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INFLUENCE OF THE MODERN ENVIRONMENT ON NORMAL *ESCHERICHIA COLI (E. COLI)* BACTERIA

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Abstract: The ecological dynamics of *Escherichia coli (E. coli)* within contemporary ecosystems are progressively influenced by multifaceted environmental determinants, necessitating a thorough comprehension of their adaptive behaviors, survival strategies, and ecological interactions. This investigation seeks to assess the effects of modern environmental drivers' urbanization, antibiotic application, agricultural practices, climate change, and industrial food production on the persistence and transformation of normal *E. coli* populations, and to appraise their related public health ramifications across varied ecosystems. Methodologically, it integrates current scientific literature, epidemiological evidence, and environmental monitoring datasets to evaluate *E. coli* prevalence and adaptive mechanisms. Results demonstrate that urban environments sustain elevated microbial diversity, enabling *E. coli* dissemination, while the excessive and improper use of antibiotics accelerates the emergence and spread of antimicrobial resistance. Agricultural systems function as critical reservoirs, with soil and water contamination posing immediate risks to human health and ecological stability. Climate change intensifies these concerns by modifying *E. coli* habitats and altering transmission dynamics. Moreover, contamination within food production networks constitutes major safety risks, reinforcing the demand for stringent monitoring and control protocols. This study advances comprehension of the nexus between environmental stressors and *E. coli* ecology, offering essential insights into microbial adaptability in the Anthropocene. Its practical relevance resides in guiding integrated management strategies to mitigate environmental hazards, strengthen public health safeguards, and promote the sustainable coexistence of human activities with microbial ecosystems. These outcomes establish a basis for formulating evidence-driven policies to confront the emerging challenges associated with *E. coli* in the modern era.
Keywords: *E.coli* ecology, Environmental stressors, Antimicrobial resistance, Climate change impact, Food safety risks.

1. Introduction

Contemporary anthropogenic pressures are transforming the ecological dynamics of *Escherichia coli* across interconnected human (Table 1), animal, and environmental reservoirs. Urban expansion, intensive agriculture, antimicrobial application, and climatic variability collectively influence *E. coli* persistence, transmission, and the evolution of antimicrobial resistance (AMR) within the human animal environment interface (One Health). Systematic reviews and surveillance syntheses consistently designate wastewater, healthcare effluents, farms, wildlife, and food systems as principal conduits for AMR determinants in surface waters and other environmental matrices Figure 1 (Kusi et al., 2022). Antibiotic selection pressures in clinical and agricultural domains expedite the emergence and dissemination of resistant *E. coli*, encompassing plasmid-mediated mechanisms such as *mcr-1* and extended-spectrum β-lactamases, with discernible population-level shifts ensuing from stewardship or policy interventions. Recent field and genomic investigations from diverse settings (e.g., livestock operations, manure, and wastewater) further substantiate the mobility of resistance genes and their dissemination across ecological compartments (Wang et al., 2020).

Moreover, in light of rapid urbanization, increasingly intensive farming practices, rampant use of antibiotics, and changing climate, *E. coli*, which at one time were considered as normal gut microbiota commensals, have evolved into important measures of environmental health and public health risk factors. Facing a large and growing human population through intense anthropogenic activities, the modern environment has become a very difficult and risky entity that

simultaneously offers hope to the agglomeration of normal *E. coli* populations. Urbanization builds tightly knit webs of human activities, allowing for the enclosure of *E. coli* in varying environmental sources, such as disposal of sewage systems and water pollution. In turn, the widespread use of antibiotics has provided resistance genes with an upper hand for evolution and dissemination, adding to the global resistance problem in the health arena. Furthermore, agricultural practices, including cattle and crop farming, release large amounts of *E. coli* in the soil and water reservoirs, multiplying the risk of infection through food and water pathways. Climate change is thought to magnify the effect by altering temperature and precipitation, thereby ultimately influencing the distribution and colonization of *E. coli* in environmental reservoirs. Thus, it is interesting how today environment would augments the prevalence of the normal *E. coli* bacteria. This brings forth the interconnection between microbial ecology, human activities, and ecological sustainability affecting public health and manteons the ecosystem as the world gets more interlinked (Foster-Nyarko & Pallen, 2022; Lenski, 2023). Food production systems persist as a predominant exposure pathway. Evidence from outbreak inquiries and food-handler assessments reveals recurrent associations between produce particularly leafy greens and Shiga toxin–producing *E. coli*, emphasizing vulnerabilities from primary production through processing and distribution that mandate reinforced surveillance and control measures (Marshall et al., 2020).

Collectively, climate change functions as a risk intensifier by reshaping environmental niches, seasonality, and transport pathways for enteric pathogens and resistance determinants. Empirical and modeling analyses indicate that heatwaves and extreme weather events can augment incidences of STEC infections and modify transmission patterns, while broader climatic trends may heighten the likelihood of foodborne and waterborne diseases (Boudou et al., 2025). These converging lines of evidence establish *E. coli* as a sentinel organism for environmental health and AMR dynamics. Mitigating the evolving risks necessitates integrated One Health approaches encompassing antimicrobial stewardship, wastewater and agricultural management, and food-safety interventions to disrupt transmission pathways and safeguard antimicrobial efficacy (Khadse et al., 2023; Larsson & Flach, 2022; Niazi et al., 2025). Thus, the present paper discusses the effects of current environmental factors on the specificities of normal Escherichia coli populations in terms of their ecological roles, potential for antimicrobial resistance, and public health concerns that would suggest the most effective surveillance and management approaches.

Table 1. Pathogens and phenotypes in antibiotic-resistant HAIs[1], adapted from NHSN[2] reports (2011–2019).

Pathogen	Phenotype	Abbreviation	Selected Group of Antimicrobials
Escherichia coli	Carbapenem-resistant (CRE)	*Ecoli*_CRE	Imipenem, meropenem, doripenem, ertapenem
	Cephalosporin-resistant	*Ecoli*_ESCeph	Ceftriaxone, ceftazidime, cefepime, cefotaxime
	Fluoroquinolone-resistant	*Ecoli*_Fluoroq	Ciprofloxacin, levofloxacin, moxifloxacin
	Multidrug-resistant (MDR)	*Ecoli*_MDR	Cephalosporins, fluoroquinolones, aminoglycosides, piperacillin/tazobactam
Enterobacter	Carbapenem-resistant (CRE)	*Entb*_CRE	Imipenem, meropenem, doripenem, ertapenem
	Cefepime-resistant	*Entb*_Cefepime	Cefepime
	Multidrug-resistant (MDR)	*Entb*_MDR	Cefepime, fluoroquinolones, aminoglycosides, piperacillin/tazobactam
Klebsiella	Carbapenem-resistant (CRE)	*Klebsiella*_CRE	Imipenem, meropenem, doripenem, ertapenem
	Cephalosporin-resistant	*Klebsiella*_ESCeph	Ceftriaxone, ceftazidime, cefepime, cefotaxim
	Multidrug-resistant (MDR)	*Klebsiella*_MDR	Cephalosporins, fluoroquinolones, aminoglycosides, piperacillin/tazobactam
Pseudomonas aeruginosa	Carbapenem-resistant	*P.aeruginosa*_Carbapenems	Imipenem, meropenem, doripenem
	Cephalosporin-resistant	*P. aeruginosa*_ESCeph	Ceftazidime, cefepime
	Fluoroquinolone-resistant	*P. aeruginosa*_Fluoroq	Ciprofloxacin, levofloxacin

	Aminoglycoside-resistant	*P. aeruginosa*_Amino	amikacin, gentamicin, tobramycin
	Piperacillin/tazobactam-resistant	*P. aeruginosa*_PiPTaz	Piperacillin, piperacillin/tazobactam
	Multidrug-resistant (MDR)	*P. aeruginosa*_MDR	Cephalosporins, fluoroquinolones, aminoglycosides, carbapenems, piperacillin/tazobactam
Enterococcus faecium	Vancomycin-resistant (VRE)	*E. faecium*_VRE	Vancomycin
	Daptomycin-resistant	*E. faecium*_Dapto	Daptomycin (NS)
Enterococcus faecalis	Vancomycin-resistant (VRE)	*E. faecalis*_VRE	Vancomycin
	Daptomycin-resistant	*E. faecalis*_Dapto	Daptomycin (NS)
Coagulase-negative *Staphylococci*	Vancomycin-resistant	CNS_Vanc	Vancomycin
Enterobacterales	Carbapenem-resistant (CRE)	CREall	Imipenem, meropenem, doripenem, ertapenem
Staphylococcus aureus	Methicillin-resistant (MRSA)	MRSA	Methicillin, oxacillin, cefoxitin
	Linezolid-resistant MRSA	MRSA_Linezolid	Linezolid
	Fluoroquinolone-resistant MRSA	MRSA_Fluoroq	Ciprofloxacin and/or levofloxacin
	Vancomycin-resistant MRSA	MRSA_Vanc	Vancomycin
	Daptomycin-resistant MRSA	MRSA_Dapto	Daptomycin (NS)
Acinetobacter	Carbapenem-resistant	*Acinetobacter*_Carbapenems	Imipenem, meropenem, doripenem
	Multidrug-resistant (MDR)	*Acinetobacter*_MDR	Cephalosporins, fluoroquinolones, aminoglycosides, carbapenems, piperacillin/tazobactam, ampicillin/sulbactam

Abbreviations: Healthcare-associated infections [HAIs][1], National Healthcare Safety Network [NHSN][2].

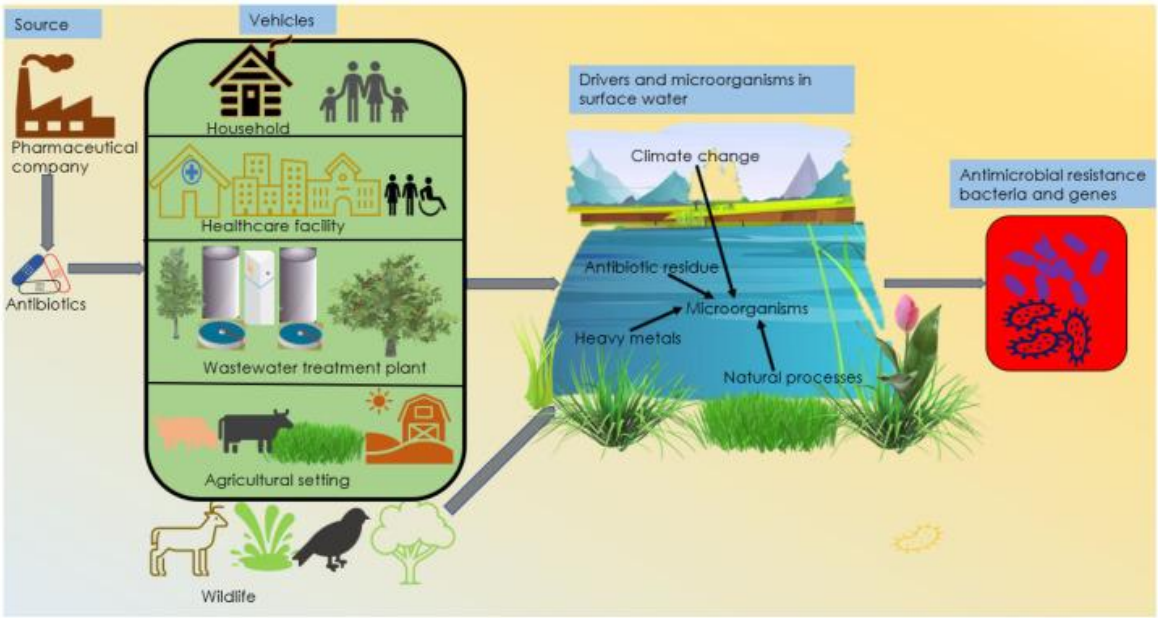

Figure 1. The model of the development pathway of antimicrobial resistance depicts the nature of sources, vehicles and the drivers of antimicrobial resistance in the water environment.

2. Materials and Method

This review article looks at how modern environmental factors affect Escherichia coli (E. coli). We searched databases such as PubMed, Scopus, and Google Scholar using keywords about E. coli ecology and environmental stressors. We chose peer-reviewed studies, epidemiological reports, and monitoring data from the past ten years that showed strong methods and important findings. We grouped the data by main environmental drivers: urbanization, antibiotic use, farming practices, climate change, and food production. This helped us analyze how these factors influence E. coli's spread and ability to adapt. We also reviewed epidemiological evidence to understand public health impacts and consulted experts to confirm our findings. Our results are meant to guide management strategies that reduce E. coli-related environmental risks and help shape evidence-based policies.

3. Results

Unequivocally, a considerable number of presumptive *E. coli* isolates from irrigation water and agricultural soil samples prior to harvest were confirmed, and many of them possessed virulence genes (for example, EAEC, EIEC, EPEC, EHEC, UPEC, NMEC) in addition to being multiresistant (MDR)-traits (Iwu et al., 2022). The highest concentration (177 423 CFU ml^{-1}) in irrigation water caused *E. coli* contamination, with retention of 15-25% on foliage, and leachate and soil retaining 231% and 116%, respectively; post-harvest, exposed foliage accumulated *E. coli* significantly (400% increase) during storage (4°C, 14 days). Of the randomly selected isolates, 81% were resistant to ampicillin and 34% to cephalothin Figure 2 (Summerlin et al., 2021). In urban and peri-urban garden ecosystems in Bangladesh, the overall presence of *E. coli* was 58.62% (95% CI: 50.48-66.31%), but varied by sites (44.7% at DNCC; 80.0% at DSCC; 76.7% at GCC). Rooftop gardens showed higher means of contamination (e.g. GCC rooftop: 93.33%) compared to surface gardens; antibiotic resistance profiling of 54 isolates revealed 100% resistance to ampicillin, with resistance also to ciprofloxacin (25.9%), tetracycline and cotrimoxazole (both 14.8%), imipenem (9.3%), fosfomycin (1.0%), and no resistance to ceftazidime, gentamicin, chloramphenicol, nitrofurantoin, or cefotaxime. Multidrug resistance (MDR) (Table 2) was observed in 14.81%, bla_TEM occurred in 81.48%, and tetA in 3.70% of these isolates (Pramanik et al., 2025).

In another rural surface waters survey, Northeast Tennessee, the prevalence of MDR E. coli was 47.5%; the susceptibility phenotypes include those for ciprofloxacin (64.2%), Nitrofurantoin (62.7%), Ceftriaxone (40.1%), and 6% of isolates were colistin resistant (Alali et al., 2024). Lastly, samples of vegetables, irrigation water, and soil from China reveal ciprofloxacin-resistant (CIP^R) *E. coli* in 44.1% of water samples, 12.0% of vegetables, and 4.5% of soil samples; of 48 such isolates, 58.3% were ESBL-producing, with bla_CTX-M-55 prevailing, and genomic sequencing indicated widespread carriage of MDR genes, including mobile genetic elements like IncFIB and IncFII plasmids (Sun et al., 2024).

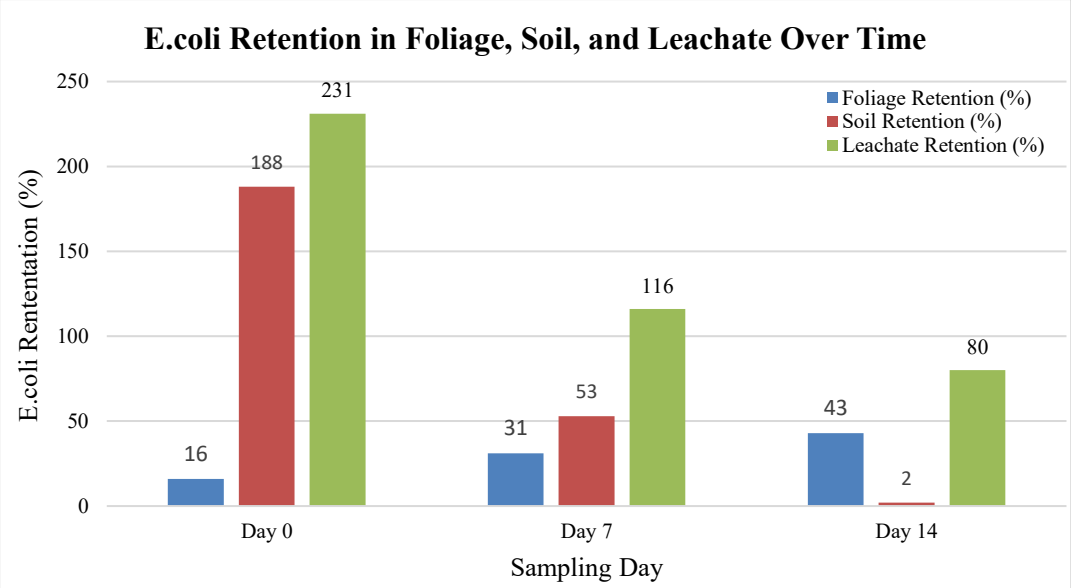

Figure 2. Temporal changes in E. coli retention in foliage, soil, and leachate, monitored over 14 days, are reflected in the chart above. The retention in foliage increased gradually from 16% at Day 0 to 43% at Day 14, which suggested gradual surface accumulation. In contrast, retention in soil decreased dramatically from 188% to 2%, revealing a rapid losing bacterial load from the soil matrix. Similarly, leachate also revealed its decreased retention from 231% to 80% during the same period, showing mobilization in the early stages of drainage pathways and later decline of *E. coli*. Most of such findings are recently emerging evidence on the persistence and mobility of *E. coli* in the environment, especially in agricultural systems.

Table 2. Multidrug resistant (MDR) *E. coli* strains resistance profiles.

Pattern No.	Resistance patterns	No. of antibiotics (classes)	No. of MDR Isolates	MDR (%)	MAR index
1	AMP, TET, CIP, COT, IMP	5(5)	7	48.14	0.45
2	AMP, TET, COT	3(3)	7		0.27
3	AMP, TET, CIP	3(3)	6		
4	AMP, CIP, COT	3(3)	6		
5	AMP, COT	2(2)	7		0.18
6	AMP, TET	2(2)	6		
7	AMP, IMP	2(2)	6		
8	AMP, FOS	2(2)	3		
9	AMP, CIP	2(2)	3		
10	AMP	1(1)	3		0.09

TET: Tetracycline, AMP: Ampicillin, CIP: Ciprofloxacin, COT: Cotrimoxazole, FOS: Fosfomycin, IMP: Imipenem.

3.1. Biology and Ecological Niche of *E.coli*

As an innovative, versatile family member among the *Enterobacteriaceae*, *E. coli* occupies all the niches comprising the gastro-intestinal tracts of humans and warm-blooded animals, soil, water, and vegetation, among others. Being a facultative anaerobe, this organism can be well considered an archetype of these biomorphs exploiting aerobic and anaerobic environments as adapted forms to flourish across oxygenated gradients with regards to oxygen levels. In fact, its genome features around 4.6 million base pairs coding for a number of distinct genes implicated in carbon utilization, stress response, and pathogenicity, thus making it superbly capable of adapting to diverse conditions of the environment. Seeming innocuous or beneficial in its commensensal state within the gut microbiota, *E. coli* contributes to the health of the host through participation in nutrient metabolism and immunomodulation, aside from which virulent strains contain the necessary virulence factors for disease causation, from simple infections of the gastrointestinal tract to severe systemic diseases. Besides human well-being, *E. coli* has many other important functions within nutrient cycles and structure and dynamics of microbial communities in environmental ecosystems, where, among others, it serves as a marker for indicating possible fecal contamination and the health of an ecosystem. Understanding *E. coli* kinetics at the organismal and ecosystem level thus becomes of paramount importance in understanding how *E. coli* relates so diversely with its hosts and its environments, thus improving initiatives that focus on the design of disease control mechanisms and environmental monitoring programs as well as discovery in microbial systems biology (Zhang et al., 2023; Yang et al., 2022).

3.2. The Ecological Role And Their Natural Habitats Of *E. Coli* In Various Environments

E. coli is very famous when it comes specifically to human and warm-blooded animals' gastrointestinal tracts. Although it is largely associated with the small intestines, *E. coli* shows remarkable variability and range in terms of ecological niches and habitat outside the confinement of the digestive system. *E. coli* is characterized as inhabiting certain other ecological niches aside from the gastrointestinal microbiota, and in a way, it can flourish and grow under a diverse range of environmental conditions, such as soil, water, vegetation, and sediment. Under these niches, *E. coli* assumes varying ecological roles including nutrient cycling, organic matter decomposition, and even influencing the dynamics of microbial communities. Since *E. coli* can use facultative anaerobic respiration, it can survive under environmental conditions with varying oxygen levels. Although some *E. coli* lineages exhibit some metabolic versatility, enabling them to degrade various energy sources and incorporate them into their environment via assimilation, *E. coli* is, in natural ecosystems, recognized as an indicator organism for fecal contamination, which provides very relevant information on water quality and overall ecosystem health. In furtherance of Environmental Management Planning and Appraisal, ecological significance of this organism can only be appreciated with knowledge of the various niches and ecological roles of this organism, thus deepening the understanding of its ecosystemic interactions (Petersen & Hubbart, 2020; Touchon et al., 2020).

3.3. Evolutionary Trajectories and Adaptive Mechanisms

The *E. coli* species have very interesting dynamics of evolution and adaptation that profess the creation of dominance and continued existence in environments in relation to ecological conditions. The ecological populations of *E. coli* are always willing, with the assistance of lateral gene transfer among groups, mutations, and genomic changes, to constantly acquire and exchange common pieces of information for the quick adaptation to environmental challenges. *E. coli* maintain their existence and confer upon themselves various phenotypic advantages by employing horizontal gene transfer. HGT mechanisms such as conjugation, transduction, and transformation enable *E. coli* to acquire antibiotic resistance and metabolic genes that are often donated to them by other bacteria within their environment. Furthermore, in terms of genetic diversity, the genetic world in the form of mutations creates variability within *E. coli* communities, the underlying nature being such that natural selection can act upon it. In this regard, *E. coli* is genetically rather flexible so that it can undergo an evolutionary change in a short time upon exposure to such selective forces as antibiotics and environmental change (Hasan & Ahn, 2022; Batarseh et al., 2023).

3.4. Genetic And Phenotypic Adaptations of *E. Coli* To Environmental Stressors

Under heterogeneous environmental stressors such as thermal fluctuations, nutrient deprivation, or chemical exposure *E.coli* undergoes genetic and phenotypic adaptation through orchestrated genomic mutations and transcriptomic reprogramming, encompassing nucleotide substitutions, insertions, deletions, chromosomal rearrangements, and copy number variations, collectively modifying regulatory networks and stress-responsive pathways to optimize survival under adverse conditions Table 3 (Jiao et al., 2024). Experimental evolution further demonstrates that *E. coli* populations consistently develop convergent genotypic adaptations frequently within global regulators such as *rpoB*/*rpoC* accompanied by parallel phenotypic shifts, exemplifying the predictability of adaptive trajectories under defined selective pressures (Table 4) (Jiao et al., 2024; Venkataraman et al., 2024).

Table 3. Genomic modifications in the genes through altered environmental selections.

Environments	Genes, types and features of mutations			Strain IDs
	Genes	Types	Features	
Nutrient stress	*hfq*, *rpoS*, *paaX*, *lrp*, *sdhB*, *dtpA*, *glpR*	nsSNP, indel, IS, deletion, amplification	Parallelism	K-12 W3110
Feast/famine cycle	*crp*, *fusA*, *ompR*, *rpoS*, *rpoB*	SNP, indel, IS, deletion	Parallelism	K-12 MG1655
Heavy metal	*araD*, *ulaD*, *galM*, *filI*, *emrB*, *nusA*, *araG*, *nepI*	BPS, indel	Metal specificity, consistency	K-12 MG1655
Antibiotic environment	*mrdAB*, *mreBCD*, *pykF*, *spoT*, *topA*	\	Parallelism, antagonistic pleiotropy	REL606 (B)
Ampicillin	*ftsI*, *phoQ*, *dauA/prs*, *mgrB/yobH*	nsSNP, sSNP, indel, intergenic, IS	Stability, parallelism	K-12 MG1655
Imipenem	*dacC*, *mrdA*, *dacD*, *mrcB*, *ftsI*, *zwf*, *ttuC*, *lpxD*, *secF*, *envZ*	sSNP, nsSNP, intergenic, insertion, deletion	\	Sx181-0-1, ATCC25922
Adaptive laboratory evolution	*pykF*, *zwf*, *spoT*, *mrdA*, *hns/tdk*, *rpo C*, *rpoB*	SNP, indel, insertion, deletion	Consistency	K-12 MG1655/W3110, BL21, C, W, Crooks
osmotic stress	*dnaQ*, *mutS*, *mutL*, *mutH*, *uvrD*, *mu tM*, *mutY*, *mutT*	sSNP, nsSNP, insertion, indel	strong hypermutator phenotypes	DH10B
Luria-Bertani broth	*cadC*, *rseB*, *cytR*, *iscR*, *gcvA*, *sspA*, *arcA*, *proQ*, *rbsR*, *rplA*, *fimH*, *fimG*, *proQ*, *lsrK*	nsSNP	Parallelism	K-12 MG1655

In parallel with these intrinsic adaptations, horizontal gene transfer (HGT) accelerates the acquisition of metabolic functions: evolutionary reconstructions reveal that *E. coli* has attained thousands of metabolic innovations through single DNA segment transfers (<30 kb), enabling proliferation in previously inaccessible environments by introducing novel enzymatic pathways and enhancing phenotypic versatility (PNAS) Figure 3 (Pang & Lercher, 2018). Moreover, genomic analyses of food-borne *E. coli* isolates corroborate their role as reservoirs of mobile genetic elements mediating the horizontal spread of both virulence and antimicrobial resistance determinants, reinforcing HGT as a pivotal mechanism underpinning genomic plasticity and adaptive potential in fluctuating ecosystems (Balbuena-Alonso et al., 2022; Xing et al., 2021; Blount et al., 2020; Rodríguez-Beltrán et al., 2021; Haudiquet et al., 2022).

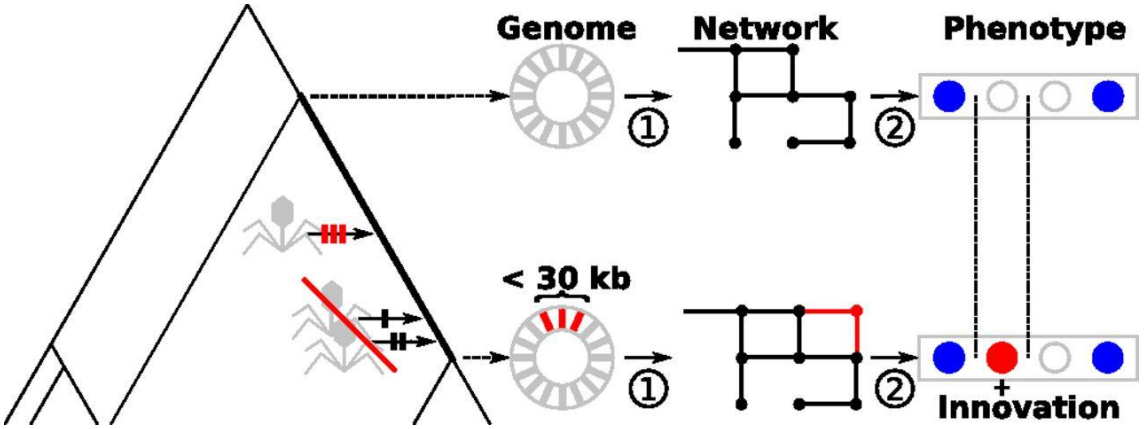

Figure 3. *E. coli* phylogeny, phenotypic innovations were detected with the use of FBA, and were found to occur as single <30 kb horizontal gene transfer events.

Table 4. Gene transcriptomic alterations in response to different environment selections.

Environments	Groups	DEGs and their involved metabolic pathways or biological functions*				Strain IDs
		Up-regulation		Down-regulation		
Carbon starvation	P1-P2	367	Fermentation and response to starvation	1542	Glucose transport	K-12 MG1655
	P2-P3	15	Acetate metabolism	855	Sugar and amino acid transport; Cell motility	
	P3-P4	107	Stress response; Motility regulation	178	Amino acid/sugar and acid transport; Catabolism	
					pathway; Cell motility	
Heat stress	Early	20 (e.g., *clpB*, *dnaKJ*, *groSL*, *grpE*, *hslVU*, *hspQ*, *htpG*, *ibpA*)	Response to heat	18 (e.g., *purB*, *purD*, *purE*, *purL*, *purT*)	Purine biosynthesis	BL21
	Middle	12	Not detected	8	Not detected	
	Late	47 (e.g., *entCEB*, *entD*, *fes-entF*)	Enterobactin metabolic process	19	Not detected	
Exposure to Cr (VI)	MDR strain	194		320		LM13, ATCC25922
		arnA, *mdtG*, *mdtL*	Antibiotic resistance	*mdtE*, *mdtF*, *mdfA*	Antibiotic resistance	
		rsmB, *rlmJ*, *rsmJ*	DNA and RNA methyltransferase	*rlmI*, *rlmG*, *rlmF*	DNA and RNA methyltransferase	
		cybB, *cysC*, *cysH*, *cysN*	Oxidative stress response			
	Susceptible strain	304		461		
		arnA, *bcr*, *mdtE*, *mdtG*, *mdtO*	Antibiotic resistance genes	*rlmN*, *rlmI*, *rlmG*, *rlmF*	DNA and RNA methyltransferase	
		ssuB, *ssuC*	Sulfur transport	*sodB*, *katG*	Oxidative stress response	
		cybB, *sodA*, *cysC*, *cysH*, *cysJ*, *cysM*	Oxidative stress response			
	Rumen	59			Drug export	EDL933

Bovine digestive content		*emrD*, *glpD*, *nem A*	Drug export	8 (e.g., *argI*, *arn A*, *arnD*/*pmrJ*, *arnT*)		
		ychH, *yhcN*	Multiple stress response			
	Small intestine	59		8 (e.g., *argI*, *arn A*, *arnC*, *arnT*, *arnD*/*pmrJ*)	Drug export	
		emrD, *glpD*, *glpT*, *tnaB*	Drug export			
		adiA, *cadB*, *caiF*	Acid resistance response			
		clpB, *dnaK*	Temperature-change response			
	Rectum	32 (e.g., *ahpC*, *ahpF*, *grxA*, *katG*, *sodB*, *trxC*, *yaaA*, *yhaK*)	Oxidative stress response	27		
				argI, *arnA*, *ar nC*, *arnT*, *arn D*/*pmrJ*	Drug export	
				gadA, *gadE*/*yh iE*	Acid resistance response	
				slp, *yhiM*, *yhi O*	Multiple stress response	
Guanylhydr-azone treatment	Pathogenic strain	383		366		RM8082, ATCC25922
		marA, *gadX*	Multidrug efflux system	*eptA*, *arnC*	Multidrug efflux system	
		cma, *cba*, *clpF*, *ea eH*	Virulence genes	*rpl*/*rpm*, *rps*	Ribosome assembly pathway	
				flgB, *flgC*, *mot A*, *motB*	Flagellar assembly pathway	
	Nonpathogenic strain	787		1169		
		mdtM, *gadX*, *mdt G*, *evgA*, *mdtH*	Multidrug efflux system	*eptA*, *yojI*, *ms bA*, arnC, *mdfA*, *e mrB*, *emrA*, *ba cA*	Multidrug efflux system	
				rpl/*rpm*, *rps*	Ribosome assembly pathway	
		clbR, *clbD*, *clbE*, *clbA*, *clbF*, *clbC*, *clbI*, *clbB*, *clbG*, *c lbQ*	Virulence genes	*flgB*, *flgC*	Flagellar assembly pathway	
				murA, *mrcA*	peptidoglycan biosynthesis	

*Genes showing the expression levels of ≥ 2- or ≤ 0.5-fold were considered DEGs.

3.5.Urbanization and *E. Coli* Distribution

Urbanization profoundly shapes the environmental prevalence and distribution of *E. coli*, as evidenced by elevated detection in urban and peri-urban gardens with an overall prevalence of 58.6% and up to 93.3% in rooftop gardens in Bangladesh, indicating substantial urban contamination reservoirs (Pramanik et al., 2025). In Nairobi, Kenya, genome sequencing of 1,338 isolates from humans, livestock, and wildlife revealed extensive intra-household strain sharing and structured resistome patterns, underscoring urban human–animal interfaces as pivotal hotspots for *E. coli* circulation and antimicrobial resistance exchange (Muloi et al., 2022). Spatial analyses within urban stream networks demonstrated that *E. coli* abundance correlates positively with sewer density and nutrient loads but inversely with phylogenetic diversity, reflecting habitat simplification that fosters dominant strains linked to sanitation infrastructure Figure 4 (Saraceno et al., 2021). Similarly, drinking water in urban Punjab exhibited 56% *E. coli* contamination substantially higher than the 21.9% in rural sources highlighting infrastructure-associated risks in urban water supply systems (Gautam et al., 2025). Environmental modeling of Pakistan's Kabul River under urbanization and development scenarios projected mid-century *E. coli* load increases of up to 111%, escalating to 201% by late century with inadequate wastewater management, whereas improved treatment could reduce loads by over 90% (Iqbal et al., 2019). In semi-arid regions, geospatial mapping

revealed widespread antibiotic-resistant *E. coli* contamination of drinking water sources, identifying urbanization-linked hotspots associated with poor infrastructure and heightened physicochemical pollutants (Srivastava et al., 2025). Temporal monitoring of the Brunei River documented escalating coliform concentrations driven by urban expansion, increased impervious surfaces, and sewage discharge despite initial estuarine mitigation, signifying intensifying urban contamination over time (Onifade et al., 2025). Moreover, highly urbanized surface water systems demonstrated elevated detection of urban-associated fecal indicators and pathogens, including STEC, implicating urban fecal pollution as a major driver of *E. coli* dissemination (Yuan et al., 2019). Collectively, these findings underscore that urbanization amplifies both the prevalence and ecological heterogeneity of *E. coli*, with infrastructure inadequacies, habitat simplification, and human–animal interfaces functioning as critical determinants of its urban distribution (Saraceno et al., 2021; Piyapong et al., 2021; Muloi et al., 2022; Lagerstrom et al., 2024; Okumu et al., 2023; Balbin et al., 2020).

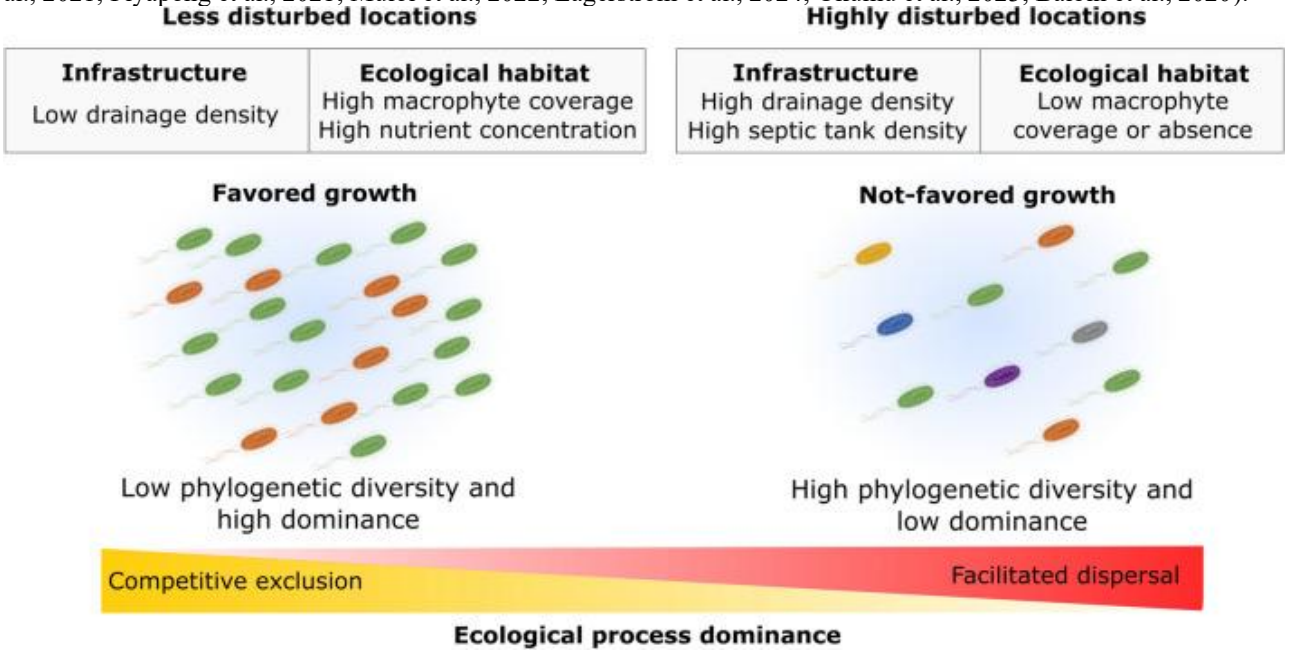

Figure 4. Schematic of ecological processes driving *E. coli* dynamics across urban gradients.

3.6.Antibiotic Resistance In *E. Coli*: Environmental Drivers

Environmental determinants profoundly shape the emergence and dissemination of antibiotic-resistant *E. coli*, with anthropogenic activities exerting a central influence. Excessive and inappropriate antibiotic use in agriculture and aquaculture generates selection pressures that favor resistant strains. For example, antibiotic application in livestock production is associated with the development of antimicrobial resistance (AMR) in *E. coli* populations (Tufa & Birhanu, 2025), for example, overall, 66.3% (61/92) of *E. coli* isolates were resistant to ≥1 antibiotic. Resistance was highest in WWTP influent (83.3%, 20/24), followed by HW (75%, 24/32), CW (50%, 9/18), and WWTP effluent (44.4%, 8/18). Most frequent resistances were to AMS (37%), CRO (30.4%), and TE (29.3%), while CN (3.3%) and IMP (6.5%) were most effective (Figure 5) ((Abdelgalel et al., 2025). Moreover, antibiotics present in wastewater effluents and untreated sewage act as reservoirs for resistant *E. coli*, promoting their persistence and environmental dissemination (Tufa & Birhanu, 2025). Climate change intensifies these challenges by modifying environmental parameters, such as temperature and precipitation patterns, thereby enhancing the survival and proliferation of resistant bacteria (Kou et al., 2025; Larsson & Flach, 2022; Iskandar et al., 2020).

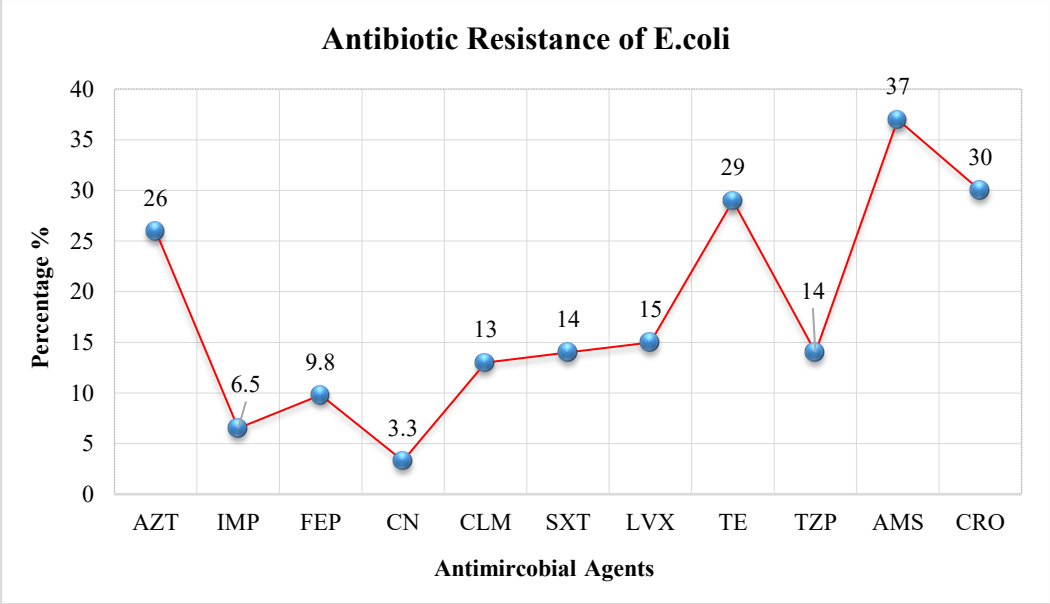

Figure 5. Percentage of *E. coli* isolates resistant to tested antimicrobial agents. Aztreonam (AZT), Imipenem (IMP), Cefepime (FEP), Gentamycin (CN), Chloramphenicol (CLM), Trimethoprim/Sulphamethoxazole (SXT), Levofloxacin (LVX), Tetracycline (TE), Piperacillin/Tazobactam (TZP), Ampicillin/Sulbactam (AMS), Ceftriaxone (CRO).

3.6.1. Impact of Antibiotic Usage And Pollution on The Development of Antibiotic Resistance In *E. Coli*

Environmental parameters govern the significantly rapid increase in *E. coli* antibiotic resistance-the resistance strains evolve due to antibiotic usage and pollution. In agriculture, aquaculture, and health care, the use of antibiotics is excessive and inappropriate, generating selective pressure on bacterial populations that facilitate the development of resistance strains. The application of antibiotics in livestock farming, for example, has been shown through studies to lead to AMR development in *E. coli* populations. Typically, pollution by unsanitary disposal, too, functions as a reservoir for resistant strains of *E. coli*. Studies show that *E. coli* from both untreated hospital effluent and the industrially polluted river has been proved resistant to antimicrobials which pose large public health threats. Industrial sources of concerned heavy metals further call for co-selection of antibiotic resistance in *E. coli*, as these metals induce stress responses useful in developing different resistance mechanisms (Tufa & Birhanu, 2025; Girijan et al., 2020; Hanna et al., 2020).

3.6.2. Spread of Antibiotic Resistance Genes in the Environment and Their Implications

The dissemination of antibiotic resistance genes (ARGs) in environmental settings poses a significant threat to public health and the efficacy of antimicrobial therapies. Environmental reservoirs such as untreated sewage, agricultural runoff, and wastewater treatment facilities serve as critical hotspots for the propagation of ARGs. Studies have identified that untreated hospital sewage and industrially polluted river water harbor antimicrobial-resistant *E. coli*, highlighting the role of environmental contamination in the spread of resistance (Tao et al., 2022). The mechanisms facilitating the horizontal gene transfer (HGT) of ARGs include conjugation, transformation, and transduction, with conjugation being the most prevalent method among *E. coli* populations. Mobile genetic elements such as plasmids, integrative and conjugative elements (ICEs), and transposons play pivotal roles in the acquisition and dissemination of resistance determinants (Tao et al., 2022). These elements enable *E. coli* to acquire resistance to multiple antibiotic classes, including extended-spectrum beta-lactamases (ESBLs), carbapenemases, and colistin resistance genes (Wang et al., 2025; Zhuang et al., 2021; Jian et al., 2021).

3.7.Agricultural Practices and *E. coli* Contamination

Agricultural practices profoundly affect the distribution and prevalence of *E.coli* in environmental matrices, with manure application and irrigation strategies identified as critical contamination pathways. Field investigations demonstrate that manure-amended soils generate substantial surface runoff during rainfall particularly in the absence of vegetative cover resulting in elevated *E. coli* loading in adjacent water bodies; notably, poultry litter application in the Chesapeake Bay region corresponded with a 6.2–18.9% increase in cephalosporin-resistant *E. coli* concentrations in streams (Sarnino et al., 2025). Soil leaching experiments further indicate that within the initial 24 hours of rainfall, swine-manured soils can transmit approximately 10^3 CFU of *E. coli* to depths of 60 cm, signifying considerable groundwater risk (Michelon et al., 2023). Comparative land-use analyses in South Carolina revealed that streams draining mixed forest/pasture landscapes contained ~2.9-fold higher *E. coli* levels than exclusively forested areas, linking pasture proximity with elevated

contamination (Britt et al., 2025). Similarly, irrigation of romaine lettuce with treated municipal wastewater resulted in retention rates of 15–25% of *E. coli* on foliage, with soil and leachate harboring even greater loads (116–231%), alongside elevated antimicrobial resistance prevalence (81% resistant to ampicillin, 34% to cephalothin) (Summerlin et al., 2021). In Ghana, *E. coli* was identified in 98% of samples from lettuce, soil, irrigation water, and poultry manure; resistance exceeded 90% for cephalosporins and beta-lactams, with blaCTX-M detected in isolates from manure, soil, and irrigation water (Appau & Ofori, 2024). On Slovak dairy farms, untreated cattle manure application introduced resistant *E. coli* into soils, contaminating crops and enabling pathogen leaching into groundwater (Dančová et al., 2024). Organic field trials demonstrated that manure application methods particularly surface application compared to incorporation affected pathogen survival and produce risk, with temperature and soil moisture interacting to modulate *E. coli* persistence (McKenzie-Reynolds et al., 2025; Lenzi et al., 2021; Rahman et al., 2021; Muirhead & Schoensee, 2023; Richter et al., 2021).

3.8.Food Production and Safety

Food production systems provide the main pathways for *E. coli* contamination continuing to pose threats to public health and safety. Manure fertilization from cattle or other livestock links very often with agricultural production issues stemming from the contamination of fresh produce with pathogenic strains of *E.coli* such as STEC and EHEC, with very low infective doses (e.g., 10^2 CFU) being able to cause disease in humans (Mueller & Tainter, 2025; Niazi et al., 2025). Outbreaks cite that pre-packaged salads as well as sandwiches contaminated with Shiga toxin-producing *E. coli* O145 in 2024 accounted for 211 confirmed cases in the UK, including one death, emphasizing that the stakes of contamination in minimally processed foods are high (Wikipedia contributors, 2024). Ready-to-eat produce remains under particular threat, especially when harvested from fields affected by environmental runoff or wildlife invasion (Bintsis, 2017). Contaminated water and poor hygiene during processing significantly heighten the risk handling lapses, inadequate sanitation, and suboptimal post-harvest controls exist, which allow pathogens to survive and spread Table 5 (Viana et al., 2025; Owade et al., 2025).

. Renowned food safety research suggests that there is an urgent need to upgrade environmental monitoring, source tracking, and genome-based surveillance to promptly detect and address any contamination in the supply chain (Kornacki, 2025). Contamination risks of extreme rainfall, as climate-induced weather irregularities, increase runoff and transport pathogens over the fields (Thorsen et al., 2025; Chandipwisa et al., 2025; EFSA BIOHAZ Panel et al., 2020; Singha et al., 2023; Gemeda et al., 2023; Christensen et al., 2021; Petrucci et al., 2021).

Table 5. The antimicrobial resistance and prevalence of food handlers exposed to *E. coli* globally.

Country	Location of Collection	Positive Isolates from Food Handler (%)	Sample Type	Antimicrobial Mechanisms	Resistance Genes	MDR (%)
Ethiopia	Multiple	95/384 (24.7%)	Hand and Fecal	Not analyzed	Not analyzed	56/95 (59.0%)
Ethiopia	University cafeterias (Including Hospital)	245/290 (84.5%)	Fecal	43 ESBL 4 Carbapenemase	Not analyzed	104/245 (42.4%)
Ethiopia	University cafeterias	119/220 (54.1%)	Fecal	29 ESBL	Not analyzed	27/119 (22.7%)
Qatar	Migrant food handlers during mandatory medical screening	78/456 (17.1%)	Fecal	7 ESBL	Not analyzed	21/78 (27.0%)
Kuwait	Commercial eateries and Healthcare settings	425/681 (62.4%)	Fecal	80 ESBL	Not analyzed	130/425 (30.6%)
Gambia	Schools	8 ESBL producing *E. coli*/565 *	Fecal	8 ESBL 4 AmpC 1 Carbapenemase	Not analyzed	8/8 (100.0%)
Indonesia	Hospitals	24/58 (41.4%)	Hand and Nasal	Not analyzed	Not analyzed	20/24 (83.3%)
Malaysia	Schools	28/1020 (2.8%)	Hands	Not analyzed	Not analyzed	4/28 (14.3%)

China	Military hospital	92/103 (89.3%)	Fecal	7 ESBL 46 intI1 2 qepA1 1 qnrS1 1 qnrB6	5 $bla_{CTX-M14}$ 1 $bla_{CTX-M79}$ 1 $bla_{CTX-M-106}$	47/92 (51.1%)
Morroco	Hospital	18/40 (45.0%)	Hands	ESBL not detected 16 metallo-β-lactamase	Not analyzed	16/18 (88.9%)
Kenya	Hotels	39/885 (4.4%)	Fecal	Not analyzed	Not analyzed	16/39 (40.2%)
Tunisia	Not mentioned	378 ESBL producing *E. coli*/2135 *	Fecal	378 ESBL	219 $bla_{CTX-M-15}$ 70 $bla_{CTX-M-1}$ 52 $bla_{CTX-M-27}$ 23 $bla_{CTX-M-14}$ 10 bla_{SHV-12} 3 bla_{SHV-2a} 1 $bla_{CTX-M-3}$	Not informed

MDR (multidrug resistant); ESBL (extended spectrum β-lactamase). *Quantity of *Escherichia coli* isolates was not provided; this study screened for ESBL and then confirmed the bacteria.

3.9.Human Health Risks and Management of *E. coli* Infections

These pathotypes of *E. coli* include enterohaemorrhagic (EHEC/STEC) and enterotoxigenic (ETEC) strains, associated mainly with diarrhea, and those causing urinary tract infections (UTIs), bacteremia, or sepsis-in-a gastric-irritated setting, adding a lot to global morbidity and health care burden (Mueller & Tainter, 2025; Doua et al., 2023). U.S. regions surveillance data indicated a monthly incidence rate of 151.7 in *E. coli*-associated UTIs, affecting predominantly older adult women, of whom 7.8% yielded extended-spectrum β-lactamase (ESBL)-producing strains with <1% resistant to carbapenems (Brandenburg et al., 2025). IED remains one of the most common causes of bloodstream infections worldwide, with high case fatality rates: 12.4% overall and up to 20% in older persons, often associated with antimicrobial resistance and delays in the initiation of therapy (Doua et al., 2023). Genomic surveillance, especially with the use of whole-genome sequencing (WGS), improves and broadens the insights into epidemiology and helps the public health interventions on outbreak detection and source tracking in STEC infections (Nouws et al., 2023). And it has been suggested that One Health genomic surveillance frameworks would trace cross-sector transmission pathways and phylogenetic linkages for targeted control strategies (Watt et al., 2025). Longitudinal resistance trend analyses further underscore the importance of continuous monitoring to inform policy changes and guide antibiotic stewardship (Nkontcho Djamkeba et al., 2024). Infection prevention and control (IPC) measures in health-care settings can reduce up to 70% the occurrence of healthcare-associated infections (HAIs), demonstrating the effectiveness of well-constructed IPC programs. These findings, together with other supporting evidence, indicate that *E. coli* is instrumental in contributing to public health. Therefore, it is very necessary to strengthen surveillance innovation and formulate an antibiotic policy and infection control strategy to reduce disease risk and the burden of antimicrobial resistance at the community as well as clinical settings (Puro et al., 2022; Niazi et al., 2024).

4. Discussion

Our findings thus demonstrate how the ecology of normal *E.coli* in contemporary ecological contexts is forged through cross-pressures of urbanization, agricultural input, selection via wastewater, and unpredictable climate effects, supporting the hypothesis that modern human-induced changes remold persistence, dispersal, and resistance capabilities of *E. coli*. Community assembly and pathotype distributions alter with urban-rural gradients along urban watersheds, thus invariably maintaining a strong relationship to sewer and nutrient increases and have been shown in more recent analyses at the scale of watersheds (Saraceno et al., 2021). Agricultural pathways continue to be key: latest synthesis and field studies show that *E. coli* manure transfers from soils to waterways and to produce, while bovine manure applications cause extended survivability across pathogenic and commensal strains-the patterns map quite directly on those observations of contamination risks before harvest (Sarnino et al., 2025; Murphy et al., 2024).

Wastewater continues to keep a significant hotspot "naturally" coevolving *E. coli* populations with treatment and antibiotic residues, while experimental work with authentic wastewater matrices indicates rapid resistance development-concordant with our inference that effluent interfaces amplify hazards of resistance to antimicrobials (AMR) (Yu et al., 2022). In urban surface waters and sewage, multisite surveys further document multidrug-resistant ESBL-producing *E. coli*, and the public health relevance of our environmental isolates is thus reinforced (Hossain et al., 2025). Superimposed climatic anomalies modulate disease risk and environmental forcing: heatwave conditions have been linked to increased incidences of STEC, and extreme temperature variability perturbs host-pathogen dynamics-mechanisms that help explain the spatiotemporal peaks we observed (Boudou et al., 2025).

At the food-environment interface, new beef systems research complements the knowledge in supply-chain contamination signals with respect to STEC hazards' resilience irrespective of feeding, thereby strengthening this towards joint success of supply reference systems (Talukder et al., 2025). In fact, all these collated and converging lines of evidence would locate our findings within One Health, where environmental compartments (soil, water, wastewater) function in connective reservoirs that select for, assemble, and disseminate *E. coli* and associated resistance (Balta et al., 2024; Alimyar et al., 2025).

Important limitations in our understanding come from relying on opportunistic environmental sampling (which is likely to underrepresent low-abundance pathotypes), and also transmission directionality is not completely solved without longitudinal genome-to-genome linkage; these drawbacks can be largely closed by continuing the wastewater and community surveillance of *E. coli* lineages (Paulshus et al., 2023). Further from these results, future research should include high-frequency environmental metagenomics in conjunction with clinical WGS; quantify selection in situ across interfaces of wastewater and agricultural solids; and explicitly model temperature-sensitive transmission for targeted intervention (Abdelgalel et al., 2025; Pokharel et al., 2023; Ananthakrishnan & Xavier, 2020; Singha et al., 2023; Jones et al., 2023; Doua et al., 2023; Sartelli et al., 2024; Martinson & Walk, 2020; Ko et al., 2020).

5. Conclusion

This paper explains how modern environmental influences such as urbanization, intensive farming, use of antimicrobials and climate change affect the ecology and adaptation of normal *Escherichia coli* to a significant extent. The results indicate that such pressures alter the persistence, dynamics of transmission, and genetic evolution of the bacterium, thus enhancing the probability of antimicrobial resistance as well as the possible transitions of pathogenicity. Regarding the research objectives, the study supports the idea that both anthropogenic and climatic ecological changes are the main factors that redesign the ecological niche and functional abilities of *E. coli*. The findings have significant public health, food safety, and environmental microbe implications, thus justifying the need to implement One Health approaches to act as a surveillance mechanism to track and control emerging microbial risks. Applications offer the possibility of focusing on increasing environmental stewardship, enacting specific microbial surveillance, and encouraging accountable use of antimicrobials in humans, wildlife, and the environment. In the future the studies are to be longitudinal and multi-regional ones, utilizing more modern genomic and metagenomic tools that will allow more confidently predicting evolutionary trends. In all, this publication helps in understanding the manner in which contemporary environmental set ups influence the biology of *E. coli*, and it gives a guideline on how to predict and manage microbial threats within the dynamic environment.

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