Positive cultures were identified using the automated systems "Urised 3 Pro" and "Vitek 2 Compact". Antibiotic susceptibility tests were performed on the bacterial isolates.

**Result:** High resistance to Ciprofloxacin (20%) and Cefotaxime (11.67%) ,Low resistance to Amikacin (1%) and Resistance to Imipenem (4.17%) in E.coli . Klebsiella pneumoniae showed high resistance to Cefotaxime (31.03%) and Ceftazidime (27.58%) and Resistance to Ertapenem (3.44%), , Morganella morganii a carbapenem-resistant strain (IMP) recorded, unprecedented reported in Algeria

**Discussion:**The predominance of Escherichia coli and high resistance to common antibiotics highlight the need for continuous surveillance and prudent use of antibiotics. Anatomical factors explain the greater susceptibility of women to UTIs

**Conclusion:**This study highlights the high prevalence of Enterobacteriaceae in communityacquired UTIs and significant antibiotic resistance in Algeria. Continuous surveillance, optimized antibiotic use, and the development of new treatments are crucial