



ISSN: 2456-2912

NAAS Rating (2026): 4.61

VET 2025; SP-11(4): 84-88

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www.veterinarypaper.com

Received: 14-01-2026

Accepted: 18-02-2026

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Molecular and antibiogram analysis of Shiga toxin-producing *E. coli* in swine farms

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DOI: <https://www.doi.org/10.22271/veterinary.2026.v11.i4Sb.3186>

Abstract

Shiga toxin-producing *Escherichia coli* (STEC) is critical to animal health and productivity inflicting economic losses, while also heightening the risk of zoonotic transmission to humans. In swine, it causes post-weaning diarrhea, dehydration and edema disease, resulting in mortality and reduced productivity, which leads to substantial economic losses. This study isolated and characterized STEC from 550 samples collected from ten organized swine farms, including oral, rectal and fecal samples from piglets, weaners, adults, and farmers. *E. coli* was molecularly characterized through PCR in 45.09% of the samples, with 30 identified as STEC and 10 confirmed as *E. coli* O157. The virulence genes *stx2* (90.32%), *stx1* (6.45%), *eaeA* (6.45%), and *hlyA* (12.90%) were found in STEC isolates. *Stx2* was linked to greater virulence and higher risk of severe infections. All STEC isolates were multidrug-resistant, with 6.66% being ESBL producers, highlighting the growing threat of antibiotic resistance.

Keywords: *E. coli*, farms, STEC, antibiotic resistance

1. Introduction

Shiga toxin-producing *Escherichia coli* (STEC) is responsible for numerous outbreaks of gastrointestinal diseases, including haemorrhagic colitis (HC) and haemolytic uremic syndrome (HUS), resulting in diarrhoea in both humans and animals, thus posing a significant threat to public health. Swine can serve as reservoirs for STEC and transmission to humans can occur through direct contact, environmental contamination, or consumption of contaminated products (Fratamico *et al.* 2008, Rajkhowa and Sarma 2014)^[1, 2]. Due to close interactions between farmers, livestock, and the farm environment, foodborne diseases caused by multidrug-resistant (MDR) STEC strains has a significant global public health concern. Limited information is available on epidemiology of STEC in pig farms, pig farmers/handlers and pig farm environment in India.

2. Materials and Methods

2.1 Sample collection

The study collected 550 samples from ten organized swine farms (Table-1), which included three samples each of feed and water, as well as 15 oral swabs, 15 rectal swabs, and 15 fecal samples representing three developmental stages: piglets, weaners, and adults from each farm. Additionally, one nasal swab, one stool sample, one urine sample, and one hand swab from pig farmers were collected from each farm. All samples were aseptically collected using cotton swabs and then transferred to the laboratory for analysis.

2.2 Isolation and identification of *E. coli*

The swabs were enriched in Tryptone Soy Broth (TSB) and incubated at 37 °C for 24 h. After enrichment, the samples were streaked onto MacConkey agar and incubated at 37 °C for 18-24 h, where lactose-fermenting colonies were subcultured onto Eosin Methylene Blue (EMB) agar to observe the characteristic metallic sheen. Pure colonies were selected, inoculated onto nutrient agar slants for biochemical tests and then prepared for DNA extraction through boil and snap chilling method. Presumptive colonies of *E. coli* were confirmed molecularly by *E.*

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coli species-specific PCR, targeting a region of E16S rRNA gene. PCR amplification was optimized according to Russel and Sambrook³ 2001. The PCR products were subjected for 1% agarose gel electrophoresis with ethidium bromide dye and visualized by gel documentation system (BIORAD).

Table 1: Sampling details of swine farms and retail outlets

S. No	Farm	Location
1	A	Veeravalli farm, Krishna District
2	B	LFC, Gannavaram, Krishna district
3	C	Department of Livestock Products Technology, Gannavaram, Krishna district
4	D	Pig breeding farm, mutyala, Jaggayyapet, Krishna district
5	E	Pig farm, Nuziveed, Eluru District
6	F	AICRP, Tirupati district
7	G	Pig farm, Vizianagaram District
8	H	Pig farm, Buddavaram, Krishna district
9	I	Akshay pig farm, Pamurru, Prakasham district
10	J	Pig farm, Chilakaluripet, Guntur district

2.3 Detection of virulence genes

All the *E. coli* isolates were tested for haemolytic activity and biofilm formation on blood agar supplemented with 5% sheep blood and Congo red agar, respectively (Katongole *et al.*, 2020) [4]. All the confirmed *E. coli* isolates were screened for STEC-associated virulence genes using four set of primers i.e., *stx1*, *stx2*, *eaeA* and *hlyA* (Table-2) by multiplex PCR (m-PCR). The cyclical conditions were followed according to Paton and Paton (1998) [5]. The PCR products were subjected for 2% agarose gel electrophoresis with ethidium bromide dye and visualized by gel documentation system (BIORAD).

2.4. Molecular detection of *E. coli* O157

The confirmed STEC isolates were cultured on CT-SMAC (Sorbitol Mac Conkey Agar supplemented with Cefixime Tellurite) agar and incubated at 37°C, 24h. The sorbitol non-fermenting *E. coli* O157 isolates were then subjected for PCR analysis (Desmarchelier *et al.* 1998) [6].

2.5. Antimicrobial resistance profile

All the confirmed STEC isolates were subjected to in-vitro antibiotic sensitivity test, carried out by Kirby Bauer disc diffusion method to assess the resistance/sensitivity patterns against 12 different antimicrobial drugs like amikacin (30 µg), nitrofurantoin (5 µg), novobiocin (10 µg), cefazolin (30 µg), co-trimoxazole (25 µg), ciprofloxacin (5 µg), chloramphenicol (30 µg), colistin (10 µg), piperacillin/tazobactam (100/10 µg), imipenem (10 µg), tigecycline (15 µg) and tetracycline (30 µg). A standardized inoculum (0.5 Mc Farland) was spread on Mueller Hinton agar (MHA) and the diameter of the inhibition zones was measured and isolates were interpreted as sensitive, intermediate resistant and resistant based on Clinical Laboratory Standards Institute (CLSI 2021) [7].

2.6. Molecular detection of β-lactamase genes

All the positive STEC isolates were subjected to a m-PCR assay (*bla*_{TEM}, *bla*_{SHV} and *bla*_{OXA}) and one uniplex PCR assay (*bla*_{AmpC}) which were standardized for the detection of β-lactamase genes (Table-2) in STEC isolates. The cyclical conditions for m-PCR and uniplex PCR assay were followed according to Dallenne *et al.* 2010 [8] and Manoharan *et al.* 2012 [9], respectively. The PCR products were subjected for 1.5% agarose gel electrophoresis with ethidium bromide dye

and visualized by gel documentation system (BIORAD).

Table 2: Oligonucleotide primers used for detection of STEC isolates

Target Gene	Primer	Sequence of the primer (5'-3')	Amplicon size (bp)
<i>stx1</i>	<i>stx1</i> : F	ATAAATCGCCATTTCGTTGACTAC	180
	<i>stx1</i> : R	AGAACGCCCACTGAGATCATC	
<i>stx2</i>	<i>stx2</i> : F	GGCACTGTCTGAAACTGCTCC	255
	<i>stx2</i> : R	TCGCCAGTTATCTTGACATTCTG	
<i>eaeA</i>	<i>eaeA</i> : F	GACCCGGCACAAGCATAAAGC	384
	<i>eaeA</i> : R	CCACCTGCAGCAACAAGAGG	
<i>hlyA</i>	<i>hlyA</i> : F	GCATCATCAAGCGTACGTTCC	534
	<i>hlyA</i> : R	AATGAGCCAAGCTTGTTAAGCT	
<i>rfb</i> O157	O157AF	AAGATTGCGTGAAGCCTTTG	497
	O157AR	CATTGGCATCGTGTGGACAG	
MultiTSO-T	<i>bla</i> _{TEM}	CATTTCCGTGTCGCCCTTATTC CGTTCATCCATAGTTGCCTGAC	800
MultiTSO-T	<i>bla</i> _{SHV}	AGCCGCTTGAGCAAATTAAC ATCCCGCAGATAAATCACCAC	713
MultiTSO-T	<i>bla</i> _{OXA}	GGCACCAGATTCAACTTTCAAG GACCCCAAGTTTCTCTGTAAGTG	564
AmpC	<i>bla</i> _{AmpC}	CCCCGCTTATAGAGCAACAA TCAATGGTCGACTTCACACC	631

Results and Discussion

Based on cultural and biochemical characteristics, 65.81% (362/550) of samples were identified as *E. coli*, with 45.09% (248/550) confirmed by PCR (Fig. 1). This finding is consistent with earlier studies by Begum *et al.* (2014) [10] and VinodhKumar *et al.* (2019) [11], which reported *E. coli* prevalence rates of 59.33% and 58.40%, respectively, in pigs. In this study, we assessed two virulence factors such as haemolytic activity and biofilm formation, using blood agar and Congo red agar, respectively. Haemolysis, a key pathogenic feature of STEC, is linked to Shiga toxin production, contributing to conditions like bloody diarrhea and HUS. We found 1.61% (4/248) of *E. coli* isolates exhibited haemolytic activity on sheep RBC, which contrasts with Meng *et al.* (2014) [12] and Baranzoni *et al.* (2016) [13] who reported 15% and 7%, respectively. The discrepancy of results may be due to methodological variations, geographical and environmental factors. The ability to form biofilm was assessed using Congo red agar, where 33.21% (92 out of 248) of the *E. coli* isolates exhibited black crystalline colonies. In contrast, Dimitrova *et al.* (2021) [14] reported a significantly higher percentage 93.75% of *E. coli* isolates displaying biofilm activity when evaluated using the microtiter plate assay. This discrepancy could be attributed to the lower specificity of the Congo red agar method compared to the microtiter plate assay. Through m-PCR, 30 isolates (12.09%, 30/248) were positive for any one of the virulence genes (Fig. 2). Among the 30 STEC isolates, *stx2* (93.33%, 28/30) was the predominant gene, followed by *hlyA* (13.33%, 4/30) and *stx1* and *eaeA* (6.66%, 2/30 each). Combination of *stx2* and *hlyA* was detected in two isolates from farm D (rectal swab of adult and faecal sample from weaner). Combination of *stx1*, *stx2* and *hlyA* was detected in faecal sample of piglet from farm B. Combination of *stx2*, *eaeA* and *hlyA* was detected in one isolate from farm D (rectal swab of adult). Only *stx2* was detected in three isolates from farm B, nine isolates from farm D, 10 isolates from farm E and two isolates from farm H. Only *eaeA* was detected in only one isolate from farm H. The virulence gene profile of STEC isolates revealed that the *Stx2* gene was the predominant gene than *stx1*, which was in line with the findings of Begum *et al.* (2014) [10] and Kataria *et al.* (2014) [15]. The *Stx2* gene is more predominant because it is

associated with higher virulence and is more cytotoxic to endothelial cells. STEC genes were exclusively identified in four swine farms, namely farms B, D, E and H. The occurrence of STEC genes were highest in farm D (40%, 12/30) followed by farm E (33.33%, 10/30), farm B (16.66%, 5/30) and farm H (10%, 3/30). The high occurrence in some farms may be due to the lack of hygiene practices, biosecurity measures, manure management and high population density. Among the different samples, the occurrence of STEC was highest in feed sample (100%, 1/1), followed by water sample (18.21%, 2/11), rectal swabs (16.88%, 13/77) and faecal samples (15.21%, 14/92) of *E. coli* isolates. In water samples, STEC showed a prevalence rate of 18.21%. However, lower prevalence rates were reported by Puii *et al.* (2019) [16] and Kobayashi *et al.* (2003) [17], at 4.4% and 2.02%, respectively. This variation could be attributed to differences in environmental conditions, water treatment methods, and agricultural practices. Among the different developmental stages, the average rate of STEC prevalence was highest in adults (16.90%; 12/71) followed by weaners (12.98%; 10/77) and piglets (6.94%; 5/72) of *E. coli* isolates. These observations were coincided with Baldo *et al.* (2020) [18] who isolated more *stx2e*-producing *E. coli* isolates in healthy adult pigs rather than weaners and piglets. Similarly, Fratamico *et al.* (2004) [19] and Remfry *et al.* (2020) [20] isolated STEC in faecal samples at the rate of 70% and 29.7% in finisher pigs, respectively. The high prevalence in adults may be due to the influence of environment stressors, management practices and dietary factors in adults. STEC was not detected in swine oral swabs, pig farmer stool and urine sample, hand and nasal swab.

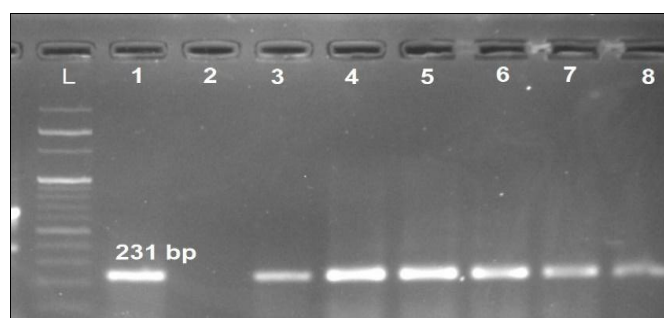
Among the 30 STEC isolates, 10 (33.33%) were identified as *E. coli* O157 using PCR (Fig. 3). Overall *E. coli* O157 prevalence was 4.03% (10/248) which was in slight agreement with the results of Keen *et al.* (2006) [21] who reported the prevalence of 1.2%. On the contrary, Fratamico *et al.* (2008) [1] isolated none from finisher pigs. The variation in *E. coli* O157 prevalence in pigs across different locations could be attributed to disparities in surveillance and detection methods, as well as differences in husbandry practices and geographical locations.

Highest resistance was observed against cefazolin and co-trimoxazole (100%; 30/30 each) followed by tetracycline, nitrofurantoin and novobiocin (93.3%; 28/30 each), colistin (90.0%; 27/30), chloramphenicol (70.0%; 21/30), ciprofloxacin (50.0%; 15/30), amikacin (33.3%; 10/30). STEC isolates showed highest sensitivity against imipenem and tigecycline (96.6%; 29/30 each) followed by piperacillin/tazobactam (66.6%; 20/30). In the present study, the resistance towards co-trimoxazole (100%) and tetracycline

(93.3%) which were in slight agreement with the findings of Baldo *et al.* (2020) [18] who reported the resistance towards tetracycline (98.25%) and co-trimoxazole (82.17%) and Meng *et al.* (2014) [12] who reported the resistance rates of 79.57% and 73.12% to co-trimoxazole and tetracycline, respectively. Resistance rates of novobiocin (93.3%) and amikacin (33.3%) were in line with the observations of Kataria *et al.* (2014) [15] who reported the resistance of 100% and 40% to novobiocin and amikacin, respectively. It may be due to the excessive use of antibiotics in the swine industry for therapeutic and growth promoting purposes. All the 30 STEC isolates (100%) showed MDR. The high prevalence of MDR bacteria in pigs can be attributed to the fact that pigs are often reared in intensive farming environments, where antibiotics are extensively utilized for purposes such as promoting growth, preventing diseases and providing treatment. In the current investigation, the MAR index of all MDR STEC isolates (100%) exceeded 0.2, while Vinodh Kumar *et al.* (2019) reported that 13.75% of isolates revealed MAR index > 0.2 and 2.64% isolates had MAR of 1.0. The higher percentage of MDR STEC isolates with MAR index > 0.2 in the current study, might be due to non-judicious use of antibiotics for longer period in both animals and humans in the study area.

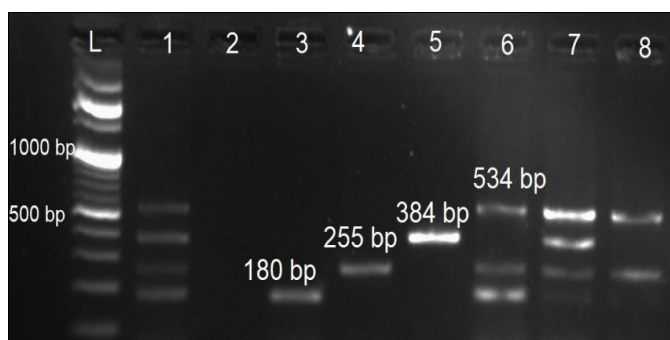
All the eight confirmed ESBL producers were subjected to m-PCR for detection of ESBL genes, where *bla*SHV was the only gene detected in two isolates, one from faecal sample of weaner obtained from farm D (DFW1) and other from faecal sample of weaner obtained from farm E (EFW3) (Fig.4). The findings align with Puii *et al.* (2019) [16] noted a higher incidence of *bla*SHV (6.03%) in piglets compared to other β -lactamase genes. However, *bla*TEM was the predominant gene in many studies (Vinodh Kumar *et al.* 2019, Tamta *et al.* 2020 [22] and Naidu *et al.* 2021) [23]. In the current study, none of the STEC isolates were positive for *bla*TEM, *bla*OXA genes. In uniplex PCR assay, six isolates were positive for *bla*AmpC gene (Fig. 5).

STEC infections in swine can lead to various clinical manifestations, including diarrhea, dehydration, edema disease and in severe cases, mortality. This not only compromises animal welfare but also raises economic implications for swine producers due to decreased productivity and increased veterinary costs. In the current study, the presence of STEC was confirmed in pig farms, farm environment and pork. Pigs can be a source of STEC which poses a potential public health risk as an emerging foodborne pathogen, with zoonotic potential. Moreover, our study revealed high antibiotic resistance patterns in STEC isolates and were MDR, a threat to both animal and public health which may provide a vector for the spread of these opportunistic pathogen.



Lane L: DNA ladder (100 bp); Lane 1: Positive control (ATCC *E. coli* O157 43888); Lane 2: Negative control; Lane 3-8: Isolates were positive for *E. coli* 16S rRNA gene (231 bp).

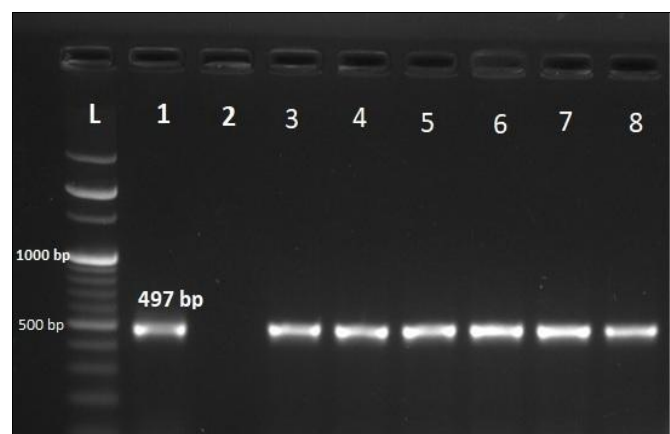
Fig 1: Gel photograph of *E. coli* species-specific gene



Lane L: Control - DNA ladder (100 bp); Lane 1: Positive control (Known DNA sample having all 4 virulence genes); Lane 2: Negative control; Lane 3: STEC isolate showing *stx1* gene (BFP2); Lane 4: STEC isolate showing *stx2* gene (EFW3); Lane 5: STEC isolate showing *eaeA* gene (HRW5); Lane 6: STEC isolate (BFP4) with three genes *stx1*; *stx2* and *hlyA*; Lane 7: STEC isolate (DRA1) with three genes *stx2*; *eaeA* and *hlyA* and Lane 8: STEC isolate with two genes *stx2* and *hlyA* (DRA3).

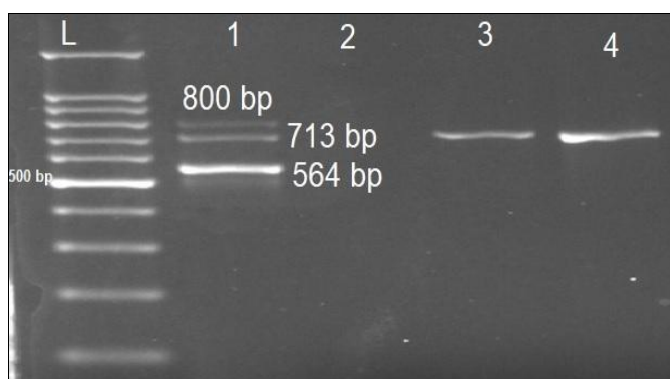
BFP2: Faecal sample of piglet₂ (Farm b); EFW3: Faecal sample of weaner₃ (Farm E); HRW5: Rectal swab of weaner₅ (Farm H); DRA1: Rectal swab of adult₁ (Farm D); BFP4: Faecal sample of piglet₄ (Farm b); DRA3: Rectal swab of adult₃ (Farm D)

Fig 2: Gel photograph of PCR assay targeting STEC virulence genes



Lane L: Control - DNA ladder (100 bp); Lane 1: Positive control (*E. coli* O157:H7 ATCC 43888); Lane 2: Negative control; Lane 3-8: Positive for *E. coli* O157:H7 (497 bp).

Fig 3: Gel photograph of *E. coli* O157



Lane L: Control - DNA ladder (100 bp); Lane 1: Positive control (Known DNA standard for *bla*_{TEM} (800 bp), *bla*_{SHV} (713 bp) and *bla*_{OXA} (564 bp)); Lane 2: Negative control; Lane 3 & 4: STEC isolates (DFW1 and EFW3) positive for *bla*_{SHV} (713 bp). DFW1: Faecal sample of weaner₁ (Farm D); efw3: Faecal sample of weaner₃ (Farm e)

Fig 4: Gel photograph of ESBL genes



Lane L: Control - DNA ladder (100 bp); Lane 1: Positive control (Known DNA standard for *bla*_{AmpC} (631 bp)); Lane 2: Negative control; Lane 3-6: STEC isolates (BFA1; DRP1; DRA3 and ERW3) positive for *bla*_{AmpC} (631 bp).

BFA1: Faecal sample of adult₁ (farm B); DRP1: Rectal swab of piglet₁ (farm D); DRA3: Rectal swab of adult₃ (farm D); ERW3: Rectal swab of weaner₃ (farm E).

Fig 5: Gel photograph of β -lactamase gene - *bla*_{AmpC}

Conflict of Interest

Authors have no conflict of interest in this study.

Author's Contribution

SS: Involved in investigation, data generation, preparing original draft; SN and ST: Engaged in conceptualization, supervision and final editing.

Acknowledgement

Authors would like to express gratitude to the Sri Venkateswara Veterinary University, Tirupathi for conducting the research.

Financial Support

Not available

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How to Cite This Article

Siripalli S, Nelapati S, Tumati SR. Molecular and antibiogram analysis of Shiga toxin-producing *E. coli* in swine farms. *International Journal of Veterinary Sciences and Animal Husbandry*. 2026;SP-11(4):84-88.

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