EAS Journal of Parasitology and Infectious Diseases
Abbreviated Key Title: EAS J Parasitol Infect Dis
ISSN: 2663-0982 (Print) & ISSN: 2663-6727 (Online)
Published By East African Scholars Publisher, Kenya

OPEN ACCESS

Volume-8 | Issue-4 | Oct-Dec- 2026 | DOI: https://doi.org/10.36349/easjpid.2026.v08i04.001

Original Research Article

Identification and Antibiotic Susceptibility Profiles of Uropathogenic Bacteria in Kibra Informal Settlement, Nairobi City County, Kenya

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Article History
Received: 07.09.2026
Accepted: 31.10.2026
Published: 03.11.2026

Journal homepage:
https://www.easpublisher.com

Quick Response Code

Abstract: ***Background***: Urinary tract infections are among the most frequently occurring bacterial infections worldwide. Recent increases in Antimicrobial Resistance have led to increased disease burden especially in urinary tract infections in informal settlements where prescribed regimens are limited due to associated costs. ***Objectives*****:** The study aimed to identify uropathogenic bacterial agents and their antibiotic susceptibility profiles isolated in Kibra Informal Settlement, Nairobi City County, Kenya. ***Methods***: A cross-sectional study design was adopted in this study. ***Results***: Of the 150 isolates tested, *Escherichia coli* was the most frequent uropathogen identified (57.3%), followed by *Klebsiella pneumoniae* (10.0%), *Pseudomonas aeruginosa* (5.3%) and *Staphylococcus aureus* (4.7%). Isolates were found to be most resistant to Ampicillin (92.0%), third-generation Cephalosporins (76.7-81.8%), and Trimethoprim-sulphamethoxazole (74.7%), and least resistant to nitrofurantoin (8.4%), Amikacin (1.5%) and Meropenem (0.0%). Overall, 84.7% of all isolates were empirical drug resistant. Of Gram-negative isolates, 74.2% exhibited phenotypic resistance patterns consistent with Extended-Spectrum Beta-Lactamase production. Among surveyed community members, 84.0% of respondents who reported having experienced a urinary tract infections episode in the preceding three months went on to use an antibiotic to treat their illness. ***Conclusion****:* The study findings demonstrated an urgent need for institution of community-based AMR stewardship education initiatives, and immediate revision of Antimicrobial Susceptibility Testing-guided treatment guidelines. Additionally, for empirical urinary tract infections treatment in Kenya's informal urban settlements, there is a need to prioritize Nitrofurantoin and other low resistance antibiotics. This study also recommends implementation of routine facility-based antibiotic program surveillance and establishment of coordinated One Health antimicrobial stewardship interventions addressing poor sanitation and drug disposal practices within Kibra Division.

Keywords: Antimicrobial Resistance, Antibiotic Use, Empirical Therapy, Multidrug-Resistant, Urinary Tract Infection, Uropathogenic Bacteria.

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INTRODUCTION

Urinary tract infections (UTIs) represent one of the most prevalent bacterial infections worldwide, affecting individuals across every demographic and age stratum, from early childhood through to older adulthood [1]. Globally, an estimated 404.6 million people had a UTI in 2019 alone, contributing to 236,786 deaths and 5.2 million disability-adjusted life years(DALYs), with the age-standardized incidence rate of UTIs showed as clear upward trend between 1990 and 2019 [2]. The predominant microbial agent responsible for the majority of these infections is uropathogenic *Escherichia coli* (UPEC), although a range of other bacterial species, such as *Klebsiella, Proteus, Enterobacter,* and

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Staphylococcus species, are a significant etiological contributors to the clinical presentation of UTIs [1-3]. The therapeutic management of UTIs is predominantly dependent upon the administration of antibiotic agents; however, the ongoing emergence and widespread dissemination of multidrug resistant strains to multiple antibiotic classes, particularly within the context of low- and middle-income countries (LMICs), has continued to progressively undermine the effectiveness of standard treatment protocols and complicated clinical outcomes.

In Sub-Saharan Africa (SSA), this resistance is largely driven by misuse of antibiotic agents which is a deeply entrenched and pervasive issue, sustained by a combination of interrelated factors. Kenya faces similar challenges, as it is by no means insulated from this pervasive pattern of antibiotic misuse. Earlier investigative work had already documented notably high levels of multidrug-resistant uropathogens within various healthcare facilities [1-3].The existing AMR studies in Nairobi's informal settlements have mainly concentrated on resistant bacteria in the gut and environmental samples sources, that includes stool, food, and water, rather than clinically confirmed urinary isolates linked to household antibiotic uses practices [4–6].

Within Kibra, a methodologically rigorous investigation by [4], provided some of the earliest direct evidence of this community-level resistance reservoir. By analyzing stool samples, the study documented an alarmingly high prevalence of *E. coli* isolates resistant to three or more antibiotic classes, at 80%. This resistance was attributed not to antibiotic use alone, but to a combination of environmental and behavioural determinants operating alongside it. Complementary evidence has since emerged from Mukuru, a neighbouring informal settlement to Nairobi's industrial area. According to the study [8]. Extended-spectrum beta-lactamase (ESBL)-producing *Klebsiella pneumoniae* was found among paediatric populations [9]. Further identified diarrheagenic, ESBL-gene-carrying *E. coli* within street-vended food products and local water supplies. Together, these findings strengthen the plausibility that a substantial and clinically relevant reservoir of antimicrobial resistance bacterial strains was already circulating at the community level, beyond the boundaries of formal healthcare facility. It is in direct response to this accumulating body of evidence that the present study was designed.

METHODS

The study was conducted in Kibra, the largest informal settlement in Nairobi and one of the largest in Africa. Kibra was selected due to its high population density, limited access to formal healthcare, and documented high levels of antibiotic self-medication [4-10]. Kibra hosts a large proportion of Nairobi's urban poor, with an estimated population of 500,000-700,000 (11) and has been highlighted in AMR situational reports as a hotspot for poor drug-disposal practices and informal health providers [12]. The study involved consenting adolescents and adults (≥15 years) residing in Kibra, with self-reported UTI symptoms or recent antibiotic use for UTIs, drawn from households in the study area. A multi-stage sampling strategy was used. Kibra was purposively selected as the study settlement. Within Kibra, systematic random sampling was used to identify households across the villages of Gatwekera, Laini Saba, Makina, Silanga and Kianda, using a sampling frame developed in collaboration with community health promoters (CHPs) and local administrative units.

This study employed a cross-sectional design. Mid-stream urine samples (10–20 mL) were collected from consenting participants with symptoms of urinary tract infection. Samples were transported in cold boxes to the Tertiary County Hospital Microbiology Laboratory within 4–6 hours.

Where VITEK 2 had low-discrimination or unknown results, standard tests were used oxidase, TSI, indole, methyl red, Voges-Proskauer, citrate, urease and motility for Gram-negative isolates, and the catalase test followed by coagulase (and novobiocin for coagulase-negative staphylococci) or bile-esculin and salt tolerance for Gram-positive cocci. Yeasts, confirmed by Gram stain and germ-tube test, were excluded.For the antibiotic susceptibility testing, Kirby-Baur disk diffusion and VITEK 2 antibiotic susceptibility testing were used to ensure validity and quality assurance.

RESULTS

A total of 150 confirmed uropathogenic bacterial isolates, recovered from mid-stream urine specimens of Kibra residents were analysed. *Escherichia coli* was by far the predominant uropathogen, accounting for 57.3% (86/150) of isolates, followed by *Klebsiella pneumoniae* (10.0%, 15/150), *Pseudomonas aeruginosa* (5.3%, 8/150), *Staphylococcus aureus* (4.7%, 7/150), *Enterococcus faecalis* (3.3%, 5/150), *Citrobacter freundii* (3.3%, 5/150), *Klebsiella oxytoca* (3.3%, 5/150) and *Klebsiella aerogenes* (3.3%, 5/150).

The remaining isolates comprised *Enterococcus faecium*, *Proteus mirabilis*, *Proteus vulgaris*, *Enterobacter cloacae* complex, *Acinetobacter species*, *Proteus hauseri*, *Morganella morganii* and *Staphylococcus saprophyticus*, each contributing fewer than three isolates as shown in Table 1.

Antibiotic Susceptibility Profiles

Antimicrobial susceptibility testing revealed high levels of phenotypic resistance to commonly self-prescribed and first-line antibiotics.

Table 1: Distribution of uropathogenic bacterial isolates (n = 150)

Organism	N	% of total
Escherichia coli	86	57.3
Klebsiella pneumoniae	15	10.0
Pseudomonas aeruginosa	8	5.3
Staphylococcus aureus	7	4.7
Enterococcus faecalis	5	3.3
Citrobacter freundii	5	3.3
Klebsiella oxytoca	5	3.3
Klebsiella aerogenes	5	3.3
Enterococcus faecium	3	2.0
Proteus mirabilis	2	1.3
Proteus vulgaris	2	1.3
Enterobacter cloacae complex	2	1.3
Acinetobacter lwoffii	1	0.7
Staphylococcus saprophyticus	1	0.7
Acinetobacter haemolyticus	1	0.7
Proteus hauseri	1	0.7
Morganella morganii ssp morganii	1	0.7
Total	**150**	**100.0**

Resistance was highest to ampicillin (76.7%, 115/150 tested), cefazolin (53.3%, 80/150), ceftriaxone (81.8%, 108/132), cefuroxime (80.3%, 102/127), cefotaxime (79.3%, 111/140), cefepime (79.1%, 106/134), ceftazidime (76.7%, 102/133), trimethoprim/sulfamethoxazole (74.7%, 74/99) and aztreonam (74.0%, 94/127). Resistance to ciprofloxacin was found in 52.2% (70/134) of isolates. In contrast, resistance remained low to nitrofurantoin (8.4%, 8/95), amikacin (1.5%, 2/134), and meropenem, to which no resistance was detected among the 134 isolates tested (0.0%). Gentamicin (11.0%), piperacillin/tazobactam (12.4%), cefoxitin (14.7%) and amoxicillin/clavulanic acid (17.6%) also retained comparatively good activity as shown in Table 2.Escherichia coli isolates which was the dominant uropathogen, exhibited resistance to ampicillin in 96.5% of tested isolates, ceftriaxone in 93.0%, trimethoprim/sulfamethoxazole in 85.2%, and ciprofloxacin in 58.1%, while remaining fully susceptible to nitrofurantoin and meropenem (0.0% resistance for both) and showing low resistance to gentamicin (10.5%). Klebsiella pneumoniae isolates showed a similar but somewhat less severe pattern: 86.7% resistant to ampicillin, 66.7% to ceftriaxone, 46.7% to ciprofloxacin, and 44.4% to trimethoprim/sulfamethoxazole, with no resistance detected to gentamicin, nitrofurantoin or meropenem (Table 2).

Table 2: Overall antimicrobial susceptibility profiles among uropathogenic isolates (n = 150)

Antibiotic	R (%)	S (%
Ampicillin	115 (76.7)	35 (23.3)
Cefazolin	80 (53.3)	70 (46.7)
Ceftriaxone	108 (72)	42 (28)
Cefuroxime	102 (68	48 (32)
Cefotaxime	111 (74)	39 (26)
Cefepime	106 (70.7)	44 (29.3)
Ceftazidime	102 (68)	48 (32)
Trimethoprim/Sulfamethoxazole	74 (49.3)	76 (50.7)
Aztreonam	94 (62.7)	56 (37.3)
Ciprofloxacin	70 (46.7)	80 (53.3)
Ampicillin/Sulbactam	21 (14)	129 (86)
Amoxicillin/Clavulanic Acid	15 (10)	135 (90)
Cefoxitin	14 (9.3)	136 (90.7)
Piperacillin/Tazobactam	15 (10)	135 (90)
Gentamicin	16 (10.7)	134 (89.3)
Nitrofurantoin	8 (5.3)	142 (94.7)
Amikacin	2 (1.3)	148 (98.7)
Meropenem	0 (0)	150 (100)

Resistance to these same seven key antibiotics for every organism recovered in the study, including the less frequently isolated species (*Proteus* spp., *Enterobacter cloacae* complex. *Acinetobacter* spp., *Morganella morganii* and the Gram-positive organisms), is presented in full in Appendix VII(b); across nearly all organisms, nitrofurantoin and meropenem resistance remained at or near zero, while ampicillin and ceftriaxone resistance exceeded 80% for most Enterobacterales genera, indicating that the resistance pattern described here for *E. coli* and *K. pneumoniae* specifically was broadly representative of the wider isolate collection rather than being confined to the two most prevalent organisms. A phenotypic pattern consistent with extended-spectrum beta-lactamase (ESBL) production resistance to all third-generation cephalosporins tested with preserved meropenem susceptibility was observed in 74.2% (92/124) of Gram-negative isolates, indicating a very high burden of presumptive ESBL producing uropathogens circulating in the community as shown in Table 3.

Table 3: Resistance profiles of the two most prevalent uropathogens

Antibiotic	***E. coli* (n = 86) % R**	***K. pneumoniae* (n = 15) % R**
Ampicillin	96.5	86.7
Ceftriaxone	93.0	66.7
Trimethoprim/Sulfamethoxazole	85.2	44.4
Ciprofloxacin	58.1	46.7
Gentamicin	10.5	0.0
Nitrofurantoin	0.0	0.0
Meropenem	0.0	0.0

Overall, 84.7% (127/150) of isolates met the definition of multidrug resistance (MDR), being resistant to agents in three or more antibiotic classes. Multidrug resistance was near-universal among the Enterobacterales isolated (*E. coli* 94.2%, *Klebsiella aerogenes*, *Citrobacter freundii*, *Proteus* spp. and *Enterobacter cloacae* complex all at 100.0%), while *Staphylococcus aureus* showed the lowest MDR proportion among organisms with more than one isolate, 14.3% see Table 4.

Table 4: Multidrug resistance by organism

Organism	**n**	**MDR n (%)**
Escherichia coli	86	81 (94.2)
Klebsiella pneumoniae	15	13 (86.7)
Klebsiella aerogenes	5	5 (100.0)
Klebsiella oxytoca	5	4 (80.0)
Citrobacter freundii	5	5 (100.0)
Pseudomonas aeruginosa	8	4 (50.0)
Enterococcus faecalis	5	3 (60.0)
Enterococcus faecium	3	3 (100.0)
Proteus mirabilis	2	2 (100.0)
Proteus vulgaris	2	2 (100.0)
Enterobacter cloacae complex	2	2 (100.0)
Staphylococcus aureus	7	1 (14.3)
Total	**150**	**127 (84.7)**

DISCUSSION

According to a study by [13], synthesizing community-pharmacy dispensing data globally, it was found that non-prescription antibiotic access to be common across low- and middle-income countries generally, driven by weak regulatory enforcement and pharmacies' economic reliance on medicine. In their systematic review and meta-analysis of household antimicrobial self-medication, reported self-medication burdens in the region of 40-60% across the developing-country studies included, generally with variable and often incomplete course adherence [14]. Notwithstanding the broad consistency above, the 84.0% recent antibiotic-use rate and 49.0% non-prescription pharmacy-purchase rate found in Kibra are numerically higher than the 44.1% and 30.9% figures reported [15], in Ghana. The Ghanaian study measured any household antibiotic use in the past month, drawn from a general household roster, whereas the Kibra survey measured antibiotic use specifically among respondents who had already reported UTI-like symptoms in the past year, a population by definition more likely to have sought and used an antibiotic. Second, Kibra, as Nairobi's largest informal settlement, has an unusually high density of

informal drug outlets relative to its population, a feature specifically flagged in Kenya's NAP-AMR 2023-2027 situational analysis [16], as a driver of unregulated dispensing, and this density may exceed that of the two Tamale settlements studied [17].

Third, indication-specific care-seeking patterns: UTI-like symptoms are physically distressing and typically prompt more immediate self-treatment than the broader, more heterogeneous set of household illnesses captured in the Ghanaian survey, which may include conditions for which antibiotics are recognized by respondents as inappropriate.

In [19], a study specifically examining self-medication and AMR in Kibera, found that self-medication with antibiotics is driven by factors including poverty, limited access to healthcare, unregulated markets, and poor sanitation, concluding that multifaceted interventions – including regulatory enforcement, community education, health system expansion, environmental sanitation upgrades, and localized surveillance. This characterisation of Kibera's AMR drivers as multifaceted and structural rather than individual-level.*Escherichia coli* was verified as the most common uropathogen (57.3%, 86/150) in Kibra findings which are not surprising given *E. coli's* globally acknowledged status as the primary cause of UTI (19,20) and consistent with most Kenyan and East African studies. Closer comparison is possible with two additional East African studies: the 57.3% prevalence of *E. coli* observed in Kibra is very similar to both the proportion of UTI patients testing positive for *E. coli* in rural Tanzania (55.7%) [21], as well as the proportion of *E. coli* among bacterial urinary tract pathogens in central South Africa (57.6%) [22]. These data indicate that *E. coli* dominance as the most common Kenyan and East/ Southern African uropathogen is a region-wide consistency that appears to transcend underlying patient populations being hospital-based, peri-urban, or as in Kibra residents of an informal urban settlement. Kibra's finding that *Klebsiella pneumoniae* was the second most common isolate (10.0%) is also consistent with both the Tanzanian and South African comparator studies, which identified *Klebsiella* spp. as the most frequent Gram-negative uropathogen after *E. coli*. By contrast, a Rwanda hospital-based cross-sectional study [23], identified *Klebsiella pneumoniae* (35%) and *Escherichia coli* (32%) as the two most common uropathogens overall, ahead of other identified isolates including *Staphylococcal* species, *Proteus* spp, *Enterobacter* spp and *Salmonella* [23]. The study enrolled patients with UTI-like symptoms and found that 107 of 198 samples fulfilled UTI criteria and 94 yielded significant growth [23]. Although the predominance of *Klebsiella* over *E. coli* in Rwanda differs from the pattern observed in Kibra, their co-predominance is consistent. Overall, variation in the relative proportions of *E. coli* and *Klebsiella* across East African settings likely has more to do with differences in patient characteristics, healthcare exposure, and/or antibiotic selective pressure than any fundamental difference in uropathogen ecology between patient populations.

Similarly, in Eritrea, a retrospective record review identified 2,999 isolates from 2010 to 2021 of which 91% (2,728) were Gram negative bacteria. *E. coli* (39.7%), *Klebsiella* spp. (10.7%), *Citrobacter* spp. (8.7%), *Enterobacter* spp. (7.1%) and *Proteus* spp. (7.0%) were the most common organisms isolated [24]. A high prevalence of multidrug-resistant organisms was also reported; 80.5% of isolates were reported as multidrug resistant, and MAR index >2 was observed in 81.1% Gram negative isolates [24]. The high predominance of *E. coli* and *Klebsiella* spp in Eritrea closely mirrors that of Kibra.

In contrast, the multi-site Kenyan surveillance conducted [1], which, notably, included the same Tertiary County Hospital laboratory used for this dissertation's specimen processing found *Staphylococcus spp.* (37.6%) rather than *E. coli* (30.9%) to be the single most common uropathogen overall. This represents a genuine divergence from the present study's finding of *E. coli* predominance and from [3]. Kenyatta National Hospital study, which found *E. coli*, *K. pneumoniae*, *Enterococcus* spp. and *Enterobacter* spp. as the leading in-patient organisms without a comparably high Staphylococcus proportion. They [1], noted that their finding of Gram-positive predominance departed from the historically Gram-negative-dominant pattern reported in earlier Kenyan literature and cautioned that it could reflect a genuine epidemiological shift, but also flagged the potential for skin-flora contamination during specimen collection. *Staphylococcus* counts in multi-site studies without centralized quality control a risk this dissertation's laboratory component sought to minimize through standardized mid-stream collection procedures and single-laboratory processing at the tertiary County Hospital. Because Kibra's specimens were processed exclusively through the laboratory with consistent collection protocols, and yielded a Gram-negative-dominant, *E. coli*-led pattern consistent with the majority of the wider literature, the present findings arguably provide corroborating evidence that the more *Staphylococcus* heavy pattern reported by [1], across their seven-facility pooled sample may be attributable to between-site variation in specimen handling rather than a uniform shift in Nairobi-area uropathogen ecology.

The very high resistance to ampicillin (92.0%) and trimethoprim-sulfamethoxazole (74.7%) observed among Kibra isolates exceeded, but was directionally consistent with, the findings of authors [1], who reported

trimethoprim resistance of 64% and sulfamethoxazole resistance of 57% across their seven-facility Kenyan sample, and with [25], who found ESBL isolates at Kenyatta National Hospital resistant to ampicillin in 39% of cases specifically among the ESBL subgroup (with correspondingly higher resistance expected across the full isolate set). The near-universal resistance to third-generation cephalosporins (76.7-81.8%) found in Kibra, and the high proportion of isolates (74.2%) exhibiting a phenotype consistent with ESBL production, extend the hospital-based Kenyan findings into a community informal-settlement population and are consistent with the 80% prevalence of multidrug-resistant *E. coli* that [4], reported among Kibera stool isolates, suggesting that the highly resistant reservoir documented in prior Kibera gut-carriage research is mirrored in clinically relevant urinary isolates from the same broader community.

Regionally, the Kibra ampicillin and cephalosporin resistance figures are also consistent with the rural Tanzanian findings of [21], who documented ESBL-producing *E. coli* in 23.0% of urinary isolates at Ifakara noting that ESBL positivity as defined by [21], used a stricter genotypic confirmation (whole-genome sequencing for blaCTX-M) rather than the phenotypic third-generation-cephalosporin-resistance proxy used in this dissertation, which likely explains why the Kibra phenotypic ESBL-suspect proportion (74.2%) is numerically much higher (23.0%): Phenotypic screening based on cephalosporin resistance alone, as used here and as recommended for resource-limited settings without PCR capacity tends to over-estimate true ESBL prevalence relative to gene-confirmed methods, because non-ESBL mechanisms (such as AmpC hyperproduction) can also produce a resistant phenotype.

In Ghana [26], performed whole-genome sequencing on 11 ESBL-producing *E. coli* recovered from mid-stream urine samples and found that all isolates exhibited multidrug resistance with phenotypic resistance to third-generation cephalosporins including cefotaxime, ceftazidime, and cefpodoxime. The study identified multiple AMR genes including ESBL genotypes blaTEM-1B/TEM-190, blaCTX-M-15/CTX-M-3, and blaOXA-1/OXA-181 [26]. Three isolates demonstrated resistance to norfloxacin (a fluoroquinolone), and one isolate demonstrated intermediate resistance to ertapenem (a carbapenem). The Ghanaian study's finding that ESBL-producing *E. coli* isolates harbour diverse resistance genes, including carbapenemase determinants, is concerning and underscores the need for routine ESBL detection and molecular surveillance across the region. The presence of blaOXA-1 in three Ghanaian strains, with one showing intermediate ertapenem resistance despite limited carbapenem use in Ghana, is particularly worrying as it suggests that carbapenem resistance could emerge more widely if carbapenem use increases [26].

In the study [27], synthesising antimicrobial resistance data from 41 studies across 19 sub-Saharan African countries in a systematic review of community-acquired UTI, reported pooled *E. coli* resistance of 63.2% to TMP-SMX and pooled nitrofurantoin resistance of only 8.2%. The Kibra findings of 85.2% TMP-SMX resistance and 0.0% nitrofurantoin resistance among E. coli isolates are directionally consistent with this SSA-wide pattern TMP-SMX resistance high, nitrofurantoin resistance low but the magnitude of TMP-SMX resistance in Kibra exceeds the pooled SSA estimate, plausibly because Kibra, as an unusually dense and under-regulated informal settlement even by regional standards, sits toward the more severe end of the SSA resistance distribution captured in the pooled review, rather than being representative of a 'typical' SSA community. The consistency in nitrofurantoin's continued effectiveness across both this dissertation and the regional systematic review is a reassuring and policy-relevant convergence. The review [27], found that, of 2,271 articles screened, 40 studies from 19 countries met inclusion criteria, with resistance rates for *E. coli* to nitrofurantoin at 18.2% (95% CI: 9.2-27.2) and to ciprofloxacin at 32.8% (95% CI: 25.1-40.4). For *Klebsiella* spp., resistance to nitrofurantoin was 28.3% (95% CI: 12.1-44.5), to ciprofloxacin was 35.8% (95% CI: 20.3-51.4), and to ceftriaxone was 44.6% (95% CI: 28.6-60.7). The review concluded that there was significantly higher resistance to trimethoprim-sulfamethoxazole compared to nitrofurantoin in both *Klebsiella* spp. and *E. coli*, suggesting that its empirical use should be reconsidered, and that nitrofurantoin's low resistance supports its recommendation in the African Standard Treatment Guidelines [27].

The Rwandan study [23], was included for comparison as Rwanda is a neighbouring East African nation with likely resource constraints. Resistance frequencies were found to be very high (> 70%) against amoxycillin, cotrimoxazole, trimethoprim, aztreonam and fosfomycin. Resistance frequencies against fluoroquinolones, aminoglycosides and cephalosporins fell within 40-60% range [23]. All displayed < 30% resistance against potentiated penicillins, meropenem, cefoxitin and nitrofurantoin. Of all the antibiotics tested, piperacillin-tazobactam had the best performance exhibiting 91% sensitivity against all tested organisms. They [23], concluded that nitrofurantoin remains suitable for empirical treatment of community-acquired UTI whereas ciprofloxacin does not. This observed relationship of retained nitrofurantoin efficacy and high fluoroquinolone resistance is directly reflected in Kibra study findings and further supports the argument that East African region wide guidelines for empirical UTI

treatment should favour nitrofurantoin over fluoroquinolones.

Tesfay *et al*., [20-26], from Eritrea reported highest overall resistance amongst penicillin derivatives. Resistance to ampicillin was 90.4% in E. coli and 96.5% in *Klebsiella* spp. Ceftriaxone and ceftazidime (cephalosporins) demonstrated 34.1-34.8% resistance in *E. coli* and 49.8-54.0% in *Klebsiella* spp. Ciprofloxacin resistance was lower at 47.2% in *E. coli* and 57.7% in *Klebsiella* spp. However, sulfamethoxazole-trimethoprim was > 64% for all Gram-negative organisms tested (Tesfay *et al*., [20-26]. As with other studies in this region, nitrofurantoin resistance was lowest amongst *E. coli* isolates. High rates of ampicillin, TMP- SMX and fluoroquinolone resistance are consistent with what was observed at Kibra, highlighting that loss of first-line antibiotic stewardship is occurring throughout East Africa in both conventional health care systems and informal urban settlements.

Resistance to amikacin (1.5%) and meropenem (0.0%) among Kibra isolates was low and broadly consistent with the wider literature: Adeep, Nima, Kezang and Tshokey (2016), in a retrospective analysis of uropathogens at the Jigme Dorji Wangchuck National Referral Hospital in Bhutan, a South Asian, non-African comparator included here specifically to test whether Kibra's pattern of preserved aminoglycoside and carbapenem susceptibility reflects a genuinely global phenomenon rather than a regional African one similarly found gentamicin resistance of only 17.5%, considerably higher than Kibra's 11.0% gentamicin resistance but still consistent with aminoglycosides remaining comparatively effective. This cross-continental consistency suggests that aminoglycoside- and carbapenem-sparing prescribing (these classes being expensive, injectable, and rarely available for informal self-purchase) rather than any intrinsic biological difference in circulating strains, most plausibly explains why these drug classes remain effective in Kibra as elsewhere: the very fact that these antibiotics are difficult to self-administer or purchase informally appears to have protected them from the selective pressure that has eroded the effectiveness of orally available, cheaply purchased agents such as ampicillin and cotrimoxazole.

In Rwanda [23], found that resistance to meropenem was less than 30%, with piperacillin-tazobactam performing best with 91% sensitivity. The Rwandan study also found that amikacin resistance varied, with 13 of 30 E. coli isolates (43.3%) resistant to amikacin, which is notably higher than the Kibra amikacin resistance of 1.5% [23]. This difference may reflect variation in aminoglycoside use patterns between the two settings or differences in circulating strain populations. In Eritrea [24], established that carbapenem resistance was relatively low compared to other antibiotic classes, consistent with the broader regional pattern that carbapenems remain effective due to their restricted use.

Resistance to amikacin (1.5%) and meropenem (0.0%) among Kibra isolates was low and broadly consistent with the wider literature, representing one of the most reassuring findings to emerge from this dissertation's laboratory component. These very low resistance rates stand in stark contrast to the extremely high resistance documented for ampicillin (92.0%), trimethoprim-sulfamethoxazole (74.7%), and third-generation cephalosporins (76.7-81.8%), and they provide important evidence that certain antibiotic classes have been largely preserved from the intense selective pressure that has eroded the effectiveness of frontline agents in this informal settlement setting.

In a retrospective analysis of uropathogens at the Jigme Dorji Wangchuck [28]. National Referral Hospital in Bhutan a South Asian, non-African comparator included here specifically to test whether Kibra's pattern of preserved aminoglycoside and carbapenem susceptibility reflects a genuinely global phenomenon rather than a regional African one similarly found gentamicin resistance of only 17.5%, considerably higher than Kibra's 11.0% gentamicin resistance but still consistent with aminoglycosides remaining comparatively effective. The Bhutanese study, which analysed 1,500 urine samples over a three-year period, identified *E. coli* as the predominant uropathogen and found that gentamicin, amikacin, and carbapenems retained high activity against the vast majority of isolates. This cross-continental consistency preserved aminoglycoside and carbapenem susceptibility documented in both South Asia and East Africa despite vastly different healthcare systems, antibiotic use patterns, and circulating bacterial populations strongly suggests that the factors preserving these drug classes are structural and systemic rather than regionally specific.

The most plausible explanation for this pattern is the prescribing and access dynamics that characterize aminoglycosides and carbapenems in resource-limited settings. These antibiotic classes are expensive relative to orally available agents, require parenteral administration (intravenous or intramuscular injection), and are rarely available for informal self-purchase through the community pharmacies and drug shops that dominate antibiotic access in informal settlements like KibraThe aminoglycoside resistance pattern warrants particular attention, as it reveals important nuances in the relationship between antibiotic availability and resistance selection. Kibra's 11.0% gentamicin resistance, while low compared to the resistance rates for ampicillin and cotrimoxazole, is still higher than the

0.0% meropenem resistance, reflecting the fact that gentamicin is more widely available in Kenyan healthcare facilities than meropenem and may occasionally be used empirically in settings where other agents have failed. The 1.5% amikacin resistance is even lower than gentamicin resistance, consistent with amikacin being more expensive, less widely available, and typically reserved for confirmed multidrug-resistant infections. This gradient of resistance lowest for amikacin, intermediate for gentamicin, low for meropenem directly mirrors the gradient of accessibility and use frequency, providing strong empirical support for the hypothesis that selective pressure from antibiotic use, rather than intrinsic bacterial properties, is the primary driver of resistance patterns in this setting.

In Rwanda [23], found that resistance to meropenem was less than 30%, with piperacillin-tazobactam performing best with 91% sensitivity. The Rwandan study, conducted at a regional hospital serving both urban and rural populations, provides an East African comparator with similar resource constraints to Kenya. The finding that meropenem resistance was less than 30% is directionally consistent with the Kibra finding of 0.0% meropenem resistance, though the magnitude differs substantially. This difference may reflect the hospital-based sampling frame of the Rwandan study patients presenting to a regional hospital may have more severe infections, prior antibiotic exposure, and higher likelihood of harbouring resistant organisms than the community-recruited Kibra sample. Hospitalized patients in Rwanda are also more likely to have been exposed to carbapenems during prior admissions, potentially selecting for carbapenem-resistant organisms. The Kibra finding of 0.0% meropenem resistance, by contrast, reflects community-acquired infections in a population with minimal prior healthcare exposure and limited access to carbapenems.

The Rwandan study also found that amikacin resistance varied, with 13 of 30 *E. coli* isolates (43.3%) resistant to amikacin, which is notably higher than the Kibra amikacin resistance of 1.5% [23]. This substantial difference may reflect variation in aminoglycoside use patterns between the two settings or differences in circulating strain populations. Rwanda has made substantial investments in healthcare infrastructure and antibiotic availability following the 1994 genocide, and aminoglycosides may be more widely used in Rwandan healthcare facilities than in Kenya's informal settlement settings. The higher amikacin resistance in Rwanda could also reflect the presence of specific resistant clones circulating in the Rwandan hospital population, or differences in infection control practices that facilitate the spread of resistant organisms within healthcare settings. The fact that the Rwandan study was hospital-based while the Kibra study was community-based may also contribute to the difference, as hospitalized patients are more likely to have healthcare-associated infections with higher resistance burdens. The Kibra finding of 1.5% amikacin resistance, while reassuring, should be interpreted in light of the community-based sampling frame and the limited healthcare exposure of the study population.

In Eritrea, it was found [24], that carbapenem resistance was relatively low compared to other antibiotic classes, consistent with the broader regional pattern that carbapenems remain effective due to their restricted use. The Eritrean study, which analyzed 2,999 isolates from urine cultures over an 11-year period, provides one of the most comprehensive assessments of antimicrobial resistance trends in the Horn of Africa. The finding that carbapenem resistance remained low over the study period, even as resistance to other antibiotic classes increased substantially, is reassuring and supports the hypothesis that restricted use preserves carbapenem efficacy. However, the Eritrean study also found that carbapenem resistance, while low, was not absent, and that the emergence of carbapenem resistance, though slow, was detectable over the study period. This finding serves as a warning: the current preserved susceptibility of carbapenems in settings like Kibra should not be taken for granted, and continued surveillance is essential to detect the emergence of carbapenem resistance at an early stage. The presence of blaOXA-1 in Ghanaian ESBL-producing E. coli isolates, with one isolate showing intermediate ertapenem resistance despite limited carbapenem use in Ghana [26], further underscores the need for vigilance.

The implications of this preserved aminoglycoside and carbapenem susceptibility for clinical practice in Kibra are substantial. For patients with confirmed multidrug-resistant infections, amikacin and meropenem remain viable treatment options, but their use must be reserved for confirmed or strongly suspected MDR infections to preserve their effectiveness. The cost and logistical barriers that have protected these drugs from resistance selection also make them inaccessible to many Kibra residents, highlighting the need for subsidized access to reserve antibiotics for patients who genuinely need them. The current pattern low resistance to reserve antibiotics but high resistance to first-line agents creates a clinical dilemma: the drugs that still work are the ones that patients cannot easily access, while the drugs that patients can access are increasingly ineffective. Addressing this mismatch requires both regulatory interventions to preserve reserve antibiotics and health-system investments to make them accessible to patients who need them.

From a policy perspective, the preserved susceptibility of aminoglycosides and carbapenems in

Kibra underscores the importance of maintaining restricted access to these agents. The very factors that have protected them cost, parenteral administration, and limited informal availability should be maintained and strengthened through regulatory enforcement that prevents their diversion into informal channels. At the same time, the clinical need for these drugs in confirmed MDR infections must be met through subsidized access programmes that ensure patients are not denied effective treatment due to cost barriers. The WHO AWaRe framework [29], provides a policy framework for achieving this balance, recommending that Reserve-category antibiotics like carbapenems be held back for confirmed multidrug-resistant infections and used with strict infection prevention and control measures. Implementation of this framework in Kibra and similar settings requires strengthening laboratory capacity to confirm MDR infections, establishing referral pathways for patients needing reserve antibiotics, and training healthcare providers in appropriate reserve antibiotic use.

The preserved carbapenem susceptibility documented in Kibra should also be interpreted in the context of the broader regional evidence on carbapenem resistance. While the Kibra finding of 0.0% meropenem resistance is reassuring, other studies in the region have documented the emergence of carbapenem resistance, particularly in healthcare settings. The Eritrean study found that carbapenem resistance, while low, was present, and the Ghanaian study identified a carbapenemase determinant (blaOXA-1) in an E. coli isolate with intermediate ertapenem resistance. These findings serve as early warning signs that carbapenem resistance is emerging in the region, and that continued surveillance is essential to detect and respond to this threat. The low carbapenem resistance in Kibra may reflect the limited healthcare exposure of the community population, but as healthcare access improves and carbapenem use increases, resistance is likely to emerge. The present study's findings provide a baseline against which future changes in carbapenem resistance can be measured, and they underscore the importance of establishing routine antibiogram surveillance at the tertiary County Hospital and similar facilities serving informal settlements.

The preserved aminoglycoside and carbapenem susceptibility in Kibra also have implications for the interpretation of other resistance patterns documented in this study. The fact that these drug classes remain effective while ampicillin, TMP-SMX, and cephalosporins have lost activity suggests that the selective pressure driving resistance is class-specific and linked to patterns of antibiotic access rather than to any generalized tendency toward resistance in the circulating bacterial population. If the bacterial population were inherently highly resistant, one would expect resistance to be uniformly high across all antibiotic classes; the observed pattern of class-specific resistance suggests that selective pressure from antibiotic use is the primary driver. This finding is reassuring from a biological perspective it suggests that the bacterial population has not developed pan-resistance but it also underscores the clinical and policy urgency of addressing the selective pressures driving resistance to the most accessible and commonly used antibiotics. In conclusion, the low resistance to amikacin and meropenem documented among Kibra isolates is a rare point of reassurance in an otherwise concerning resistance profile.

Nitrofurantoin resistance in Kibra (8.4% overall, 0.0% among *E. coli* specifically) is one of the most consistent findings across the entire comparator literature assembled for this discussion. The study [27], reported a pooled sub-Saharan African *E. coli* nitrofurantoin resistance of 8.2%, essentially identical to the Kibra figure;(1) reported 9% nitrofurantoin resistance across seven Kenyan facilities including the tertiary County Hospital; and [22], reported 4% resistance in central South Africa. Even the Bhutanese comparator [28], found nitrofurantoin resistance of only 3.4%, reinforcing that nitrofurantoin's continued effectiveness against community uropathogens is a genuinely cross-regional and, on this evidence, near-global pattern, unlike the highly setting-dependent picture for cephalosporins and fluoroquinolones discussed above. This consistency provides one of the more confidently generalizable, policy-actionable conclusions to emerge from this dissertation.

Future Direction

Future studies should link individual-level community antibiotic-use behaviour directly to laboratory-confirmed resistance profiles from the same participants (rather than parallel samples, as in this study), to strengthen causal inference, following the linked-data approach demonstrated at multi-country scale by the HATUA Consortium [31]. Molecular characterization of resistance genes (e.g., blaCTX-M, blaTEM) among the ESBL-suspect isolates identified in this study would confirm the phenotypic findings and support genomic surveillance of AMR in Kibra; the substantially lower genotypically confirmed ESBL prevalence reported in rural Tanzania (23.0%; [21], relative to this study's phenotypic estimate (74.2%) illustrates why such confirmation matters for accurate cross-study comparison.

ETHICA APPROVAL:

Approval to conduct the study was secured from the Kenyatta University Ethics Review Committee (PKU/3323/140329) together with permission granted by the National Commission for Science, Technology

and Innovation (NACOSTI), license no. P/26/4190549 before any research activities commenced. All participants provided written informed consent prior to enrolment. For participants aged 15–17 years, written consent was obtained from a parent or legal guardian, in addition to the adolescent's written assent. To safeguard participant privacy, all data and biological specimens were assigned unique identification codes instead of personal identifiers. Any names recorded in the original laboratory documentation were replaced with anonymized study codes (e.g., KB-001) before data analysis and dissemination of the findings.

Informed Consent: Informed consent was obtained from all individual participants included in the study (Appendix 1)

Data Accessibility: Supplementary data is provided in the appendices attached.

Funding: The author(s) received no specific grant or external funding from any funding agency in the public, commercial, or not-for-profit sectors for the research, authorship, and/or publication of this article.

Disclosures: The authors declare that they have no potential, actual, or perceived conflicts of interest, financial or otherwise, relevant to the contents of this manuscript.

APPENDICES

Appendix 1: Informed Consent Form

Study title: Identification and Antibiotic Susceptibility Profiles of Uropathogenic Bacteria in Kibra Informal Settlement, Nairobi City County, Kenya

Participant ID: _____
I have read (or the information sheet has been read to me) the information about this study. I have had the chance to ask questions and they have been answered to my satisfaction. I understand that my participation is voluntary and that I can withdraw at any time.

I agree to:
i. Answer the questionnaire / take P art in the focus grouped discussion.
ii. Provide a mid-stream urine sample for laboratory analysis (survey/laboratory component only).
iii. Allow the research team to use my anonymized data for research and publications.

Name of participant: _____ Signature/thumbprint: _____ Date: __/__/____

Name of person obtaining consent: _____Signature: _____ Date: __/__/____

Witness (if participant illiterate): _____ Signature: _____ Date: __/__/____
(For 15-17 year olds: assent form plus parent/guardian consent was used.)

Appendix II

Anonymized Laboratory Dataset – Uropathogenic Isolates (n = 150)

The table below lists all 150 uropathogenic bacterial isolates included in the analytic dataset for this study, following anonymization of patient identifiers and exclusion of unidentified-organism and fungal (Candida spp.) reports from the original laboratory records. Antibiotic abbreviations follow standard CLSI nomenclature (e.g., AMP = ampicillin, SXT = trimethoprim/sulfamethoxazole, CRO = ceftriaxone, CIP = ciprofloxacin, NIT = nitrofurantoin, MEM = meropenem). A supplementary anonymised electronic workbook containing the full antimicrobial susceptibility panel (zone/MIC interpretations for all antibiotics tested per isolate) and the complete community-survey dataset was submitted alongside this dissertation.

Lab ID	Age	Sex	Organism	Antibiotics with resistance detected (abbreviated)	MDR
KB-001	9	F	*Escherichia coli*	AMP, AMC, SAM, TZP, CFZ, CXM, FOX, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-002	43	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, SXT	Yes
KB-003	39	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes

KB-004	36	F	*Escherichia coli*	AMP, SXT	No
KB-005	1	M	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, GEN, SXT	Yes
KB-006	30	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, GEN, CIP, SXT	Yes
KB-007	30	M	*Escherichia coli*	AMP, SAM, SXT	Yes
KB-008	50	M	*Escherichia coli*	AMP, AMC, SAM, TZP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP	Yes
KB-009	27	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-010	41	M	*Klebsiella pneumoniae*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP	Yes
KB-011	58	M	*Staphylococcus aureus*	SXT, VAN, TET	Yes
KB-012	2	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-013	33	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, SXT	Yes
KB-014	31	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, SXT	Yes
KB-015	34	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP	Yes
KB-016	34	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, SXT	Yes
KB-017	1	M	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, CIP, SXT	Yes
KB-018	50	F	*Klebsiella pneumoniae*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-019	31	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP	Yes
KB-020	43	M	*Klebsiella pneumoniae*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM	Yes
KB-021	49	M	*Klebsiella pneumoniae*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-022	23	M	*Enterococcus faecalis*	LVX, ERY, VAN, TET	Yes
KB-023	27	M	*Klebsiella pneumoniae*	AMP, SAM, SXT	Yes
KB-024	27	M	*Escherichia coli*	CIP, SXT	No
KB-025	18	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM	Yes
KB-026	37	F	*Proteus mirabilis*	AMP, NIT, SXT	Yes
KB-027	24	F	*Escherichia coli*	AMP, AMC, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, GEN, SXT	Yes
KB-028	22	M	*Escherichia coli*	AMP, AMC, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-029	28	M	*Proteus vulgaris*	AMP, CFZ, CXM, NIT	Yes
KB-030	41	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, GEN, CIP, SXT	Yes
KB-031	40	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, CIP	Yes

KB-032	33	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP	Yes
KB-033	45	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-034	21	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-035	37	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, GEN, CIP, SXT	Yes
KB-036	52	M	*Citrobacter freundii*	AMC, CFZ, CXM, FOX, CTX, CAZ, CRO, FEP, ATM, GEN, CIP, NIT, SXT	Yes
KB-037	24	F	*Escherichia coli*	AMP, SXT	No
KB-038	31	M	*Pseudomonas aeruginosa*	CFZ, CTX	No
KB-039	28	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-040	35	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-041	43	F	*Acinetobacter lwoffii*	CIP, SXT	No
KB-042	39	F	*Escherichia coli*	AMP, AMC, CFZ, CXM, FOX, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-043	24	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, CIP, SXT	Yes
KB-044	43	F	*Klebsiella oxytoca*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-045	27	F	*Enterococcus faecalis*	ERY, TET	No
KB-046	24	F	*Staphylococcus saprophyticus*	SXT, ERY	No
KB-047	23	M	*Enterococcus faecalis*	NIT, LVX, ERY, VAN, TET	Yes
KB-048	43	F	*Klebsiella oxytoca*	AMP	No
KB-049	45	F	*Citrobacter freundii*	AMC, CFZ, CXM, FOX, CTX, CRO, ATM, AMK, CIP, SXT	Yes
KB-050	41	M	*Klebsiella aerogenes*	AMC, CFZ, CXM, FOX, CTX, CRO, SXT	Yes
KB-051	32	F	*Klebsiella pneumoniae*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM	Yes
KB-052	34	F	*Proteus vulgaris*	AMP, CFZ, CXM, NIT	Yes
KB-053	27	F	*Klebsiella pneumoniae*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-054	32	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-055	27	F	*Enterobacter cloacae complex*	AMC, CFZ, CXM, FOX	Yes
KB-056	37	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-057	40	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, GEN, CIP, SXT	Yes
KB-058	50	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-059	50	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes

KB-060	21	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-061	39	M	*Pseudomonas aeruginosa*	CFZ, CTX	No
KB-062	27	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-063	50	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-064	26	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-065	81	M	*Citrobacter freundii*	AMC, CFZ, CXM, FOX, SXT	Yes
KB-066	24	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, GEN, CIP, SXT	Yes
KB-067	21	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-068	26	F	*Acinetobacter haemolyticus*	SAM, FEP	No
KB-069	24	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, CIP, SXT	Yes
KB-070	31	F	*Proteus hauseri*	AMP, CFZ, CXM, NIT	Yes
KB-071	42	M	*Citrobacter freundii*	AMC, CFZ, CXM, FOX, CTX, CRO, FEP, ATM, GEN, SXT	Yes
KB-072	1	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-073	39	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP	Yes
KB-074	23	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-075	33	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, GEN, SXT	Yes
KB-076	31	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-077	32	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-078	30	F	*Klebsiella pneumoniae*	AMP	No
KB-079	48	F	*Klebsiella pneumoniae*	AMP	No
KB-080	26	F	*Escherichia coli*	CFZ, CXM, CTX, CRO, FEP, ATM, CIP, SXT	Yes
KB-081	20	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, GEN, CIP, SXT	Yes
KB-082	72	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-083	28	M	*Pseudomonas aeruginosa*	CFZ, CTX, CAZ, GEN, CIP	Yes
KB-084	53	M	*Proteus mirabilis*	AMP, NIT, SXT	Yes
KB-085	37	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, SXT	Yes
KB-086	53	F	*Pseudomonas aeruginosa*	CFZ, CTX	No
KB-087	49	F	*Pseudomonas aeruginosa*	CFZ	No

KB-088	33	F	*Escherichia coli*	AMP, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-089	24	M	*Citrobacter freundii*	AMC, CFZ, CXM, FOX, SXT	Yes
KB-090	21	F	*Escherichia coli*	CAZ	No
KB-091	67	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-092	48	F	*Staphylococcus aureus*	SXT	No
KB-093	32	M	*Escherichia coli*	CFZ, FOX	No
KB-094	39	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-095	45	F	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, SXT	Yes
KB-096	78	M	*Escherichia coli*	AMP, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, CIP, SXT	Yes
KB-097	28	F	*Morganella morganii ssp morganii*	AMP, AMC, CFZ, CXM, NIT, SXT	Yes
KB-098	30	M	*Staphylococcus aureus*	SXT, VAN	No
KB-099	18	F	*Enterococcus faecalis*	GEN, LVX, ERY, TET, QUIN	Yes
KB-100	27	F	*Enterococcus faecalis*	QUIN	No
KB-101	41	F	*Escherichia coli*	AMP, AMC, SAM, CFZ, CXM, CTX, CAZ, CRO, FEP, ATM, SXT	Yes
KB-102	44	F	*Enterobacter cloacae complex*	AMC, CFZ, CXM, FOX	Yes
KB-103	41	F	*Enterococcus faecium*	AMP, CTX, FOX, CRO, CLI, SXT, ERY, GEN, OXA, PEN, PENI, TET	Yes
KB-104	41	F	*Enterococcus faecium*	AMP, CTX, FOX, CRO, CLI, SXT, ERY, GEN, OXA, PEN, PENI, TET	Yes
KB-105	61	F	*Enterococcus faecium*	AMP, CTX, FOX, CRO, CLI, SXT, ERY, GEN, OXA, PEN, PENI, TET	Yes
KB-106	22	M	*Staphylococcus aureus*	CTX	No
KB-107	42	M	*Staphylococcus aureus*	CTX	No
KB-108	62	M	*Staphylococcus aureus*	CTX	No
KB-109	22	M	*Staphylococcus aureus*	CTX	No
KB-110	46	M	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP, TZP	Yes
KB-111	22	M	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-112	62	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM	Yes
KB-113	34	M	*Klebsiella pneumoniae*	AMP, ATM, FEP, CTX, CAZ, CRO, CIP, TZP	Yes
KB-114	19	F	*Klebsiella aerogenes*	AMP, ATM, FEP, CAZ, CRO, CIP, TZP	Yes
KB-115	55	M	*Klebsiella oxytoca*	AMP, ATM, FEP, CTX, CAZ, CRO, CIP, TZP	Yes

KB-116	12	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-117	37	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-118	26	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-119	50	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM	Yes
KB-120	24	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CIP, TZP	Yes
KB-121	19	M	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-122	34	M	*Klebsiella pneumoniae*	AMP, ATM, FEP, CTX, CAZ, CRO, CIP, TZP	Yes
KB-123	29	F	*Klebsiella aerogenes*	AMP, ATM, FEP, CAZ, CRO, CIP, TZP	Yes
KB-124	95	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-125	106	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM	Yes
KB-126	79	F	*Klebsiella aerogenes*	AMP, ATM, FEP, CAZ, CRO, CIP, TZP	Yes
KB-127	56	M	*Klebsiella oxytoca*	AMP, ATM, FEP, CTX, CAZ, CRO, CIP, TZP	Yes
KB-128	13	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-129	38	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-130	66	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-131	51	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM	Yes
KB-132	23	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CIP, TZP	Yes
KB-133	16	M	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-134	76	M	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP, TZP	Yes
KB-135	55	M	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-136	63	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM	Yes
KB-137	63	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM	Yes
KB-138	27	M	*Klebsiella pneumoniae*	AMP, ATM, FEP, CTX, CAZ, CRO, CIP, TZP	Yes
KB-139	29	F	*Klebsiella aerogenes*	AMP, ATM, FEP, CAZ, CRO, CIP, TZP	Yes
KB-140	15	F	*Klebsiella pneumoniae*	ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-141	74	F	*Pseudomonas aeruginosa*	AMP, ATM, FEP, CAZ, CRO, CXM, CIP	Yes
KB-142	74	F	*Klebsiella oxytoca*	ATM, FEP, CAZ, CRO, CXM, CIP	Yes
KB-143	12	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CRO, CXM	Yes

KB-144	44	M	*Pseudomonas aeruginosa*	AMP, ATM, FEP, CTX, CAZ, CRO	Yes
KB-145	35	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO	Yes
KB-146	90	M	*Klebsiella pneumoniae*	AMK, ATM, FEP, CTX, CXM	Yes
KB-147	34	M	*Klebsiella pneumoniae*	AMP, ATM, FEP, CTX, CAZ, CXM, CIP	Yes
KB-148	87	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CRO, CXM	Yes
KB-149	43	F	*Escherichia coli*	AMP, ATM, FEP, CTX, CAZ, CRO, CXM, CIP	Yes
KB-150	74	F	*Pseudomonas aeruginosa*	AMP, ATM, FEP, CAZ, CRO, CXM, CIP	Yes

Appendix III

Resistance to Key Antibiotics by Organism (n resistant / n tested)

This table disaggregates the overall resistance figures presented by organism, for the seven antibiotics most central to empirical UTI treatment decisions in Kibra. A dash (-) indicates that fewer than one isolate of that organism was tested against the antibiotic in question. AMP = ampicillin, CRO = ceftriaxone, SXT = trimethoprim/sulfamethoxazole, CIP = ciprofloxacin, GEN = gentamicin, NIT = nitrofurantoin, MEM = meropenem.

Organism	**n**	**AMP**	**CRO**	**SXT**	**CIP**	**GEN**	**NIT**	**MEM**
Escherichia coli	86	82/85	80/86	52/61	50/86	9/86	0/61	0/86
Klebsiella pneumoniae	15	13/15	10/15	4/9	7/15	0/14	0/9	0/15
Pseudomonas aeruginosa	8	3/3	3/3	-	3/8	1/8	-	0/8
Staphylococcus aureus	7	0/4	-	3/7	-	0/6	0/3	-
Klebsiella aerogenes	5	4/4	5/5	1/1	4/5	0/5	0/1	0/5
Citrobacter freundii	5	-	3/5	5/5	2/5	2/5	1/5	0/5
Enterococcus faecalis	5	-	-	-	-	1/2	1/5	-
Klebsiella oxytoca	5	4/5	4/5	1/2	3/5	0/5	0/2	0/5
Enterococcus faecium	3	3/3	3/3	3/3	-	3/3	-	-
Proteus vulgaris	2	2/2	0/2	0/2	0/2	0/2	2/2	0/2
Proteus mirabilis	2	2/2	0/2	2/2	0/2	0/2	2/2	0/2
Enterobacter cloacae complex	2	-	0/2	0/2	0/2	0/2	0/2	0/2
Staphylococcus saprophyticus	1	-	-	1/1	-	0/1	0/1	-
Morganella morganii ssp morganii	1	1/1	0/1	1/1	0/1	0/1	1/1	0/1
Proteus hauseri	1	1/1	0/1	0/1	0/1	0/1	1/1	0/1
Acinetobacter haemolyticus	1	-	0/1	0/1	0/1	0/1	-	0/1
Acinetobacter lwoffii	1	-	0/1	1/1	1/1	0/1	-	0/1

Appendix IV

Community Survey Dataset – Variable Codebook (n = 100)

This codebook describes the 100-respondent community survey dataset (participant IDs CS-001 to CS-100).

Variable codebook

Variable	**Description / response categories**
participant_id	Unique anonymised respondent code (CS-001 to CS-100)
age, sex	Respondent age (years) and sex (Male/Female)
Education	None / Primary / Secondary / Tertiary
Occupation	Informal trade/business, Casual labourer, Unemployed, Formal employment, Student, Domestic work
hh_size	Household size (number of people)
Income	Monthly household income band (KES)
uti_symptoms_12m, episodes	UTI-like symptoms in past 12 months (Yes/No) and number of episodes
care_source	First source of care sought for most recent UTI episode
diagnostic_test	Whether a diagnostic urine test was performed (Yes/No/Don't know)

took_antibiotic	Took an antibiotic for the most recent episode (Yes/No)
source_ab	Source through which the antibiotic was obtained
completed_course, stop_reason	Whether the full course was completed, and reason if stopped early
bought_without_prescription	Frequency of ever buying antibiotics without a prescription
keep_leftover	Frequency of keeping leftover antibiotics for future use
know_bacterial, know_viral_wrong_belief	Knowledge items on antibiotic effectiveness against bacterial/viral illness
believe_stop_when_better_safe	Belief that stopping antibiotics once feeling better is safe
perceive_amr_serious	Perceived seriousness of antimicrobial resistance
disposal, share_antibiotics, use_in_animals	Disposal method, sharing behaviour, and use of antibiotics in animals
water_source, sanitation, animal_work	Household water source, sanitation type, and animal-related occupation

Appendix V
Full Demographic Frequency Distributions – Community Survey (n = 100)
Socio-demographic and household variables

Variable	Category	% (n=100)
Sex	Female	62.0
Sex	Male	38.0
Education	Secondary	37.0
Education	Primary	36.0
Education	Tertiary	18.0
Education	None	9.0
Occupation	Informal trade/business	29.0
Occupation	Casual labourer	24.0
Occupation	Student	13.0
Occupation	Unemployed	12.0
Occupation	Formal employment	11.0
Occupation	Domestic work	11.0
Monthly income (KES)	<10,000	53.0
Monthly income (KES)	10,000-25,000	24.0
Monthly income (KES)	25,000-50,000	14.0
Monthly income (KES)	>50,000	5.0
Monthly income (KES)	Prefer not to say	4.0

Appendix VI
Health-seeking and antibiotic-use variables

Variable	Category	% (n=100)
UTI symptoms, past 12 months	Yes	75.0
UTI symptoms, past 12 months	No	25.0
First source of care	Pharmacy/drug shop	53.0
First source of care	Public clinic	22.0
First source of care	Private clinic	13.0
First source of care	Self-care at home	10.0
First source of care	Traditional healer	2.0
Diagnostic test performed	No	60.0
Diagnostic test performed	Yes	31.0
Diagnostic test performed	Don't know	9.0
Took antibiotic	Yes	84.0
Took antibiotic	No	16.0
Antibiotic source	Bought from pharmacy/drug seller	49.0
Antibiotic source	Prescription from clinician	27.0
Antibiotic source	Given by friend/family	17.0
Antibiotic source	Leftover from previous illness	7.0
Completed full course	No	44.0

Completed full course	Yes	42.0
Completed full course	Don't remember	14.0
Reason stopped early	Symptoms improved	69.0
Reason stopped early	Ran out of money	19.0
Reason stopped early	Side effects	9.0
Reason stopped early	Other	3.0
Bought antibiotics without prescription	2-5 times	39.0
Bought antibiotics without prescription	>5 times	25.0
Bought antibiotics without prescription	Never	18.0
Bought antibiotics without prescription	Once	18.0
Keeps leftover antibiotics	Occasionally	35.0
Keeps leftover antibiotics	Often	29.0
Keeps leftover antibiotics	Never	25.0
Keeps leftover antibiotics	Always	11.0

REFERENCES

1. Kiiru S, Maina J, Katana J, Mwankii J, Kiiru J. Bacterial etiology of urinary tract infections in Kenyan health facilities and resistance towards commonly used antibiotics. medRxiv. 2023.
2. Zeng Z, Zhan J, Zhang L, Chen X, Cheng J. Global, regional, and national burden of urinary tract infections from 1990 to 2019: A systematic analysis for the Global Burden of Disease Study 2019. Frontiers in Public Health. 2022;10:1025063.
3. Magale HM, Nyerere A, Mwangi J. Antimicrobial resistance among uropathogens at Kenyatta National Hospital, Kenya. East African Medical Journal. 2015;92(5):245–52.
4. Omulo S, Lofgren ET, Lockwood S, Thumbi SM, Bigogo G, Ouma A, et al. Carriage of antimicrobial-resistant bacteria in a high-density informal settlement in Kenya is associated with environmental risk factors. Antimicrobial Resistance & Infection Control. 2021;10:18. doi:10.1186/s13756-021-00886-y
5. Juma J, Ochieng JB, Saha D, Feasey N, Kariuki S. Antimicrobial resistance among enteric bacterial pathogens recovered from residents of a Nairobi informal settlement between 2018 and 2020. Journal of Global Antimicrobial Resistance. 2025;40:12–8.
6. Mundalo P, Kariuki S, Omulo S. Diarrheagenic Escherichia coli recovered from street food and water sources in Mukuru informal settlement, Nairobi. Foodborne Pathogens and Disease. 2026;23(4):278–86.
7. Okeke IN, Laxminarayan R, Bhutta ZA. Antimicrobial resistance: One Health approach. In: Disease Control Priorities. 3rd ed. World Bank; 2020.
8. Saisi HN. Antimicrobial resistance patterns in extended-spectrum beta-lactamase producing Klebsiella pneumoniae isolates from children in Mukuru slums, Nairobi, Kenya [(Master's thesis).]. Jomo Kenyatta University of Agriculture and Technology; 2021.
9. Mundalo S, Ntabo R, Kering K, Too R, Kariuki K, Imoli D, et al. Molecular detection and antibiotic resistance of diarrheagenic Escherichia coli from street food and water in Mukuru slums, Nairobi County. PLOS ONE. 2026;21(1):0340081. doi:10.1371/journal.pone.0340081
10. Karimi KJ, Ahmad A, Duse A, Mwanthi M, Ayah R. Prevalence of antibiotic use and disposal at household level in informal settlements of Kisumu, Kenya. International Journal of Environmental Research and Public Health. 2022;20(1):287. doi:10.3390/ijerph20010287
11. U.N.-Habitat. World Cities Report 2020: The Value of Sustainable Urbanization. United Nations Human Settlements Programme; 2020.
12. Kenya National Action Plan on Antimicrobial Resistance (NAP-AMR) 2023–2027. 2023.
13. Auta A, Hadi MA, Oga E, Adewuyi EO, Abdu-Aguye SN, Adeloye D, et al. Global access to antibiotics without prescription in community pharmacies: A systematic review and meta-analysis. Journal of Infection. 2019;78(1):8–18. doi:10.1016/j.jinf.2018.07.001
14. Ndaki PM, Mushi MF, Mwanga JR, Konje ET, Ntinginya NE, Mmbaga BT, et al. Dispensing antibiotics without prescription at the community pharmacies and accredited drug dispensing outlets in Tanzania: A cross-sectional study. Antibiotics. 2021;10(8):1025. doi:10.3390/antibiotics10081025
15. Vicar EK, Walana W, Mbabila A, Darko GK, Opare-Asamoah K, Majeed SF, et al. Drivers of household antibiotic use in urban informal settlements in Northern Ghana: Implications for antimicrobial resistance control. Health Science Reports. 2023;6(7):1388. doi:10.1002/hsr2.1388
16. One Health Trust & World Health Organization. Ministry of Health; 2023.
17. Vicar EK, Yakubu I, Ndanu TA. Household antibiotic use in informal settlements in the Tamale metropolis, Northern Ghana. Journal of Global Antimicrobial Resistance. 2023;35:78–85.

18. Obhai G. From self-medication to antimicrobial resistance: Socioeconomic realities and public health implications in Kibera. Nairobi: AMR Insights; 2025.
19. Flores-Mireles AL, Walker JN, Caparon M, Hultgren SJ. Urinary tract infections: Epidemiology, mechanisms of infection and treatment options. Nature Reviews Microbiology. 2015;13(5):269–84. doi:10.1038/nrmicro3432
20. Zeng Z, Zhan J, Zhang K, Chen H, Cheng S. Global, regional, and national burden of urinary tract infections from 1990 to 2019: An analysis of the Global Burden of Disease Study 2019. World Journal of Urology. 2022;40(3):755–63. doi:10.1007/s00345-021-03913-0
21. Macha J, Mushi MF, Hokororo A, Mshana SE. Antimicrobial resistance patterns of uropathogens in Ifakara, Tanzania: A cross-sectional study. BMC Infectious Diseases. 2025;25:156.
22. Fourie CM, Makhathini KB, Moodley P, Govender S. Community-acquired urinary tract infections in central South Africa: Prevalence, antimicrobial resistance and associated factors. Southern African Journal of Infectious Diseases. 2021;36(1):256.
23. Odongo CO, Niyibizi JB, Bizimana E, Nshimiyimana I, Ruhangaza D. Diversity and antibiotic sensitivity of uropathogens from a regional hospital in Rwanda. African Health Sciences. 2024;24(3):21–9.
24. Tesfay T, Ghebrehiwet S, Andemichael G, Tekle Y. Patterns and trends of antimicrobial resistance in bacterial isolates from urine cultures in Eritrea, 2010–2021: A secondary dataset analysis. BMC Infectious Diseases. 2026;26:303.
25. Magale HI, Kassim IA, Odera SA, Omolo MJ, Jaoko WG, Jolly PE. Antibiotic susceptibility of organisms causing urinary tract infection in patients presenting at Kenyatta National Hospital, Nairobi. East African Medical Journal. 2015;92(7):333–7.
26. Dayie NTKD, Egyir B, Amoa-Owusu F, Owusu-Nyantakyi C, Adu B, Kotey FCN, et al. Genome sequences of extended-spectrum beta-lactamase-producing Escherichia coli recovered from mid-stream urine samples in Accra, Ghana. Microorganisms. 2024;12(6):1139.
27. Williams-Walker A, Lammie L, Moore CE. Prevalence of antimicrobial resistance in acute community-acquired urinary tract infections in Sub-Saharan Africa: A systematic review [Conference abstract P57. JAC-Antimicrobial Resistance. 2025;7(Suppl. 4). doi:10.1093/jacamr/dlaf230.064
28. Adeep M, Nima T, Kezang W, Tshokey T. A retrospective analysis of the etiologic agents and antibiotic susceptibility pattern of uropathogens isolated in the Jigme Dorji Wangchuck National Referral Hospital, Thimphu, Bhutan. BMC Research Notes. 2016;9:42. doi:10.1186/s13104-015-1728-1
29. Yonga P, Pulcini C, Skov R, Paño-Pardo JR, Schouten JA. The case for the Access, Watch, and Reserve (AWaRe) universal guidelines for antibiotic use. Clinical Microbiology and Infection. 2024;30(7):848–9.
30. Kimera ZI, Matee MI. Antimicrobial resistance epidemiology, economic impact and policy in sub-Saharan Africa between 2015 and 2025. Tropical Medicine and International Health. 2026;31(2):145–62.
31. Sado K, Keenan K, Manataki A, Kesby M, Mushi MF, Mshana SE, et al. Treatment seeking behaviours, antibiotic use and relationships to multi-drug resistance: A study of urinary tract infection patients in Kenya, Tanzania and Uganda. PLOS Global Public Health. 2024;4(2):0002709. doi:10.1371/journal.pgph.0002709.

Cite This Article: Ahatha, A. M., Menza, N., & Kimani, H. (2026). Identification and Antibiotic Susceptibility Profiles of Uropathogenic Bacteria in Kibra Informal Settlement, Nairobi City County, Kenya. *EAS J Parasitol Infect Dis, 8*(4), 99-117. https://doi.org/10.36349/easjpid.2026.v08i04.001