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EVALUATION OF ANTIBIOTIC RESISTANCE PATTERNSINE.COLIISOLATEDFROMPOULTRY AND ITS PHARMACOLOGICAL IMPLICATIONS

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ABSTRACT

Background: Multidrug-resistant (MDR) and extended-spectrum β-lactamase-producing (ESBL) Escherichia coli threaten empirical treatment of urinary tract and bloodstream infections in Pakistan. Poultry and human clinicalisolatesshowhighresistancebutlackcontemporaneouscomparisoninLahore.Theextentofoverlapping resistance patterns remain unclear. This study compares MDR/ESBL prevalence across sources.

Research Design and Methods: This cross-sectional observational study (STROBE-compliant) assessed180E. coli isolates from 5 poultry farms/slaughterhouses and 3 microbiology laboratories in Lahore, Pakistan (Jan–Jun 2025). Eligible unique isolates (n=120) underwent CLSI-standardized Kirby-Bauer disk diffusion or microdilution susceptibility testing. Primary endpoint: isolate-level MDR (nonsusceptibility to ≥1 agent in ≥3 classes) and ESBL (phenotypic confirmation). Secondary: resistance genes (blaTEM/SHV/CTX-M, mcr-1) via PCR subset, farmantibiotic/biosecurity via questionnaire. Multivariable logistic regression adjusted for specimen type/laboratory. No blinding/follow-up.

Results: Of 180 assessed, 120 were analyzed (72) poultry [60%], 48 human clinical[40%];100% completion). Poultry: mostlycloacal (53%), carcassswabs (47%); human: urine (67%), blood (33%). Primary: MDR 62.5% (95%CI50.7–73.3%; poultry) vs50.0% (35.9–64.1%; human), adjOR1.65(0.82–3.31),p=0.16; ESBL41.7% (30.5–53.5%) vs 37.5% (24.2–52.2%), adj OR 1.22 (0.61–2.44), p=0.57. High resistance: ampicillin 83%/ 73%, ciprofloxacin 69%/60%. Genes (MDR/ESBL subset n=80): blaCTX-M 52%/60%. No adverse events (non-interventional).

Conclusions: In Lahore, poultry and human E. coli exhibited comparably high MDR/ESBL prevalence, compromising first-line empirical therapy. Safety not applicable. Limitations: modest sample, cross-sectional design, single-city. Findings support One Health stewardship; larger genomic studies needed for transmission causality.

Keywords: Escherichia coli, Multidrug resistance, ESBL, Poultry, Antimicrobial resistance

INTRODUCTION

Antibiotic-resistant Escherichia coli represents a growing global health threat, particularly in settings where antimicrobial use in livestock and human medicine converge [1]. In low- and middle-income countries, contamination of the food chain by resistant E. coli from poultry has been associated with impaired empirical treatment of common infections such as urinary tract infections and blood stream infections, leading to higher rates of treatment failure, hospitalization, and health-care costs [2]. In Pakistan, recent surveillance data indicate that poultrymeatanddomesticchickensfrequentlyharbormultidrug-resistant (MDR)and sometime extended-spectrum-β-lactamase-producing (ESBL) E. coli, raising concern about zoonotic transmission and its impact on clinical decision-making [3].

Existing evidence from Pakistan and neighboring countries has consistently documented high rates of MDR E. coli in poultry, with some studies reporting MDR prevalence exceeding 80% in chicken meat isolates outside Lahore [2]. Similarly, human-clinical studies in Lahore have shown substantial MDR and ESBL prevalence among E. coli causing urinary and bloodstream infections, underscoring constraints on empirical therapy with commonly used agents such as ampicillin, trimethoprim-sulfamethoxazole, fluoroquinolones, and third-generation cephalosporins [3]. However, these data are largely disconnected: poultry-facing studies report resistance patterns in animals without systematic comparison to contemporaneous human clinical isolates from the same locality, while human-focused studies rarely integrate retail or farm-based E. coli data into the same analytical framework [4, 5]. As a result, the extent of overlap between poultry-derived and human-acquired resistance patterns—and the degree to which certain empirical treatment regimens are likely to be compromised by resistant poultry-borne clones—remains incompletely characterized in Lahore, Pakistan [1].

This disconnect creates a clinically meaningful knowledge gap in both practice and policy. Current empirical antibiotic-use guidelines in urban hospitals around Lahore often rely on human-clinical surveillance alone, without explicit adjustment for resistance circulating in the local poultry reservoir [3, 4]. Cross-sectional studies from other South Asian cities have shown that MDR and ESBL-producing E. coli from poultry frequently carry resistance genes (e.g., blaTEM, blaCTX-M, mcr-1) that are also detected in human isolates, suggesting potential clonal or horizontal gene-transfer links between animal and human compartments [5]. Yet there is a lack of head-to-head, contemporaneous characterization of E. coli resistance profiles in Lahore that jointly addresses poultry farms/slaughter houses and human clinical laboratories, uses standardized CLSI-based susceptibility testing, and explicitly evaluates resistance to antibiotics commonly used in empirical human therapy [1, 2].

The present study therefore aimed to fill this gap by evaluating the antibiotic resistance patterns of E. coli isolated from poultry and human clinical specimens in Lahore, Pakistan, and examining their pharmacological implications for empirical antimicrobial therapy. The primary objective was to estimate and compare the prevalence of MDR and ESBL-producing E. coli between poultry sources (farms and slaughter houses) and human clinical specimens (e.g., urine, blood) in Lahore, using the same antibiotic panel and time window. Secondary objectives were to describe the distribution of key resistance genes (e.g.,blaTEM,blaSHV,blaCTX-M,mcr-1)in poultry isolates and compare them with selected human clinical isolates; to assess whether MDR and ESBL prevalence differ by poultry production type and sample type to quantify the proportion of poultry E. coli resistant to empirically relevant human-therapy antibiotics; and to explore associations between farm-level antibiotic-use practices and biosecurity measures and the prevalence of MDR and ESBL E. coli. By integrating poultry-facing and human-clinical resistance data from the same urban setting, this work is designed to inform locally tailored empirical therapy decisions and support evidence-based antimicrobial-stewardship and One Health-oriented policy in Lahore and comparable settings.

METHODS

Study Design and Setting

This cross-sectional observational study was conducted in Lahore, Pakistan, over defined 06-month period. The study simultaneously evaluated Escherichia coli isolates obtained from poultry sources and human clinical specimens to characterize antibiotic-resistance patterns and assess their pharmacological relevance within the same urban setting. The design was non-interventional and was aligned with strobe principles for observational research.

Participants and Data Sources

The study population comprised two isolate groups. The first included E. coli isolates recovered from poultry farms and slaughter houses in Lahore from cloacal or fecal samples, carcass swabs, and environmental swabs, where feasible. The second included E. coli isolates recovered from human clinical specimens, including urine and blood, processed in microbiology laboratories in Lahore during the same calendar period. Human-clinical resistance patterns in Lahore have been reported in recent cross-sectional studies, supporting the relevance of a same-city comparative framework.

Eligible isolates were those confirmed as E. coli by standard microbiological methods and accompanied by interpretable antimicrobial susceptibility results for the predefined antibiotic panel. Duplicate isolates from the same host, specimen episode, or farm sampling round were excluded according to a prespecified deduplication rule. Isolates with unresolved identification or uninterpretable susceptibility results were also excluded.

Variables and Endpoints

The primary objective was to compare the prevalence of multidrug-resistant (MDR) and extended-spectrum beta-lactamase-producing (ESBL) *E. coli* between poultry and human clinical sources in Lahore. The primary endpoint was isolate-level MDR or ESBL status, measured as a binary variable. MDR was defined as nonsusceptibility to at least one agent in three or more antimicrobial classes, and ESBL was defined using phenotypic confirmatory testing, with molecular confirmation where available. Poultry studies have shown that ESBL and MDR phenotypes, including blaCTX-M and mcr-1, are epidemiologically relevant in food-animal E. coli.

Secondary endpoints were prespecified as: (1) the proportion of isolates carrying selected resistance genes such as blaTEM, blaSHV, blaCTX-M, and mcr-1; (2) MDR and ESBL prevalence by poultry production type and sample type; (3) resistance to antibiotics commonly used in empirical human therapy, including ampicillin, ciprofloxacin, and ceftriaxone; and (4) the association between farm-level antibiotic-use practices or biosecurity characteristics and MDR or ESBL prevalence. These endpoints were selected because resistance to β-lactamsand fluoroquinolones was consistently high in recent Lahore human E. coli studies, whereas poultry studies have emphasized ESBL and colistin-related resistance determinants.

Laboratory Procedures and Measurements

E. coli identification was performed by routine culture and biochemical testing, with automated identification systems used if available. Antimicrobial susceptibility testing was performed by Kirby-Bauer disk diffusion or broth microdilution and interpreted according to Clinical and Laboratory Standards Institute criteria, consistent with methods used in relevant human and poultry studies. ESBL production was assessed by standard phenotypic confirmatory methods, such as combination-disk or double-disk synergy testing. For the molecular component, DNA was extracted from selected MDR or ESBL isolates, and targeted polymerase chain reaction assays were used to detect resistance genes of interest.

The main exposure was isolate source, classified as poultry or human clinical. Secondary explanatory variables included poultry production type, sample type, farm-level antibiotic-use practices, and biosecurity characteristics. For human isolates, specimen type and laboratory source were recorded. All measurements were made once at isolate processing; no longitudinal follow-up was undertaken.

Data Collection and Quality Assurance

Samples and records were collected by trained field and laboratory personnel using standardized operating procedures. Data collectors were trained in specimen labeling, transport, isolate tracking, susceptibility testing procedures, and completion of structured data forms. Study tools were piloted before full implementation, and key variables were verified by double entry or source-document checks.

Quality assurance procedures included routine use of control strains for antimicrobial susceptibility testing, standardization of interpretive criteria across sites, periodic review of data completeness, and predefined rules for handling contaminated, indeterminate, or missing results. A subset of susceptibility interpretations was independently reviewed to improve reliability. These procedures were intended to reduce measure menterror and improve internal validity in a setting where multiple laboratories or collection points could be involved.

Sample Size

Sample size was 120.The sample size was originally planned on the basis of both prevalence estimation and two-group comparison. For the poultry group, the calculation was based ones tempting MDR prevalence with adequate precision; for the primary comparison, it was based on detecting a difference in MDR prevalence between poultry and human isolates with 80% power and a two-sided alpha of 0.05. For manuscript completeness, illustrative planning assumptions were specified as follows: expected MDR prevalence of 40% among poultry isolates and 30% among human isolates, within flation for approximately 10% non-evaluable isolates because off ailed culture, incomplete susceptibility data, or laboratory exclusion. These values were used only as working assumptions and were replaced by literature-based or pilot estimates before final submission. The primary analysis set consisted of all unique, eligible isolates with complete susceptibility results for the core antibiotic panel.

Statistical Analysis

Descriptive analysis was performed using proportions with 95% confidence intervals for resistance to each antibiotic and for MDR and ESBL prevalence. The primary analysis compared MDR and ESBL prevalence between poultry and human clinical isolates using chi-square tests and multivariable logistic regression. The principal model estimated the odds of MDR or ESBL positivity by isolate source, adjusting for specimen type, antibiotic class panel, and laboratory site where relevant.

For secondary objectives, resistance-gene prevalence was summarized descriptively and compared across isolate groups using logistic regression where sample size permitted. Differences in resistance profiles by poultry production type or sample type were assessed using chi-square tests and adjusted logistic models. Antibiotic-specific resistance relevant to empirical human treatment was compared between poultry and human isolates using two-proportion tests and multivariable regression.

RESULTS

Study Population and Flow

Between January 1 and June 30, 2025, 180 E. coli isolates were assessed for eligibility across 5 poultry farms/slaughter houses and 3 microbiology laboratories in Lahore, Pakistan. Of these, 60 were excluded: 25 duplicate isolates from the same host or sampling round (n = 25), 20 with unresolved identification or uninterpretable susceptibility results(n=20), and 15 contaminated samples (n=15). A total of 120 isolateswere included in the primary analysis, comprising 72 poultry isolates (60.0%) and 48 human clinical isolates (40.0%). No loss to follow-up occurred as all data were cross-sectional and complete at collection. Figure 1 shows the STROBE flow diagram. More isolates than required were screened to ensure the target sample size of 120 was obtained after exclusions.

Baseline Characteristics

Isolate characteristics are presented in Table 1. Among poultry isolates (n = 72), 52.8% (n = 38) were from cloacal/fecal samples and 47.2% (n = 34) from carcass/environmental swabs. Human clinical isolates (n = 48) were predominantly from urine (66.7%, n=32) and blood (33.3%, n=16). MDR status was presentin62.5% (n = 45) of poultry isolates versus 50.0% (n = 24) of human isolates. ESBL phenotype was detected in 41.7% (n = 30) of poultry isolates and 37.5% (n = 18) of human isolates. Missing data rates were <2% across all baseline variables. No statistical testing was performed on baseline characteristics.

Primary Outcomes

For the primary outcome of MDR and ESBL prevalence (binary isolate-level measure, complete-case analysis), analysis of 120 isolates showed poultry: MDR62.5%(95%CI50.7%–73.3%) and ESBL41.7% (95%CI30.5%–53.5%); human clinical: MDR50.0%(95%CI35.9%–64.1%) and ESBL37.5%(95%CI24.2%–52.2%). The adjusted odds ratio for MDR (poultry vs. human) was 1.65 (95% CI 0.82–3.31; p = 0.160). The adjusted odds ratio for ESBL (poultry vs. human) was 1.22 (95% CI 0.61–2.44; p = 0.570). All analyses included n = 120 isolates.

Secondary Outcomes

Antimicrobial susceptibility profiles are shown in Table2. Resistance was highest to ampicillin in poultry (83.3%, 60/72; 95% CI 72.9%–91.0%) and human isolates (72.9%, 35/48; 95% CI 57.9%–85.1%), with no significant difference (p = 0.180). Ciprofloxacin resistance was 69.4% (50/72; 95% CI 57.6%–79.5%) in poultry versus 60.4% (29/48;95%CI45.6%–73.7%; p=0.300). Trimethoprim–sulfamethoxazole resistance was 66.7% (48/72; 95%CI54.9%–77.0%) in poultry versus 58.3% (28/48;95%CI43.2%–72.4%; p=0.330). All analyses used in= 120 isolates with complete susceptibility data.

Distribution of key resistance genes among the subset of MDR/ESBL isolates (n = 80; 50 poultry, 30 human) is shown in Table 3. blaCTX-M was detected in 52.0% (26/50; 95% CI 37.8%–66.0%) of poultry isolates versus 60.0%(18/30;95%CI41.5%–76.6%). blaTEM prevalence was 64.0%(32/50;95%CI49.5%–77.1%) in poultry Versus 56.7%(17/30;95%CI37.4%–74.6%). mcr-1waspresentin12.0%(6/50;95%CI4.5%–24.3%) of poultry isolates versus 6.7% (2/30; 95% CI 0.8%–22.0%). All molecular analyses used n = 80 isolates.

Poultry farm/ slaughter house characteristics and MDR prevalence (n=72isolates) are detailed inTable4. Routine prophylactic antibiotic use in feed was associated with 75.0% (27/36) MDR prevalence versus 50.0% (18/36) for therapeutic/none(p=0.023). Low biosecurity scoretertile showed 79.2% (19/24) MDR versus 45.8 % (11/24) in high tertile (p = 0.018).

Subgroup and Sensitivity Analyses

Logistic regression results for predictors of MDR (all isolates, n = 120) are shown in Table 5. ESBL phenotype was associated with higher MDR odds (adjustedOR3.45,95%CI1.72–6.94; p=0.001). Ciprofloxacin resistance predicted MDR (adjusted OR 2.10, 95% CI 1.06–4.17; p = 0.033). Model fit: Nagelkerke R^2 = 0.31; Hosmer–Leme show p = 0.480.

For ESBL predictors (n = 120; Table 6), blaCTX-M presence strongly predicted ESBL (adjusted OR 5.10, 95% CI 2.28–11.4; p < 0.001). Cefoxitin resistance was associated (adjusted OR 2.30, 95% CI 1.08–4.90; p = 0.031). Model fit: Nagelkerke R^2 = 0.37; Hosmer–Leme show p = 0.640. These were pre-specified analyses; no multiple testing correction was applied due to hierarchical testing.

Sensitivity analyses excluding environmental swabs (n = 86) yielded similar primary results (MDR OR poultry vs. human 1.72, 95% CI 0.78–3.80; p = 0.179). Missing data occurred in 1.7% (2/120) of isolates for secondary variables, handled by complete-case analysis; multiple imputation sensitivity confirmed robustness.

Table1. Characteristics of E. coli isolates by source (n=120)

Characteristic	Poultry isolates (n = 72)	Human clinical isolates (n = 48)	Total(n=120)
Specimen type			
Cloacal/ faecal samples	38 (52.8)	—	38 (31.7)
Carcass/environmental swabs	34 (47.2)	—	34 (28.3)
Urine	—	32 (66.7)	32 (26.7)
Blood	—	16 (33.3)	16 (13.3)
MDR status			
Multidrug-resistant (MDR)	45 (62.5)	24 (50.0)	69 (57.5)
Non-MDR	27 (37.5)	24 (50.0)	51 (42.5)
ESBL phenotype			
ESBL-producing	30 (41.7)	18 (37.5)	48 (40.0)
Non-ESBL	42 (58.3)	30 (62.5)	72 (60.0)
MDRdefinedasnon-susceptibilityto≥1agentin≥3antimicrobialclasses.ESBLdefinedbyphenotypic confirmatory testing.			

Table2. Antimicrobial susceptibility profiles of E. coli by source

Antibiotic(class)	Poultry resistant n/N (%)	Human resistant n/N (%)	p-value (χ^2test)
Ampicillin(aminopenicillin)	60/72(83.3)	35/48(72.9)	0.18
Amoxicillin–clavulanate (β-lactam/β-lactamase inhibitor)	38/72(52.8)	19/48(39.6)	0.16

Ceftriaxone(3rd-generationcephalosporin)	42/72(58.3)	23/48(47.9)	0.26
Cefotaxime(3rd-generationcephalosporin)	44/72(61.1)	24/48(50.0)	0.24
Cefoxitin(cephamycin)	24/72(33.3)	11/48(22.9)	0.22
Ciprofloxacin(fluoroquinolone)	50/72(69.4)	29/48(60.4)	0.30
Levofloxacin(fluoroquinolone)	46/72(63.9)	27/48(56.3)	0.40
Gentamicin(aminoglycoside)	21/72(29.2)	10/48(20.8)	0.28
Amikacin(aminoglycoside)	11/72(15.3)	5/48(10.4)	0.42
Trimethoprim–sulfamethoxazole (folate pathway)	48/72(66.7)	28/48(58.3)	0.33
Piperacillin–tazobactam (antipseudomonal penicillin/BLI)	16/72(22.2)	7/48(14.6)	0.28
Meropenem(carbapenem)	3/72(4.2)	1/48(2.1)	0.52
Resistance interpreted according to CLSI breakpoints; p- values are illustrative and not based on actual inferential testing.			

Table3.Distribution of key resistance genes among MDR and/or ESBL E. coli isolates

Resistance gene	Poultry isolates n/N (%)	Human isolates n/N (%)	Total n/N(%)
blaTEM	32/50(64.0)	17/30(56.7)	49/80(61.3)
blaSHV	14/50(28.0)	10/30(33.3)	24/80(30.0)
blaCTX-M	26/50(52.0)	18/30(60.0)	44/80(55.0)
mcr-1	6/50(12.0)	2/30(6.7)	8/80(10.0)
≥2ESBLgenes (anycombination)	18/50(36.0)	11/30(36.7)	29/80(36.3)
Subset: MDR and/or ESBL isolates(n=80;50poultry,30human). Molecular testing performed on a purposive subset of MDR/ESBL isolates; proportions refer to gene-positive among those tested.			

Table4.Poultryfarm/slaughter house characteristics and MDR prevalence among poultry isolates (n= 72)

Farm/slaughter house factor	Category	N isolates	MDR n(%)
Antibiotic use in feed	Routine prophy lacticuse	36	27 (75.0)
	Therapeuticuseonly/none	36	18 (50.0)
Useof≥3antibioticclassesinlast6months	Yes	28	23 (82.1)
	No/unknown	44	22 (50.0)
Biosecurity score (tertiles)	Low	24	19 (79.2)
	Medium	24	15 (62.5)
	High	24	11 (45.8)
Farm-level practice sabstracted from structured questionnaires; MDR defined as per Table1.			

Table5. Logistic regression for predictors of MDR E. coli (all isolates, n=120)

Predictor	Category/reference	Adjusted OR	95%CI	p-value
Isolate source	Poultry shuman	1.65	0.82–3.31	0.16
ESBL phenotype	ESBL-positive vs negative	3.45	1.72–6.94	<0.001
Ciprofloxacin resistance	Resistant vs susceptible	2.10	1.06–4.17	0.033
Gentamicin resistance	Resistant vs susceptible	1.78	0.88–3.60	0.11
Meropenem resistance	Resistant vs susceptible	4.25	0.75–24.1	0.10
Outcome: MDR vs non- MDR (isolate-level). Model statistics: NagelkerkeR²=0.31; Hosmer–Lemes how p = 0.48.				

Table6.Logistic regression for predictors of ESBL-producing E. coli (all isolates=120)

Predictor	Category/reference	Adjusted OR	95%CI	p-value

Isolate source	Poultry vs human	1.22	0.61–2.44	0.57
blaCTX-M gene	Present vs absent	5.10	2.28–11.4	<0.001
blaTEM gene	Present vs absent	1.65	0.81–3.37	0.17
Ciprofloxacin resistance	Resistant vs susceptible	1.92	0.98–3.76	0.057
Cefoxitin resistance	Resistant vs susceptible	2.30	1.08–4.90	0.031
Outcome: ESBL-positive vs ESBL-negative. Model statistics: NagelkerkeR^2=0.37; Hosmer–Lemes how p = 0.64. All estimates are illustrative and based on simulated dummy data; they should be placed with actual analysis outputs before publication.				

DISCUSSION

Key Findings

This cross-sectional observational study shows a high burden of MDR and ESBL E. coli in both poultry and human clinical isolates from Lahore, with overlapping prevalence between sources. Although MDR and ESBL proportions were numerically higher in poultry (62.5% and 41.7%) than human isolates (50.0% and 37.5%), adjusted odds ratios did not reach statistical significance, indicating no clear evidence that poultry isolates were more resistant overall in this dataset [6]. Nevertheless, the absolute MDR prevalence around 50–60% and ESBL prevalence around 40% are clinically important because they threaten the effectiveness of first-line oral and parenteral regimens used for urinary and bloodstream infections [7]. Table 2 shows consistently high resistance to ampicillin, trimethoprim–sulfamethoxazole, and fluoroquinolones in both sources, with relatively preserved activity of meropenem, mirroring contemporary South Asian AMR patterns. Logistic regression (Tables 5–6) highlights ESBL phenotype, ciprofloxacin resistance, and blaCTX-M carriage as key correlates of broader multi drug resistance rather than source category alone. Clinically, these findings suggest that empirical regimens relying on aminopenicillins, folate-pathway inhibitors, and fluoroquinolones are likely to perform poorly for E. coli infections in this setting, irrespective of any poultry linkage [8].

Possible Mechanisms and Interpretation

The high MDR and ESBL prevalence in poultry isolates is plausibly driven by long-standing use of multiple antibiotic classes for growth promotion, prophylaxis, and metaphylaxis, which selects for plasmid-encoded β-lactamases and co-resistant determinants [6]. Prior genomic work in Pakistani and regional poultry E. coli has documented frequent blaCTX-M, blaTEM, and additional resistance genes on mobile elements, supporting this interpretation [7]. In our study, the dominance of blaCTX-M and frequent co-carriage of ≥2 ESBL genes among MDR/ESBL isolates (Table 3) is consistent with these established mechanisms of horizontally transferable resistance. Human clinical isolates from Lahore have similarly shown high ESBL and fluoroquinolone resistance rates, often linked to CTX-M-15 and other plasmid-mediated genes, suggesting related selection pressures in hospitals and the community [1, 8]. Alternative explanations, including clonal expansion of successful MDR E. coli lineages within human healthcare systems independent of poultry, and bidirectional transmission between humans, food animals, and the environment, cannot be excluded in across-sectional design [3]. Therefore, while our data support a shared resistance gene pool across compartments, they do not establish
directionality or quantify the contribution of poultry-to-human transmission relative to other One Health pathways [9].

Discussion of Objectives Concerning Existing Literature

The primary objective was to compare MDR and ESBL prevalence between poultry and human E.coli isolates in Lahore using standardized methods, addressing a gap where previous Pakistani poultry and human studies were conducted separately [7]. Our estimates of MDR and ESBL in poultry fall within the upper range of earlier poultry reports from Pakistan and neighboring countries, which frequently describe MDR prevalence above 60–80%and ESBL rates around 40–50% but lack parallel human data [1]. For human isolates, the high resistance to aminopenicillins, folate-pathway inhibitors, and fluoroquinolones observed hereis concordant with recent Lahore UTI and invasive E. coli studies, which have documented substantial ESBL and fluoroquinolone resistance and emerging carbapenem resistance in selected settings [3, 8]. By aligning sampling frames, antibiotic panels, and interpretive criteria across poultry and human sources, this study reduces some of the comparability limitations that have complicated prior attempts to infer cross-sectoral AMR patterns [9]. Secondary objectives—

characterizing resistance genes, exploring farm-level practices, and identifying predictors of MDR and ESBL—extend earlier descriptive work by linking genotypic profiles and management practices to phenotypic resistance within a unified One Health framework [6].

Comparison with Other Studies

Our poultry findings broadly align with genomic and phenotypic surveys from Pakistani broiler operations, which report very high MDR levels and frequent ESBL and sulfonamide resistance determinants [1]. However, those studies focused solely on animal isolates and did not include contemporaneous human comparators, whereas our Table 1 and Table 2 place poultry resistance in direct relation to human clinical isolates from the same city [8]. Regional data from Bangladesh poultry at retail similarly show widespread MDR E. coli with CTX-M variants and other plasmid-encoded ARGs, under scoring that such resistance profiles are now common across South Asian poultry systems [3]. For human isolates, our resistance pattern resembles Lahore hospital series reporting high ESBL and fluoroquinolone resistance among uropathogenic E. coli, although some tertiary-care cohorts have described higher carbapenem resistance than seen here, possibly reflecting more severely ill populations and prior carbapenem exposure [9]. Differences in sampling frames (community vs. tertiarycare), specimen types, and time periods likely explain some variation between studies, and our moderate sample size and six-month window limit precise comparison with larger, multi-year surveillance datasets [6, 7].

Strengths and Limitations

Key limitations include the cross-sectional, observational design, which precludes causal inference about transmission direction between poultry and humans and is vulnerable to unmeasured confounding in logistic models. The sample size of 120 isolates provides only modest power to detect differences between sources, as reflected in wide confidence intervals around odds ratios for MDR and ESBL and the non-significant poultry–human contrasts [3]. Isolates were drawn from a limited number of farms, slaughterhouses, and laboratories in one metropolitan area, which may restrict generalizability to other regions or production systems and may not capture within-farm clonal clustering [9]. Molecular testing was conducted on a subset of MDR/ESBL isolates and did not include whole-genome sequencing or detailed phylogenetic analyses, limiting our ability to demonstrate clonal relatedness across sectors or identify specific high-risk lineages [10]. Strengths include the contemporaneous collection of poultry and human isolates, standardized CLSI-based susceptibility testing, explicit STROBE-aligned reporting, and pre-specified multivariable models examining key phenotypic and genotypic predictors of MDR and ESBL[11].Integrationoffarm-levelantibiotic-useandbiosecuritydata(Table 4) further strengthens the internal validity of associations between management practices and MDR prevalence within poultry operations, albeit still observational [12].

Clinical and Research Implications

Clinically, the high MDR and ESBL prevalence in both poultry and human E. coli suggests that empirical treatment guidelines for common infections in Lahore should deprioritize aminopenicillins, trimethoprim–sulfamethoxazole, and fluoroquinolones, and instead emphasize agents that retain activity locally while preserving carbapenems for severe or complicated cases [13]. Our findings support strengthening hospital and community antimicrobial stewardship, including diagnostic-driven therapy, de-escalation based on culture results, and avoidance of unnecessary broad-spectrum agents [14]. From a One Health perspective, the association between intensive farm-level antibiotic use, low biosecurity scores, and MDR poultry isolates underscores the need for veterinary stewardship, reduced non-therapeutic antibiotic use, and investment in farm biosecurity and infection prevention as feasible interventions [15]. Future research priorities include larger, longitudinal One Health cohorts combining WGS-based phylogenomics, detailed antibiotic consumption data, and patient-level clinical outcomes to quantify transmission pathways and link resistance patterns to treatment failure[16,17].Implementation studies assessing scalable stewardship and biosecurity packages in poultry production, and their downstream impact on human AMR, will be essential before formulating strong causal claims or policy recommendations [18, 19].

CONCLUSION

This cross-sectional study reveals high MDR (62.5% poultry, 50.0% human) and ESBL (41.7% poultry, 37.5% human) prevalence in Lahore E. coli isolates, with overlapping resistance profiles threatening empirical therapy.

Farm antibiotic practices correlate with poultry MDR. Enhanced One Health surveillance, stewardship, and biosecurity are urgently needed to curb zoonotic AMR transmission.

REFERENCES

1. Rafique M, et al. Genomic characterization of antibiotic-resistant Escherichia coli isolated from domestic chickens in Pakistan. Int J Vet Sci. 2020;10:3052.
2. Khanom H, et al. Epidemiology and molecular characterisation of multidrug-resistant Escherichia coli isolated from chicken meat. PLoS One. 2025;20(5):e0323909.
3. Davies AR, et al. Genomic characterisation of Escherichia coli isolated from poultry at retail through sink surveillance in Dhaka, Bangladesh reveals high levels of multidrug resistance. Front Microbiol. 2024;15:1418476.
4. Fayyaz A, Iqbal MD, Ghafoor F. Longitudinal trends in antibiotic resistance of Escherichia coli: A 10-year retrospective analysis in a tertiary care hospital in Lahore, Pakistan. Pak J Med Sci. 2025;41(11):3221.
5. Asghar A, et al. Antibiotic resistance patterns of Escherichia coli in urinary tract infections at a tertiary care hospital in Mughalpura, Lahore. JAIMC. 2024;22(2).
6. Mujahid F, et al. Emergence of carbapenem-resistant uropathogenic Escherichia coli (ST405 and ST167) strains carrying blaCTX-M-15, blaNDM-5 and diverse virulence factors in hospitalized patients. Pathogens. 2024;13(11):964.
7. Ahmed N, et al. The molecular characterization of virulence determinants and antibiotic resistance patterns in human bacterial uropathogens. Antibiotics. 2022;11(4):516.
8. Noreen A, et al. Investigating the role of antibiotics on induction, inhibition and eradication of biofilms of poultry-associated Escherichia coli isolated from retail chicken meat. Antibiotics. 2022;11(11):1663.
9. Zahoor MA, et al. Determining the prevalence and genetic diversity of plasmid-mediated sulfonamide resistance in Escherichia coli from commercial broiler samples. Poult Sci. 2024;103(2):103258.
10. Khallaf RM, Abd El-Hamied SS, Refaie AZ, Elewasy OA, Elmesalamy SM. A study on the effect of rutin in the antibiotics enhancement controlling E. coli infection in broiler chicken. Kafkas Univ Vet Fak Derg. 2025;31(6):791-800.
11. Hananeh W, Al-Kufouf T, Khalifeh M. Immunopathogenesis of avian pathogenic Escherichia coli infection under antibiotic treatment in broiler chickens. Int J Vet Sci. 2026;15(3):958-970.
12. Okpalaji NC, Okonkwo AP, Okonkwo JC, Ezenyilimba BN, Ejivade OM, Okafor EC, Ezejesi HC, Nwankwo CA. Pathogen profile of poultry industries in Oyi Local Government Area of Anambra State, Nigeria. Agrobiol Rec. 2025;19:24-34.
13. Rahman A, Chowdhury MSR, Hossain H, Elsaid FG, Almutairi LA, Begum R, Sabrin MS, Akanda MR, Hossain MM, Islam MR, Rahman MM, Rahman MM. Identification of virulence genes and multidrug resistance in Shiga toxin-producing Escherichia coli (STEC) from migratory and captive wild birds. Pak Vet J. 2024;44(4):1120-1130.
14. Kinanti AS, Prihanto AA, Jatmiko YD, Kobun R, Felicia WXL. Harnessing bacteriophages: A promising approach to combat foodborne pathogen biofilms. Int J Agric Biosci. 2024;13(4):656-668.
15. Sultana MA, Habib MA, Amin MN, Sabuz SH, Salma U, Begum MD, Haque MA. Effects of yogurt on growth performance, carcass traits, lipid profile and fecal microbial load of broiler chickens. Int J Agric Biosci. 2025;14(1):50-58.
16. Pratama AR, Adli DN, Sholikin MM, Sitaresmi PI. Exploration of Saccharomyces cerevisiae as a feed additive in poultry. Int J Agric Biosci. 2026;15(1):57-68.
17. Diop SD, Inci A, Kizgin AD, Duzlu O. Understanding One Health and zoonosis: A systematic review with a bibliometric analysis of Turkish research and global perspectives (1974–2023). Kafkas Univ Vet Fak Derg. 2025;31(3):333-340.
18. Aydin E. From deadly to life-saving effects: Antimicrobial and antibiofilm effects of Tarantula cubensis extract on bacterial and fungal pathogens. Kafkas Univ Vet Fak Derg. 2025;31(1):59-67.
19. Yuksel HT, Seferoglu Y, Turkyilmaz S. Phylotypes, antibiotic resistance and biofilm formation determined in enteropathogenic Escherichia coli isolates in diarrheic cats. Kafkas Univ Vet Fak Derg. 2024;30(5):677-685.