# Shiga toxin-producing *Escherichia coli* (STEC) detection and characterization among US swine herds

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## Introduction

Shiga toxin (Stx2e)-producing *Escherichia coli* (STEC) is one of the most important *E coli* pathotypes in animals and is the etiologic agent of edema disease (ED) in pigs.[1] ED has been reported to have a mortality rate from 50- 90% in herds and can often reoccur.[2] ED still occurs, despite vaccination attempts.[3] A review of historical diagnostic data from a veterinary diagnostic laboratory, revealed that approximately 19% of *E coli* isolates from pigs genotyped from 2009 to 2019 were positive for the Stx2e gene. This study investigated the presence of fimbrial and toxin genes from hemolytic *E coli* isolates of swine to estimate the frequency of STEC in US swine herds. Isolates were further characterized by colony morphology on blood agar (BA) plates and antimicrobial susceptibility.

## Materials and methods

Rectal swabs were collected from commercial pigs suspected to be clinically affected by ED (neurologic deficits). Pigs ranged from three to nine weeks of age and were located at 47 different sites; each site was strategically sourced from 13 production companies across the United States. Five clinical pigs were sampled from three, non-adjacent pens for a total of 15 samples per site. Individual pig care criteria was used to identify acute and subacute pigs displaying the following clinical signs: depression, anorexia, central nervous system deficits and/or diarrhea. The producer captured ancillary information including site information, *E coli* history, clinical signs and individual fecal consistency and color. All samples were submitted to a veterinary diagnostic laboratory for bacterial isolation. Once *E coli* was isolated and identified, hemolytic isolates with different colony morphologies (rough, smooth, or smooth-mucoid) were selected to represent all positive pens from each site. This subset of isolates were further characterized by genotyping via polymerase chain reaction (PCR) and antimicrobial susceptibility testing (AST) via broth microdilution.

## Results and discussion

Two hundred and thirty-eight of the 690 isolates (34%) were hemolytic on BA, as expected for STEC isolates. One hundred and ten of the 238 hemolytic isolates (46%) were selected systematically by pen to have a mixture of colony morphology for PCR and AST. Twenty-one of the 110 isolates (19%) were characterized as STEC, 16 of the 21 isolates (76%) were PCR positive for the Stx2e toxin gene and the F18 fimbrial gene. The remaining five isolates carried the Stx2e toxin gene, but no fimbrial genes for attachment. The 16 isolates originated from 9 sites (19% of sites); none which vaccinated with a F18 *E. coli* vaccine. As indicated in Table 1, all 16 STEC (F18 + Stx2e) isolates had smooth or smooth-mucoid colony morphologies. AST results were similar to historic diagnostic susceptibility data with significant resistance to several labeled antimicrobials. If a producer has reoccurring *E coli* breaks with high mortality and neurological clinical signs considerations should include, utilizing a vaccine that contains a F18 component and incorporating AST results to guide antimicrobial selection. In congruence with historical diagnostic data, disease-capable STEC is present in approximately 15% of the herds sampled in this study. US veterinarians should focus on *E coli* isolates that are hemolytic on BA with smooth and smooth-mucoid colony morphologies when considering further diagnostic evaluation of suspected STEC cases. Resistance patterns continue to support the need for additional management strategies.

## References

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**Table 1:** Morphology, genotyping and antimicrobial susceptibility results of the STEC (F18 + Stx2E) isolates

| Number of STEC isolates (F18 + Stx2E) | | | Number of susceptible STEC isolates (F18 + Stx2E) |
|---|---|---|---|
| **Morphology** | | **Antimicrobial** | |
| Smooth | 7/16 | Ampicillin | 9/16 |
| Mucoid | 0/16 | Ceftiofur | 16/16 |
| Smooth-mucoid | 9/16 | Clindamycin | 0/16 |
| **Gene** | | Danofloxacin | NI* |
| STb (toxin) | 14/16 | Enrofloxacin | 3/16 |
| STa (toxin) | 9/16 | Florfenicol | 6/16 |
| K99 (pilus) | 0/16 | Gamithromycin | NI* |
| LT (toxin) | 14/16 | Gentamicin | 5/16 |
| F18 (pilus) | **16/16** | Neomycin | 4/16 |
| 987P (pilus) | 0/16 | Penicillin | 0/16 |
| K88 (pilus) | 0/16 | Sulfadimethoxine | 2/16 |
| F41 (pilus) | 0/16 | Spectinomycin | NI* |
| Stx2e (toxin) | **16/16** | Tetracycline | 0/16 |
| EAST1 (toxin) | 7/16 | Tiamulin | 0/16 |
| Stx1 (toxin) | 0/16 | Tildpirosin | NI* |
| Stx2 (toxin) | 16/16 | Tilmicosin | 0/16 |
| AIDA (adhesin) | 0/16 | Trimethoprin/Sulphametoxazole | 0/16 |
| EAEA (adhesin) | 0/16 | Tulathromycin | NI* |
| PAA (adhesin) | 2/16 | Tylosin | NI* |

* NI = no interpretation available based on antimicrobial, organism, species, and tissue combination