Eurasia Specialized Veterinary Publication

International Journal of Veterinary Research and Allied Science

ISSN:3062-357X

2021, Volume 1, Issue 2, Page No: 83-92

Available online at: www.esvpub.com/

Drug Resistance Patterns and Antimicrobial-Resistance Genes of Escherichia coli in Cats and Their Drinking Water

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ABSTRACT

The growing threat of antimicrobial resistance (AMR) is driven by the exchange of resistant bacteria and resistance genes among humans, animals, and the environment. This research explored the occurrence and characteristics of resistant Escherichia coli in domestic cats by examining isolates from both their feces and drinking water sources. A total of 104 samples were analyzed (52 fecal and 52 water samples). E. coli was recovered from every fecal sample and from 23 of the water samples (44.2%). All isolates remained fully sensitive to amikacin and imipenem, whereas resistance to clindamycin was most frequent. The predominant multidrug resistance patterns involved β-lactam antibiotics combined with other classes, including aminoglycosides, fluoroquinolones, sulfonamides, macrolides, and occasionally carbapenems. Resistance to quinolones showed very strong mutual correlations ($r > 0.8$, $p < 0.01$), while azithromycin and trimethoprim-sulfamethoxazole displayed a moderate association ($r = 0.5253$, $p < 0.01$). No carbapenemase-encoding genes were found. Among ESBL genes, blaTEM was the most common, followed by blaSHV and blaCTX-M. Phylogenetic analysis revealed that phylogroup B2 was the most prevalent, particularly in isolates from feces. Resistant E. coli, especially strains belonging to phylogroup B2 and carrying the blaTEM gene, are present in both the intestinal tract of pet cats and their drinking water, suggesting that household cats and their immediate environment may serve as reservoirs for antimicrobial resistance.

Keywords: Water, Antimicrobial resistance, Contamination, Extended-spectrum β-lactamase, E. coli

Received: 06 May 2021
Revised: 19 August 2021
Accepted: 21 August 2021

How to Cite This Article: Romani M, De Luca S. Drug Resistance Patterns and Antimicrobial-Resistance Genes of Escherichia coli in Cats and Their Drinking Water. Int J Vet Res Allied Sci. 2021;1(2):83-92. https://doi.org/10.51847/9WuM9qa95j

Background

Escherichia coli strains are classified into eight phylogenetic groups (A, B1, B2, C, D, E, F, and clade I) according to the presence of specific genomic pathogenicity markers [1]. The most commonly encountered groups are A, B1, B2, and D [1, 2]. Commensal, non-pathogenic E. coli typically belong to groups A and B1, whereas potentially pathogenic or extraintestinal pathogenic strains are more often associated with the other groups [2].

Extended-spectrum β-lactamase (ESBL)-producing E. coli have been widely documented in both healthy and sick animals [3–8]. These resistance genes (particularly blaTEM, blaSHV, and blaCTX-M) are frequently detected in commensal and clinical isolates from cats and their human companions, reflecting bidirectional plasmid-mediated transfer between pets, owners, and the shared environment [8–11]. The enzymes encoded by these genes inactivate β-lactam antibiotics, thereby contributing to antimicrobial resistance (AMR) [12].

Antimicrobial resistance now constitutes a major public health challenge affecting both human and veterinary medicine [13–16]. Overuse and misuse of antibiotics, combined with limited awareness, have accelerated the

emergence and dissemination of resistant bacteria [13–16]. High levels of resistance in E. coli have been reported in companion animals and people alike [8, 17, 18]. The close physical contact between cats and owners, along with shared living spaces, facilitates the exchange of resistant bacteria and mobile genetic elements through direct contact or environmental contamination [14, 19]. Previous work has shown that drinking water bowls of cats, especially long-haired breeds, are often contaminated with coliform bacteria [20]. Despite this, data remain scarce regarding the resistance profiles of E. coli present in cat feces compared with those in their drinking water. Comparing these two reservoirs can reveal differences in resistance prevalence and help pinpoint potential transmission routes. A clearer understanding of AMR dynamics in household cats may guide more prudent antimicrobial use in veterinary and human health settings.

The present study aimed to: (i) assess the occurrence and degree of association of antimicrobial-resistant E. coli in cat feces and drinking water, (ii) characterize resistance patterns and correlations between bactericidal and bacteriostatic agents, and (iii) determine the phylogenetic groups of isolates as well as the presence of carbapenemase and ESBL-encoding genes.

Methods

Animals and sampling

Fifty-two client-owned cats presenting for routine care at the Kasetsart University Veterinary Teaching Hospital (Bangkok, Thailand) were enrolled. Median age was 4.0 years (range 0.7–15.9 years); the group comprised 17 females and 35 males. Breeds included Domestic Shorthair (n = 38), Persian (n = 8), British Shorthair (n = 3), and Scottish Fold (n = 3). The study was approved by the Kasetsart University Institutional Animal Care and Use Committee (ACKU62-VET-030) and the National Research Council of Thailand.

Fresh fecal samples were collected by veterinary staff using rectal swabs, placed immediately into sterile tubes, and delivered to the microbiology laboratory within 15 minutes. Simultaneously, owners collected approximately 50 mL of water from the cat's home drinking bowl using a sterile syringe (Nipro Corporation, Thailand) and a disinfecting cap (3M Curos, USA). Water samples were kept on ice during transport to the laboratory.

Bacterial isolation and identification

Fecal swabs were directly streaked onto MacConkey agar and incubated aerobically at 37 °C for 18–24 hours. Lactose-fermenting colonies were subcultured and subjected to standard biochemical confirmation (triple sugar iron agar, indole, methyl red, Voges-Proskauer, and citrate utilization—IMViC). Isolates showing the typical E. coli pattern (++--) together with indole positivity and citrate negativity were confirmed as E. coli.

For water samples, 1 mL was spread onto MacConkey agar and incubated at 37 °C for 24 hours. Suspected E. coli colonies were confirmed using the same biochemical panel as for fecal isolates.

Antimicrobial susceptibility testing

Antibiotic susceptibility was assessed by the Kirby-Bauer disk diffusion method using 14 antimicrobial agents: amikacin (30 µg), gentamicin (10 µg), imipenem (10 µg), meropenem (10 µg), ceftriaxone (30 µg), cephalexin (30 µg), amoxicillin (10 µg), amoxicillin-clavulanic acid (20/10 µg), ciprofloxacin (5 µg), enrofloxacin (5 µg), norfloxacin (10 µg), clindamycin (2 µg), azithromycin (15 µg), and sulfamethoxazole-trimethoprim (25 µg) (Mastdiscs® AST, Mast Group Ltd., UK). Results were interpreted according to the latest Clinical and Laboratory Standards Institute (CLSI) guidelines [21], with E. coli ATCC 25922 serving as the quality-control strain.

Pure E. coli cultures were adjusted to 0.5 McFarland turbidity, lawn-inoculated onto Mueller-Hinton agar, and incubated at 37 °C for 16–20 hours after placement of antibiotic disks. Zone diameters were measured and categorized as susceptible, intermediate, or resistant per CLSI criteria.

Detection of β-lactamase genes and phylogenetic grouping

Genomic DNA was extracted from each E. coli isolate using a commercial kit (GF-1 Bacterial DNA Extraction Kit, Vivantis, Malaysia). Multiplex PCR was performed to screen for carbapenemase genes (blaIMP, blaKPC, blaNDM, blaOXA-48-like) and ESBL genes (blaTEM, blaSHV, blaCTX-M) following previously published protocols [22, 23]. Thermal cycling conditions were: 95 °C for 3 min; 30 cycles of 95 °C for 5 s, 56 °C for 30 s, and 72 °C for 45 s; final extension at 72 °C for 5 min.

Phylogenetic grouping was carried out using the updated Clermont multiplex PCR method [24, 25]. Amplification conditions consisted of initial denaturation at 94 °C for 4 min, followed by 30 cycles with varying annealing temperatures (57 °C for group E, 59 °C for groups A, B, C, D, F; 63 °C for group B2), and a final extension at 72 °C for 5 min. PCR products were separated by electrophoresis on 2% agarose gels in 0.5× TBE buffer, stained with ethidium bromide, and visualized under UV light. Band sizes were compared against a 100-bp DNA ladder (GeneRuler 100 bp Plus, Thermo Fisher Scientific, USA).

Statistical analysis

Data were analyzed using GraphPad Prism 6.0 (GraphPad Software, USA) and STATA 12 (StataCorp, USA). Resistance prevalence was expressed as percentages. Differences in resistance between fecal and water isolates were evaluated with Fisher's exact test. Pairwise correlations of resistance profiles among bactericidal and bacteriostatic agents were calculated; correlation strength was classified as negligible (0.00–0.09), weak (0.10–0.39), moderate (0.40–0.69), strong (0.70–0.89), or very strong (0.90–1.00) [26]. Statistical significance was set at $p < 0.05$.

Results

E. coli was recovered from all 52 fecal samples (100%) and from 23 of 52 drinking water samples (44.2%).

Fecal and water isolates exhibited varied resistance profiles across antibiotic classes **(Table 1)**. No resistance was observed to amikacin, and resistance to gentamicin was rare (7.7% fecal, 4.4% water). Carbapenem resistance was almost absent; only one fecal isolate was resistant to meropenem, and none were resistant to imipenem. High-level resistance to clindamycin was recorded in both sources (76.9% fecal, 73.9% water). Amoxicillin resistance was common, particularly in water isolates (56.5% vs. 38.5% in feces), while resistance to amoxicillin-clavulanic acid was significantly associated between feces and water ($p = 0.0038$); no other drugs showed such an association ($p \geq 0.05$).

Overall susceptibility (no resistance to any tested agent) was higher among fecal isolates (46.2%) than water isolates (17.4%) **(Table 2)**. The most frequent resistance phenotype in both sample types involved β-lactam antibiotics. Fecal isolates often displayed multidrug resistance combining β-lactams with one, two, or three additional drug classes, whereas water isolates typically showed resistance to β-lactams plus one or two other classes.

Table 1. Prevalence of antimicrobial resistance patterns in E. coli isolated from feces and drinking water of 52 cats

Group of antimicrobials	Antimicrobial class	Antimicrobial name	E. coli from feces (n=52)		E. coli from cat's drinking water (n=23)		p-value
			No. of isolates tested	Resistant (%)	No. of isolates tested	Resistant (%)	
Bactericidal agents	**Aminoglycosides**	Amikacin	52	0 (0%)	23	0 (0%)	-
		Gentamicin	52	4 (7.7%)	23	1 (4.4%)	0.5924
	Carbapenems	Imipenem	52	0 (0%)	23	0 (0%)	-
		Meropenem	52	1 (1.9%)	23	0 (0%)	0.5032
	Cephalosporins	Cephalexin	52	9 (17.3%)	23	7 (30.4%)	0.2007
		Ceftriaxone	52	4 (7.7%)	23	1 (4.4%)	0.5924
	Penicillins	Amoxicillin	52	19 (36.5%)	23	13 (56.5%)	0.1463
		Amoxicillin + clavulanic acid	52	3 (5.8%)	23	7 (30.4%)	**0.0038**
	Quinolones	Ciprofloxacin	52	9 (17.3%)	23	3 (13.0%)	0.6423
		Enrofloxacin	52	9 (17.3%)	23	2 (8.7%)	0.3310
		Norfloxacin	52	9 (17.3%)	23	2 (8.7%)	0.3310
Bacteriostatic agents	**Lincomycins**	Clindamycin	52	40 (76.9%)	23	17 (73.9%)	0.7784
	Macrolides	Azithromycin	52	4 (7.7%)	23	1 (4.4%)	0.5924

	Sulfonamides	Sulfamethoxazole + trimethoprim	52	7 (13.5%)	23	1 (4.4%)	0.2384

Table 2. Antimicrobial resistance patterns of *E. Coli* from cat feces and cat drinking water

Class of antimicrobial agents	**Resistance patterns**	**Cat drinking water**		**Cat feces**	
		No. of isolates (%)	**95% CI**	**No. of isolates (%)**	**95% CI**
0	-	4 (17.4)	5.0-38.8	24 (46.2)	32.2–60.5
1	β-lactams	13 (56.5)	34.5–76.8	14 (26.9)	15.6–41.0
2	β-lactams + quinolone β-lactams + sulfonamide β-lactams + aminoglycoside β-lactams + macrolide β-lactams + carbapenem	2 (8.7) 1 (4.3) - 1 (4.3) -	1.1–28.1 0.1–22.0 - 0.1–22.0 -	3 (5.8) 1 (1.9) 1 (1.9) 1 (1.9) 1 (1.9)	1.2–16.0 0.1–10.3 0.1–10.3 0.1–10.3 0.1–10.3
3	β-lactams + quinolone + aminoglycoside β-lactams + sulfonamides + macrolide	1 (4.3) 1 (4.3)	0.1–22.0 0.1–22.0	1 (1.9) 1 (1.9)	0.1–10.3 0.1–10.3
4	β-lactams + quinolone + sulfonamide + aminoglycoside β-lactams + quinolone + sulfonamide + macrolide	- -		2 (3.9) 1 (1.9)	0.5–13.2 0.1–10.3
5	β-lactams quinolone + sulfonamide + aminoglycoside + macrolide	-		2 (3.9)	0.5–13.2

Analysis of pairwise correlations for bactericidal antibiotics demonstrated a very strong positive association among the quinolones (ciprofloxacin, enrofloxacin, and norfloxacin), with correlation coefficients exceeding 0.8 ($p < 0.01$; **Table 3**). Cephalosporins showed a moderate level of correlation ($r = 0.5586$, $p < 0.05$). Moderate correlations ($r = 0.4–0.6$, $p < 0.05$) were also observed between gentamicin and each of the tested quinolones, as well as between amoxicillin-clavulanic acid and cephalexin.

Among bacteriostatic agents, sensitivity patterns revealed a moderate positive correlation between azithromycin and sulfamethoxazole-trimethoprim ($r = 0.5253$, $p < 0.01$; **Table 4**).

Table 3. Pairwise correlation of antimicrobial resistance among bactericidal agents

Antimicrobial agents	**Amoxicillin**	**Amoxicillin + clavulanic acid**	**Ceftriaxone**	**Cephalexin**	**Ciprofloxacin**	**Enrofloxacin**	**Gentamycin**	**Meropenem**	**Norfloxacin**
Amoxicillin	—	0.2480*	0.2991**	0.2687*	0.3523	0.3459**	0.2418*	0.1024	0.3316**
Amoxicillin + clavulanic acid	0.2480*	—	0.2696*	0.5262**	0.2382*	0.1577	0.0488	-0.0545	0.1664
Ceftriaxone	0.2991**	0.2696*	—	0.5586**	0.3603**	0.3756**	0.3624**	-0.0395	0.3829**
Cephalexin	0.2687*	0.5262**	0.5586**	—	0.2844*	0.2232	0.2358*	-0.0719	0.2349*
Ciproflox acin	0.3523	0.2382*	0.3603**	0.2844*	—	0.9442**	0.4549**	-0.0557	0.9558**
Enrofloxacin	0.3459**	0.1577	0.3756**	0.2232	0.9442**	—	0.4731**	-0.0551	0.9886**
Gentamycin	0.2418*	0.0488	0.3624**	0.2358*	0.4549**	0.4731**	—	-0.0429	0.4813**
Meropene	0.1024	-0.0545	-0.0395	-0.0719	-0.0557	-0.0551	-0.0429	—	-0.0532

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Norfloxacin	0.3316**	0.1664	0.3829**	0.2349*	0.9558**	0.9886**	0.4813**	-0.0532	—

*p < 0.05, **p < 0.01

Table 4. Pairwise correlation of antimicrobial resistance among bacteriostatic agents

Antimicrobial agents	Sulfamethoxazole + trimethoprim	Azithromycin
Clindamycin	0.0476	0.0444
Azithromycin	0.5253**	
Sulfamethoxazole + trimethoprim		0.5253**

**$p < 0.01$

The investigation did not detect any carbapenemase genes among the E. coli isolates examined. According to the data presented in **Table 5**, the ESBL-producing strains showed a clear predominance of blaTEM, which appeared in 88.7% of the isolates. Combinations of resistance determinants were far less common: blaTEM + blaCTX−M occurred in 8.5%, blaTEM + blaSHV in 1.4%, and the triple profile blaTEM + blaSHV + blaCTX−M was likewise observed in 1.4% of isolates. Only seven isolates (9.9%)—specifically six from fecal material and one from drinking water—carried blaCTX−M, a gene associated with the hydrolysis of cephalosporins **(Table 5)**.
Phylogenetic classification revealed that phylogroup B2 was the most frequent overall (42.3%), with the majority originating from fecal samples (32.4%). The next most common cluster was phylogroup B1 (15.5 percent), followed by those categorized as unknown (14.1 percent), F (9.8 percent), A or C (7.0 percent), E or clade I (7.0 percent), and D or E (4.2 percent). Isolates within phylogroup B2 predominantly harbored blaTEM. Notably, a single fecal isolate (1.4%) belonging to phylogroup D or E contained the full set of three ESBL genes—blaTEM, blaSHV, and blaCTX−M. The detailed allocation of bla genes across the different phylogroups is provided in **Table 5**.

Table 5. Overview of the occurrence of extended-spectrum β-lactamase genes (blaTEM, blaSHV and blaCTX−M) identified in E. coli isolated from cat feces (n = 54) and drinking water samples (n = 17)

Clermont phylogroup	bla_{TEM}, bla_{SHV}, bla_{CTX-M} (*n*; %)	bla_{TEM}, bla_{CTX-M} (*n*; %)	bla_{TEM}+, bla_{SHV} (*n*; %)	bla_{TEM} (*n*; %)	Isolation source (*n*; %)
A or C	N/A	1 (25.0%)	N/A	3 (75.0%)	Feces (*n* = 4; 5.6%)
	N/A	N/A	N/A	1 (100.0%)	Water (*n* = 1; 1.4%)
B1	N/A	N/A	N/A	7 (100.0%)	Feces (*n* = 7; 9.9%)
	N/A	N/A	N/A	4 (100.0%)	Water (*n* = 4; 5.6%)
B2	N/A	1 (4.3%)	N/A	22 (95.7%)	Feces (*n* = 23; 32.4%)
	N/A	1 (14.3%)	1 (14.3%)	5 (71.4%)	Water (*n* = 7; 9.9%)
D or E	1 (33.3%)	N/A	N/A	2 (66.7%)	Feces (*n* = 3; 4.2%)
	N/A	N/A	N/A	N/A	Water (*n* = 0; 0%)
E or Clade I	N/A	N/A	N/A	3 (100.0%)	Feces (*n* = 3; 4.2%)
	N/A	N/A	N/A	2 (100.0%)	Water (*n* = 2; 2.8%)
F	N/A	1 (20.0%)	N/A	4 (80.0%)	Feces (*n* = 5; 7.0%)
	N/A	N/A	N/A	2 (100.0%)	Water (*n* = 2; 2.8%)
Unknown	N/A	2 (22.2%)	N/A	7 (77.8%)	Feces (*n* = 9; 12.7%)
	N/A	N/A	N/A	1 (100.0%)	Water (*n* = 1; 1.4%)
Total	1 (1.4%)	6 (8.5%)	1 (1.4%)	63 (88.7%)	71 (100%)

N/A = Not available

Discussion

This study examined the prevalence of antimicrobial resistance (AMR) in Escherichia coli isolates obtained from cat feces and environmental sources, specifically cat drinking water. The isolates demonstrated the highest susceptibility to aminoglycosides and carbapenems, while exhibiting the greatest resistance to lincomycin. A significant association in resistance to amoxicillin + clavulanic acid was observed between fecal and drinking-water isolates. The majority of E. coli isolates from both sources displayed resistance patterns to β-lactam antibiotics. The β-lactamase genes blaTEM, blaSHV, and blaCTX-M were detected in isolates from both feces and drinking water. Phylogroup B2 emerged as the most prevalent phylogroup. These results provide critical insights into AMR patterns in E. coli associated with cats and offer practical guidance for selecting appropriate antimicrobial therapy in feline bacterial infections.

Both fecal and environmental E. coli isolates exhibited elevated resistance to clindamycin. The marked resistance to cephalosporins and penicillins can likely be explained by the widespread use of penicillins in human medicine [27], combined with cross-resistance in strains harboring β-lactamase genes that hydrolyze the β-lactam ring of these antibiotic classes. Overall, fecal E. coli isolates showed higher resistance levels than their environmental counterparts, with the exceptions of cephalexin, amoxicillin, and amoxicillin + clavulanic acid. A notable association of resistance to amoxicillin + clavulanic acid was identified between fecal and water isolates. These observations highlight potential zoonotic and household transmission risks of AMR between cats and humans sharing the same environment.

Multidrug resistance, including simultaneous resistance to β-lactams combined with aminoglycosides, quinolones, sulfonamides, macrolides, or carbapenems, was detected in E. coli from both feces and drinking water. Certain isolates exhibited complex resistance profiles spanning multiple antimicrobial classes, posing significant therapeutic challenges. Interestingly, fecal isolates more frequently showed resistance to one or two antimicrobial classes, whereas environmental isolates more commonly displayed resistance to three to five classes. This difference underscores the need for tailored management strategies to mitigate AMR in both cats and their immediate surroundings.

Pairwise correlation analysis revealed a spectrum of associations among antimicrobial agents. Very strong positive correlations suggest shared resistance mechanisms, similar modes of action, or common pathways of resistance development. Weaker or absent correlations indicate distinct resistance profiles. Moderate to very strong correlations within the same antimicrobial category point to predictable co-resistance patterns, emphasizing the importance of avoiding drugs from the same class to slow further resistance emergence. Notably, meropenem showed no significant correlations with any other agent, reflecting its unique sensitivity profile. These findings have direct implications for treatment selection, optimization of therapeutic success, and the urgent need for culture and antimicrobial susceptibility testing prior to initiating therapy.

The rising prevalence of antimicrobial-resistant E. coli, including strains carrying resistance genes, in humans, animals, and the environment represents a major global public health threat. The detection of extended-spectrum β-lactamase (ESBL)-producing E. coli in cats and their environment raises concerns about the dissemination of resistance genes. Previous studies have documented β-lactamase genes in feline populations [8, 10, 11]. AMR E. coli can colonize healthy animals [6, 7], and ESBL genes in commensal strains may transfer horizontally to pathogenic bacteria, potentially reaching humans via fecal-oral routes [9, 28, 29]. In the present study, blaTEM, blaSHV, and blaCTX-M genes were identified, with blaTEM being the most common. A higher prevalence of blaCTX-M was noted in fecal isolates, suggesting a heightened risk of transmission from cats to the environment and to humans. No carbapenemase genes were detected, consistent with the very low meropenem resistance observed in only one isolate, likely due to restricted clinical use of carbapenems [30]. Consequently, carbapenems should remain reserved as last-line agents rather than empirical choices.

E. coli isolates from cats presenting to a veterinary hospital exhibited widespread resistance to most tested antimicrobials, confirming the high burden of AMR and ESBL-producing strains in this population and underscoring risks to both animal and public health. Resistant bacteria and their genes can spread from cats to humans through direct contact (e.g., petting) or indirectly via fomites such as litter boxes and water bowls (Fig. 1). Factors including antimicrobial exposure, illness, stress, aging, diet, and lifestyle influence gut microbiota composition [31], and dysbiosis has been linked to inflammatory bowel disease and difficult-to-treat infections caused by resistant organisms [31]. The recent U.S. CDC-identified outbreak of E. coli O157:H7 linked to contaminated slivered onions in a fast-food chain [32] further illustrates the broader food-safety implications. Given that companion animals, particularly cats, can act as reservoirs for multidrug-resistant enteropathogenic E. coli [33], a One Health approach is essential. Practical preventive measures include thorough hand washing after

handling cats, cleaning litter, or feeding, as well as caution regarding pet licks or close contact for immunocompromised individuals, especially since E. coli has been isolated from cats with gingivitis [34].

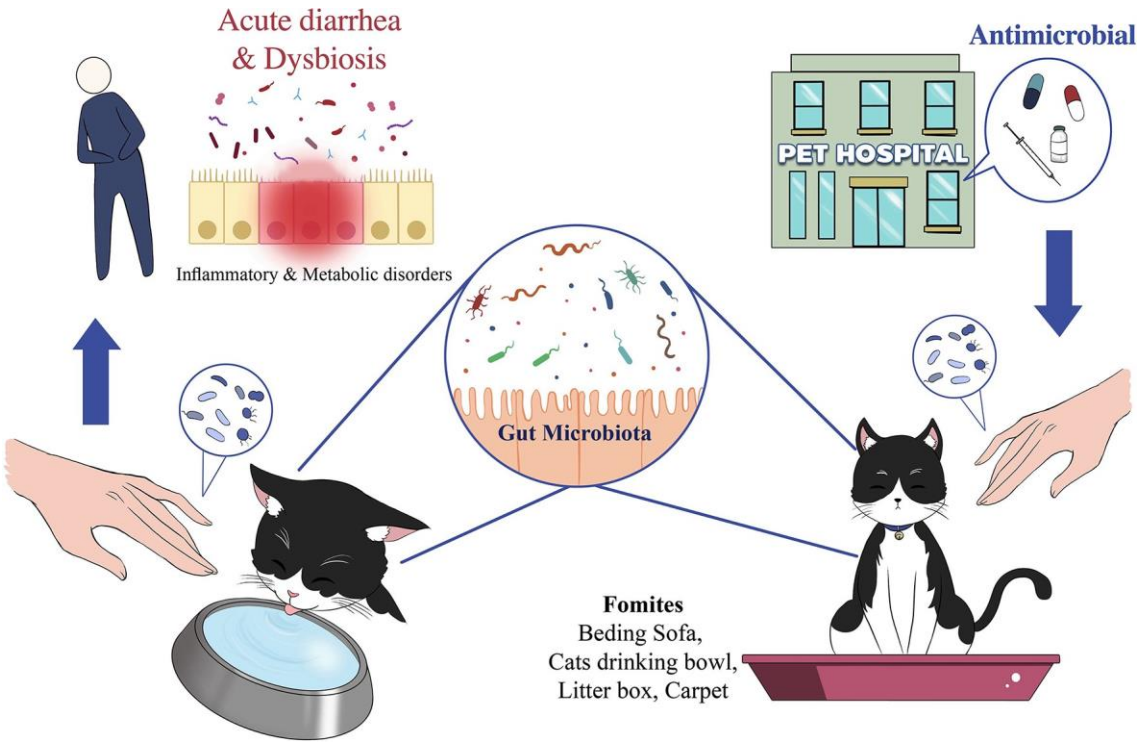

Figure 1. Horizontal transmission of microbes and antimicrobial resistance genes from cats to humans occurs via direct contact (e.g., petting) and indirect contact through fomites (e.g., water bowls, litter boxes). Antimicrobial resistance genes carried by the gut microbiota can colonize both cats and humans. Occasionally, fecal-oral transmission of enteropathogenic E. coli may cause acute diarrhea in humans. Furthermore, overgrowth of resistant E. coli is linked to gut dysbiosis, which can trigger intestinal inflammation and contribute to the development or progression of various gastrointestinal disorders

Phylogroups A and B1, which primarily consist of commensal E. coli strains, are most frequently detected in humans, whereas phylogroup B2 predominates in herbivorous and omnivorous mammals [35]. Phylogroup E is typically associated with cattle [36]. In the present study, nearly half of the E. coli isolates, especially those from fecal samples, belonged to phylogroup B2, suggesting increased pathogenic potential in feline-derived strains. Close human–pet interactions facilitate the bidirectional exchange of pathogens and AMR genes [7, 37]. To mitigate these risks, robust antimicrobial stewardship programs that promote prudent prescribing in both human and veterinary medicine are essential [38]. Strict infection prevention and control measures, including rigorous hygiene practices and effective sanitation protocols, must be enforced in healthcare facilities and households to curb the dissemination of resistant bacteria [38]. Moreover, sustained investment in research and development of novel antimicrobials—particularly agents active against ESBL-producing organisms—remains critical to outpace evolving resistance mechanisms.

One limitation of this cross-sectional study is the lack of detailed clinical histories or records of pre-existing conditions for the enrolled cats. As a result, associations between specific health statuses, prior antimicrobial exposure, and the emergence of AMR could not be established. Additionally, minimum inhibitory concentration (MIC) testing was not conducted, limiting the ability to precisely quantify resistance levels. Although the study successfully documented AMR in feline and environmental E. coli, future investigations should explore how particular feline health conditions or treatment histories contribute to resistance development. It should also be noted that E. coli isolates from healthy cats and their drinking water may not fully represent clinical enteropathogenic scenarios in cats. From a One Health perspective, broader studies examining resistance patterns across humans, companion animals, and the shared environment are warranted. The current work focused solely on the pet–environment interface and did not include human E. coli isolates; thus, additional research is needed to elucidate the interconnected dynamics of AMR among humans, pets, and their surroundings.

Conclusions

This study offers important insights into the intricate patterns of antimicrobial resistance in Escherichia coli isolated from cat feces and drinking water. The isolates exhibited the lowest resistance to carbapenems and the highest resistance to lincomycin (clindamycin). Widespread multidrug resistance, coupled with the predominance of phylogroup B2, highlights significant therapeutic challenges in the feline population. The β-lactamase gene blaTEM was the most frequently detected resistance determinant in both fecal and environmental isolates, underscoring the entrenched nature of β-lactam resistance and the overall complexity of AMR in cats and their immediate environment.

Abbreviations

AMR Antimicrobial resistance
ESBL Extended-spectrum β-lactamase
E. coli Escherichia coli
PCR Polymerase chain reaction

Acknowledgments: The authors would like to acknowledge the Faculty of Veterinary Medicine, Kasetsart University. The authors also extend their gratitude to the staff at the Kasetsart Veterinary Teaching Hospital, Bangkhen, and the cat owners who participated in this study.

Conflict of Interest: None

Financial Support: This research is financial supported from Faculty of Veterinary Medicine, Kasetsart University: FFK (VET.KU2023-KPVRF01).

Ethics Statement: This animal use protocol was submitted and reviewed by the Kasetsart University Institutional Animal Care and Use Committee (ID#ACKU62-VET-030) and found to be in accordance with the guidelines of animal care and use under the Ethical Review Board of the National Research Council of Thailand for the conduct of scientific research. The committee approved and permitted the animal care and use to be conducted as stated in the animal use protocol for this research study. The study was carried out in compliance with the ARRIVE guidelines.

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