Experimental and Field Studies of *Escherichia coli* O157:H7 in White-Tailed Deer

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Received 25 May 2000/Accepted 19 December 2000

Cattle are considered a major reservoir of *Escherichia coli* O157:H7 because consumption of undercooked meat products from cattle has been associated with many human infections and because the bacterium has been detected in bovine feces throughout the United States, Canada, and Europe (7). However, the possible role of wild deer in the epidemiology of *E. coli* O157:H7 began to receive attention as early as 1988 when *E. coli* O157:H7 bacteria were recovered from the gastrointestinal tracts of deer necropsied from 4 to 26 DPI, attaching and effacing lesions were not apparent in any deer. Results are similar to those of inoculation studies in calves and sheep. In field studies, *E. coli* O157 was not detected in 310 fresh deer fecal samples collected from the ground. It was detected in feces, but not in meat, from 3 of 469 free-ranging deer in 1997. In 1998, *E. coli* O157 was not detected in 140 deer at the single positive site found in 1997; however, it was recovered from 13 of 305 dairy and beef cattle at the same location. Isolates of *E. coli* O157:H7 from deer and cattle at this site differed with respect to pulsed-field gel electrophoresis patterns and genes encoding Shiga toxins. The low overall prevalence of *E. coli* O157:H7 and the identification of only one site with positive deer suggest that wild deer are not a major reservoir of *E. coli* O157:H7 in the southeastern United States. However, there may be individual locations where deer sporadically harbor the bacterium, and venison should be handled with the same precautions recommended for beef, pork, and poultry.

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bers from the large intestine, although pathological lesions were not apparent in steers and adult cattle (4), and from the forestomachs and colon of weaned calves in which lesions also were absent (2).

The objectives of our laboratory studies were to evaluate the clinical response, fecal shedding, sites of bacterial localization, and associated lesions in a small group of young white-tailed deer inoculated with *E. coli O157:H7*. The protocols of a previous bovine trial (2) were followed to facilitate comparison of deer inoculations with calf inoculations. In the field study, we cultured feces of free-ranging white-tailed deer in the southeastern United States, primarily in Georgia. Deer fecal samples were collected from the ground during summer, which, in some reports, is the season of highest prevalence of fecal shedding of *E. coli O157:H7* by domestic cattle (8, 9) and sheep (13). Samples also were collected directly from deer killed by hunters during the fall hunting season, which is the time of greatest human exposure to wild deer. Additionally, we cultured uncooked venison from deer with *E. coli O157:H7* in their feces. During the second year of the study, wild deer and domestic cattle at the single positive site found in the first year were cultured for *E. coli O157:H7*, and genomic subtypes of deer and cattle isolates were compared.

[Portions of this work were presented at the Third International Symposium and Workshop on Shiga Toxin (Vero cytotoxin)-Producing *Escherichia coli* Infections (J. R. Fischer, M. P. Doyle, T. Zhao, C. A. Brown, and C. T. Sewell, abstr. V233/II, 1997).]

**MATERIALS AND METHODS**

**Experimental design.** Nine 3-month-old white-tailed deer were hand raised on milk replacer prior to their arrival at the University of Georgia. They subsequently were weaned and were fed a commercial pelleted deer ration (Checkers; Purina Mills, St. Louis, Mo.) and hay ad libitum during the 9-day acclimation period and throughout the study. Two deer each were housed in separate bio-safety level 2 concrete rooms with floor drains to facilitate daily cleaning with water. The animals remained clinically normal during the acclimation period. All deer were screened twice and found negative by culture for fecal *E. coli O157:H7* during the acclimation period. Blood samples were collected the day after arrival, and a sandwich enzyme-linked immunosorbent assay (ELISA) was performed on serum from each deer for antibodies against *E. coli O157:H7*. On day 10 following arrival, six deer were orally administered 10^8 CFU of nalidixic acid-resistant *E. coli O157:H7*. Two deer received a similar inoculum of nalidixic acid-resistant, nonontoxic *E. coli*, and one received no inoculum. To assess contact transmission, the uninoculated deer was cohoused with an inoculated deer shedding *E. coli O157:H7* from 12 to 26 days postinoculation (DPI). All deer were examined daily for signs of infection including diarrhea, lethargy, and anorexia. Fecal samples were collected per rectum from all deer at 1- to 3-day intervals throughout the 4-week trial. Blood samples were collected for serologic testing at 7-day intervals following the day of inoculation and at the time of euthanasia.

Deer were necropsied at intervals during the trial. Of deer inoculated with *E. coli O157:H7*, two were necropsied at 4 DPI to determine whether attaching and effacing lesions were present, one was necropsied at 11 DPI, two were necropsied at 25 DPI, and one was necropsied at 26 DPI along with the uninoculated contact transmission animal 14 days postcontact (DPC). One deer inoculated with nonontoxic *E. coli* was necropsied at 7 DPI, and the other was necropsied at 26 DPI.

**Bacterial inoculum.** Deer received 10^8 CFU of a five-strain mixture of enterohemorrhagic *E. coli O157:H7* consisting of one venison isolate (E0139), three cattle isolates (E009, E0108, and E0122), and one human isolate (932). Inoculum preparation was similar to that of a previous study (2). Briefly, individual *E. coli O157:H7* strains and the nonontoxic strain were selected for nalidixic acid resistance. Nonontoxic strains did not produce Shiga toxins and were negative for fluorescent antibody tests for pilus antigens F41 and K99. Each strain of nalidixic acid-resistant *E. coli* was grown individually in tryptic soy broth (TSB; Difco Laboratories, Detroit, Mich.) containing nalidixic acid (50 μg/ml), sedimented by centrifugation, and washed three times as described elsewhere (2). Phosphate-buffered saline (PBS) was added to sedimented bacteria in an amount needed to obtain an optical density of 0.5 at 630 nm, representing approximately 10^9 CFU/ml, and 0.8 ml of each of the five strains of *E. coli O157:H7* was mixed in 16 ml of sterilized skim milk immediately prior to direct inoculation into individual nursing bottles. The concentration of each strain and the final *E. coli O157:H7* concentration in the skim milk were confirmed by standard dilution plating on tryptic soy agar and MacConkey sorbitol agar (Difco) containing 50 μg of nalidixic acid per ml (MSA-NA) in duplicate.

** Necropy of deer.** Animals were euthanatized with intravenous sodium pentobarbital, and a complete necropy was performed. Two deer that died spontaneously were necropsied shortly after death. At necropy of all deer, the gastrointestinal tract (GI tract) was clamped at the esophagus and rectum and removed in toto. Five-centimeter lengths of the duodenum, midjejunum, ileum, cecum, spiral colon, and descending colon were double clamped with plastic bands to allow enumeration of *E. coli O157:H7* bacteria in the tissues and contents of each section without cross-contamination. Sections and contents of rumen, reticulum, omasum, and abomasum also were collected for culture and enumeration of *E. coli O157:H7* bacteria. Samples of all GI tissues as well as visceral organs and lymph nodes were placed in 10% buffered formalin for histologic examination. Histopathology and immunohistochemistry. Formalin-fixed tissues were embedded in paraffin, sectioned at 5 μm, stained with hematoxylin and eosin, and examined by light microscopy. Paraffin-embedded, formalin-fixed sections of GI tract tissues from which large populations (>3 logCFU/g) of *E. coli O157:H7* were cultured were selected and treated in an automated immunohisto staining machine to identify *E. coli O157:H7*. A positive control slide consisted of an enteric section from a neonatal calf with experimental *E. coli O157:H7* infection (5), and a negative control consisted of an intestinal section from an uninoculated deer. Tissues were deparaffinized in xylene for 15 min, rehydrated through graded alcohols, rinsed in dilute citrate buffer (Antigen Retrieval Citra 10× concentrate; Biogenex Laboratories, San Ramon, Calif.), and then steamed in citrate buffer in a covered steam chamber for 20 min. Sections then were placed into an automated immunohisto staining machine, rinsed in citrate buffer, covered with blocking antibody (rabbit serum; Vector Laboratories, Burlingame, Calif.) for 5 min, rinsed in buffer, and then covered with *E. coli O157:H7*-specific goat antiserum (0.1 μg/ml; Kirkegaard and Perry Laboratories, Inc., Gaithersburg, Md.) and incubated for 30 min. Sections were rinsed in buffer and covered with rabbit biotinylated antibody (Supersensitive link; Biogenex Laboratories) for 25 min. Sections were rinsed in buffer and covered with 3% hydrogen peroxide for a total of 7.5 min, rinsed, and then covered with avidin-biotin complex (Elite ABC kit; Vector Laboratories) according to the manufacturer's instructions. After rinsing, sections were treated with 3,3′-diaminobenzidine (DAB peroxi- dase substrate; Sigma Chemical Co., St. Louis, Mo.) per the manufacturer's instructions for a total of 12 min, rinsed, and stained with Gillis 2 hematoxylin (Surgipath Medical Industries, Richmond, Ill.) for 3 min. Sections were rinsed, dehydrated, cleared, mounted (Floxetx; Lerner Laboratories, Pittsburgh, Pa.), and examined by light microscopy.

**Serologic testing of experimental deer.** ELISA was performed in 96-well poly- styrene immunosorbent-immunoperoxidase-immunoperoxidase plates (GIBCO, Grand Island, N.Y.). Each well was coated with 10^6 formaldehyde-killed *E. coli O157:H7* whole cells in 100 μl of 50 mM carbonate buffer, pH 9.6, by incubation at 25°C overnight with agitation (150 rpm). The plate was washed four times with 50 mM Tris, pH 7.4, containing 150 mM NaCl (Tris-buffered saline [TBS]), and remaining binding sites were blocked with milk diluent-blocking solution (Kirkegaard and Perry Laboratories, Inc.) for 1 h at 37°C. The blocking agent was removed, and 100 μl of double-diluted deer serum per well was added and incubated at 37°C for 1 h. Serum from a fetal deer was used as a negative control. The wells were washed four times with TBS containing 0.05% Tween 20 (TBS-T), and 100 μl of goat anti-deer immunoglobulin M and G conjugated to alkaline phospha- tase (diluted 1:800 in TBS-T; Kirkegaard and Perry Laboratories, Inc.) per well was added and incubated at 37°C for 1 h. Wells were washed four times with TBS-T, and 100 μl of p-nitrophenylphosphate in 1 M 2-amino-2-methyl-1-pro-panol (Sigma), pH 9.9 (1 mg/ml), was added to each well. The plate was incubated at 37°C for 1 h, and the optical density at 405 nm of each well was determined with an EL 312e microplate reader (Bio-Tek Instrument, Winooski, Vt.). An optical density reading of 0.1 above the negative control was considered positive for that dilution.

**Collection of samples from deer and cattle in the field.** In 1997, approximately 60 fresh deer fecal samples were collected from the ground in July-August at each of five public wildlife areas and directly from hunter-killed deer in October to December at the same five areas plus an additional site (sites GA1 to GA6). The wildlife areas were widely scattered throughout Georgia. Fecal samples also
were collected directly from 98 deer from April to December 1997 in Arkansas, Georgia, Mississippi, South Carolina, Tennessee, and West Virginia. Age and sex were recorded for all individually sampled deer. Frozen meat samples were obtained from deer that had E. coli O157:H7 in feces.

In November and December of 1998, fecal samples were collected per rectum from hunter-killed deer at site GA4, where E. coli O157:H7 was detected in deer in 1997. Site GA4 consisted of an approximately 20,000-acre public wildlife area adjacent to property on which a dairy herd and a beef herd were present. From November 1998 to February 1999, fecal samples were obtained per rectum from cattle belonging to the two herds at site GA4.

**Isolation of E. coli O157:H7 from feces and tissue samples.** Techniques used to enumerate E. coli O157:H7 bacteria in deer feces and GI tissues were similar to those previously described (2). Briefly, 10 g of feces was placed in 15 ml of Cary-Blair medium (Remel Co., Lenexa, Kans.), held at 5°C, and transported to the laboratory within 72 h. One gram of feces was serially diluted to 10^−3 CFU/g, and the dilution was plated in duplicate on MSA-NA and incubated. GI tissue samples were weighed and washed gently in sterile PBS to remove the contents of the tubular organs. The rinsed tissue samples were resuspended in PBS and homogenized. Tissue sample suspensions and content samples were plated separately in quadruplicate on MSA-NA plates. When the direct plating technique was negative, selective enrichment was employed (2).

For samples collected from wild deer and domestic cattle, isolation techniques were similar to those used in previous surveys of domestic cattle (22). Briefly, fecal samples were plated in Cary-Blair medium as described above and then plated and incubated on sorbitol-MacConkey agar plates (Unipath, Oxoid Division, Ogdensburg, N.Y.) containing 0.1% 4-methylumbelliferyl-β-D-glucuronide (Sigma Chemical Co.). For enrichment, samples were placed in TSB containing novobiocin and incubated at 37°C. Following incubation, a sandwich ELISA using a specific monoclonal antibody against E. coli O157:H7 was performed on each enriched sample as described elsewhere (22). For immunomagnetic selective isolation, 1 ml of enrichment medium from ELISA-positive samples was incubated with 20 μl of immunomagnetic beads coated with antibody to E. coli O157 (anti-E. coli O157 Dynabeads; Dynal, Oslo, Norway) and processed according to the manufacturer's instructions. Sixteen samples of frozen ground venison, cubed steak, roast, and hindquarter from three feral-culture-positive deer were assayed for E. coli O157:H7. For each sample, 25 g of thawed meat was placed in 225 ml of enrichment broth and processed as described above for wild deer and cattle fecal samples.

**Characterization of E. coli O157:H7 isolates from laboratory and field studies.** Isolates were identified as E. coli O157 as described elsewhere (22). Latex agglutination assay-positive colonies were further characterized by an H7 anti-serum motility inhibition assay and by biochemical tests with the API 20E miniaturized diagnostic test (Analytab Products, Division of Sherwood Medical, Plainview, N.Y.). For determination of Shiga toxin production by the 16 cattle and deer E. coli O157 isolates obtained in field studies, isolates were tested by PCR using oligonucleotide primers for Stx1a (GAAGAAGTCGGTGATTACG), Stx1b (AGCAACGACGATTTAATAG), Stx2a (TTAACCACACCACGGCAGT), and Stx2b (GCTCAGATGCATCTGTG). The primers were based on published sequences for the stx1 and stx2 genes (16). The sizes of the amplified products were 130 bp for Stx1 primers and 346 bp for the Stx2 primers.

**RESULTS**

**Serologic and clinical response of experimental deer.** Antibodies against E. coli O157:H7 were not detectable by ELISA in deer prior to the trial, at weekly intervals following inoculation, or at the time of euthanasia. Deer did not exhibit anorexia or lethargy following inoculation with enterohemorrhagic or nontoxicogenic E. coli. Two deer that received E. coli O157:H7 developed mild nonhemorrhagic diarrhea. One deer (deer 9) had mild diarrhea from 1 to 14 DPI and subsequently had soft, unformed feces through 25 DPI. The other deer (deer 13) had mild diarrhea at 3 and 10 DPI and died spontaneously of a noninfectious digestive problem (pyloric stenosis) at 11 DPI. Additionally, one deer (deer 10) that received nontoxicogenic E. coli died spontaneously of a similar noninfectious digestive problem at 7 DPI. The spontaneous deaths of these animals underscore the husbandry difficulties associated with this species. The other six deer were clinically normal during the trial.

**Patterns of fecal shedding of E. coli O157:H7 by experimental deer.** Inoculated deer were shedding 3.5 to 5.1 (average, 4.3) log_{10} CFU of E. coli O157:H7/g of feces by 1 DPI. Fecal shedding of E. coli O157:H7 by deer decreased substantially during the first 10 DPI but was consistently or intermittently detectable in three of three deer for the remainder of the 26-day study (Fig. 1 and 2). Two cohoused deer (deer 1 and 2) were euthanatized and necropsied at 4 DPI after shedding 3.4 to 4.8 log_{10} CFU of E. coli O157:H7/g of feces at 1 and 3 DPI. Two cohoused deer (deer 9 and 6) shed decreasing numbers of E. coli O157:H7 bacteria until 12 and 17 DPI, respectively, and intermittent shedding of low numbers of E. coli O157:H7 bacteria (<10 CFU/g) was detectable only by enrichment culture through 25 DPI (Fig. 1).

In the contact transmission trial, shedding by the two un inoculated deer decreased during the first 10 DPI (Fig. 2). One deer (deer 13) died spontaneously of unrelated causes at 11 DPI. The remaining deer was shedding 1.9 log_{10} CFU/g of feces at 12 DPI when an un inoculated deer was introduced to the room for the remainder of the trial. By 2 DPI, the un inoculated deer was shedding 2.1 log_{10} CFU/g of feces, and it continued to shed from 1.6 to 3.2 log_{10} CFU/g through 14 DPI. After introduction of the un inoculated deer, fecal shedding of E. coli O157:H7 by the inoculated animal increased (from 1.9 to 3.9 log_{10} CFU/g), and it remained detectable by standard plating throughout the trial (Fig. 2).
Recovery of *E. coli* O157:H7 from experimental deer at necropsy. Inoculated deer were necropsied 4, 11, 25, and 26 DPI, and the uninoculated contact deer was necropsied 14 DPC. *E. coli* O157:H7 bacteria were enumerated in contents and washed tissues at 10 sites of the GI tract of all deer (Table 1). Moderate or high (1 to 3 or >3 log_{10} CFU/g, respectively) numbers of enterohemorrhagic or nontoxigenic *E. coli* were recovered from the contents and tissues of the forestomachs and the intestines of deer necropsied 4 to 11 DPI. Low populations (<10 CFU/g) of *E. coli* O157:H7 were detected using enrichment culture in the contents but not in the mucosa of the large intestines of two deer (deer 6 and 9) necropsied at 25 DPI (Table 1). Moderate to high populations of *E. coli* O157:H7 were found in contents of all sections of the GI tract except in the abomasal contents of the inoculated deer in the contact transmission trial at 26 DPI and in all sections except the abomasum, duodenum (detectable only using enrichment culture), and spiral colon of the uninoculated contact animal. A consistent finding for all deer was smaller populations of *E. coli* O157:H7 bacteria in washed tissue than in the contents of the corresponding GI tract site (data not shown). This difference ranged from 0.1 to 2.9 log_{10} CFU/g.

Gross, histological, and immunohistological findings for experimental deer. Two deer that died spontaneously had distended forestomachs and abomasums, and there was moderate pyloric hypertrophy and stenosis with a paucity of digesta distal to the pylorus. The deer inoculated with *E. coli* O157:H7 that died at 11 DPI also had mild multifocal intracorneal microabscessation of the reticulum mucosa. Additional gross or microscopic lesions, including attaching and effacing lesions suggestive of enterohemorrhagic *E. coli* infection, were not apparent in these animals. The gross and microscopic findings for these animals are consistent with pyloric stenosis that most likely was caused by dietary changes in the animals as they were weaned. Gross lesions were not apparent in deer that were euthanized and necropsied per protocol. Adherent bacteria were not observed on mucosal surfaces of hematoxylin-and-eosin- or immunohistochemically stained sections of the GI tract. Attaching and effacing lesions were not observed in any deer.

Characterization of *E. coli* O157:H7 isolates from experimental deer. PFGE patterns of *XbaI* endonuclease digests of genomic DNA from eight *E. coli* O157:H7 isolates obtained at necropsy on day 26 from two deer of the contact transmission trial were indistinguishable. This pattern matched the fingerprint of the venison strain (E0139) of the inoculum.

Prevalence and characteristics of *E. coli* O157:H7 in free-ranging deer and cattle. *E. coli* O157:H7 was isolated from 3 of 919 deer fecal samples. The bacterium was detected in feces of 3 of 609 (0.5%) individually sampled deer, but it was not found in 310 fresh fecal samples collected from the ground. *E. coli* O157:H7 was isolated from feces of 3 of 469 individually sampled deer in 1997 and in 0 of 140 deer at GA4 in 1998. The three positive animals (2.5- and 3.5-year-old males and a 0.5-year-old female) were among 77 hunter-killed deer sampled in 1997 and in 0 of 140 deer at GA4 in 1998. The three positive animals (2.5- and 3.5-year-old males and a 0.5-year-old female) were among 77 hunter-killed deer sampled during three consecutive days in November 1997 at site GA4. Isolates were obtained only after enrichment indicating less than 5 CFU/g of feces. All three *E. coli* O157:H7 isolates were positive for stx1 and stx2 genes. *E. coli* O157:H7 was not isolated from 16 venison samples from the three positive deer.

*E. coli* O157 was isolated from feces of 13 of 305 (4.3%)...
cattle in 1998-1999 at site GA4. The isolates were obtained from seven dairy and five beef cattle in November 1998 and from one beef animal in February 1999. Eleven isolates were _E. coli_ O157:H7 and had only _stx2_ genes. Two were _E. coli_ O157:NM; both were positive for _stx1_ genes, and one also had _stx2_ genes.

Characteristics of _E. coli_ O157 from deer and cattle are found in Table 2. Genomic DNA of all _E. coli_ O157 isolates from wild deer and cattle was evaluated by PFGE (Fig. 3). Two deer isolates had indistinguishable PFGE patterns; the third isolate was genetically distinct. Bovine isolates comprised four PFGE patterns: one pattern was found in one beef and six dairy cattle, a second PFGE pattern was found in four beef animals, and the two _E. coli_ O157:NM isolates each had distinct patterns. The PFGE patterns of bovine isolates did not match patterns of deer isolates.

## DISCUSSION

The clinical response of inoculated deer is consistent with the subclinical fecal shedding of _E. coli_ O157:H7 by naturally or experimentally exposed domestic cattle and sheep (1, 2, 4, 8, 13, 14). Although two inoculated deer had mild nonhemorrhagic diarrhea during a portion of the trial, it is unknown if the loose feces was caused by _E. coli_ O157:H7 or by other variables. The lack of attaching and effacing lesions or adherent bacteria in any deer and the subclinical shedding by five other deer suggest that _E. coli_ O157:H7 is mildly pathogenic or nonpathogenic in deer of this age.

Patterns of fecal shedding of _E. coli_ O157:H7 and necropsy findings among inoculated deer were quite similar to those of inoculated cattle and sheep. Although individual variation has been observed, inoculated adult cattle, steers, calves, and sheep transiently shed high numbers of _E. coli_ O157:H7 bacteria shortly after inoculation, with fecal shedding decreasing dramatically by 2 weeks and often becoming intermittently detectable thereafter (1, 2, 4, 13, 14). Similarities of deer fecal shedding patterns to those of domestic ruminants suggest that shedding by deer may be transient, as it is in cattle and sheep. However, additional studies are necessary to evaluate persistence and long-term shedding of _E. coli_ O157:H7 in deer.

Necropsy results suggest that _E. coli_ O157:H7 may be found in contents at nearly all levels of the GI tract in deer recently exposed to the bacterium, but the large intestine may be the site where the bacteria persist for longer periods. These sites are similar, but not identical, to those of inoculated calves in which the rumen and colon were the primary sites of long-term localization of _E. coli_ O157:H7 (2). The presence of higher populations of _E. coli_ O157:H7 bacteria in GI contents than in corresponding mucosa and the lack of adherent _E. coli_ O157:H7 suggest that bacterial localization and proliferation in deer occurred within the contents of the GI tract in the absence of mucosal colonization as hypothesized for calves (2).

The failure to detect antibodies against _E. coli_ O157:H7 in exposed deer most likely was due to minimal interaction between the bacteria and the host as evidenced by the lack of adherent bacteria or mucosal lesions in GI sections of the animals. Antibody responses to _E. coli_ O157:H7 lipopolysaccharide were prompt and sustained in cattle inoculated with 10^10 CFU of _E. coli_ O157:H7, but inoculation of steers with 10^7 CFU resulted in little or no change in preinoculation titers (4). Thus, antibodies against _E. coli_ O157:H7 may have been detectable if deer had received a greater inoculum or if ELISA

![Fig. 3](http://aem.asm.org/DownloadedFrom/3371377)

**FIG. 3.** PFGE of _XbaI_ digests of enterohemorrhagic _E. coli_ O157 isolates from cattle and free-ranging white-tailed deer. Isolates are _E. coli_ O157:H7 except where noted. Lanes: a, j, and t, DNA pulse marker; b, cattle 547; c, cattle 541; d, cattle 57; e, cattle 197; f, cattle 472; g, cattle 117; h, cattle 302; i, cattle 128; l, cattle 538; m, cattle 317; n, cattle 249; o, cattle 486 ( _E. coli_ O157:NM); p, cattle 186 ( _E. coli_ O157:NM); q*, deer 602; r*, deer 594; s*, deer 569.
was performed using highly purified lipopolysaccharide, rather than whole cells.

Five strains were used in the inoculum because E. coli O157:H7 strains may differ in their virulence attributes and in their ability to colonize an animal species (2). Eight E. coli O157:H7 isolates obtained from two deer at completion of the contact transmission trial were genetically indistinguishable and matched the inoculum strain from venison jerky associated with human infections (12). Although evaluation of isolates from only two animals provides limited information, findings suggest that E. coli O157:H7 from a cervine source may persist longer and/or spread more readily in deer than other strains derived from cattle or humans. However, additional inoculation trials using several E. coli O157:H7 isolates from deer, domestic ruminants, humans, and other sources are necessary to confirm this preliminary observation.

Our field study represents the first investigation of the prevalence of E. coli O157:H7 in sympatric free-ranging deer and domestic cattle. Previous surveys of deer for E. coli O157:H7 involved environmental samples of deer feces, and the actual number of deer sampled was unknown (17, 19). The results of the current study suggest that the prevalence of E. coli O157:H7 is low among wild deer in the southeastern United States; however, there may be scattered locations where deer sporadically harbor the bacterium.

The role of wild deer in the epidemiology of E. coli O157:H7 in cattle is undetermined. Genetically indistinguishable isolates of E. coli O157:H7 were obtained from deer and cattle feces on the same Texas ranch, and deer have been suggested as reservoirs or disseminators of the bacterium (17, 19). However, the design of surveys to date has precluded determining the direction of the spread of this organism between wild and domestic animals. Deer isolates from the current study differed genetically from cattle isolates of this study as well as from 40 banked isolates of E. coli O157:H7 at the University of Georgia’s Center for Food Safety and Quality Enhancement (T. Zhao and M. P. Doyle, unpublished data). The apparent lack of maintenance of E. coli O157:H7 in the wild deer from one year to the next and the disparity between the strains found in deer and cattle in the current study suggest that deer were not serving as a reservoir of E. coli O157:H7 for cattle at this site.

The factors that lead to E. coli O157:H7 carriage by deer at specific locations are unknown. The presence of cattle, the primary reservoir, may be a necessary or contributing factor. A single strain of E. coli O157:H7 disseminated through cattle via contaminated animal drinking water, and the strain was also isolated from feed, flies, and a wild pigeon on a Wisconsin farm (20), which suggests that a similar scenario could result in E. coli O157:H7 exposure of wild deer frequenting cattle farms. Cattle were present at the site where positive deer were found in the current study; however, wild deer and domestic cattle shared range at other sites in our study, and E. coli O157:H7 was not recovered from those deer populations. Cattle at those sites were not surveyed for E. coli O157:H7.

E. coli O157:H7 was not detectable in uncooked meat from the positive deer identified in this study. However, because E. coli O157:H7 has been detected in deer feces, the bacteria have been recovered from multiple levels of the GI tract of inoculated deer, and contaminated meat from deer has been confirmed as the source of human infections, deer carcasses and venison should be handled with the same precautions recommended for beef, pork, and poultry.

ACKNOWLEDGMENTS

We are grateful to W. E. Keene for providing the venison jerky from which E. coli O157:H7 strain E0139 was isolated and to E. A. Dean-Nystrom for providing a positive control tissue sample for immunohistochemical studies. We appreciate the assistance of Gordon Luke and Melissa Finke and personnel of the Southeastern Cooperative Wildlife Disease Study and the Georgia Department of Natural Resources in sample collection. Ping Zhao of the University of Georgia Center for Food Safety and Quality Enhancement (CFSQE) performed microbiological work and genetic subtyping.

These studies were supported by UGA’s Veterinary Medical Experiment Station and CFSQE. Sponsorship also was provided by the fish and wildlife agencies of Alabama, Florida, Georgia, Kansas, Kentucky, Louisiana, Maryland, Mississippi, Missouri, North Carolina, Puerto Rico, South Carolina, Tennessee, Virginia, and West Virginia. Funds were provided by the Federal Aid to Wildlife Restoration Act (50 Stat. 917) and through grant agreement 1445-GT09-906-006, Biological Resources Division, U.S. Geological Service, U.S. Department of the Interior. Support also was provided through cooperative agreement 97-9613-0032 CA, Veterinary Services, Animal and Plant Health Inspection Service, U.S. Department of Agriculture.

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