Studies were conducted to evaluate fecal shedding of Escherichia coli O157:H7 in a small group of inoculated deer, determine the prevalence of the bacterium in free-ranging white-tailed deer, and elucidate relationships between E. coli O157:H7 in wild deer and domestic cattle at the same site. Six young, white-tailed deer were orally administered 10⁸ CFU of E. coli O157:H7. Inoculated deer were shedding E. coli O157:H7 by 1 day postinoculation (DPI) and continued to shed decreasing numbers of the bacteria throughout the 26-day trial. Horizontal transmission to an un inoculated deer was demonstrated. Although 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.
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 (Verocytotoxin)-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 10⁸ CFU of nalidixic acid-resistant *E. coli* O157:H7. Two deer received a similar inoculum of nalidixic acid-resistant, nonontoxicogenic *E. coli*, and one received no inoculum. To assess contact transmission, the uninoculated deer was cohoosed 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 un inoculated contact transmission animal 14 days postcontact (DPC). One deer inoculated with nonontoxicogenic *E. coli* was necropsied at 7 DPI, and the other was necropsied at 26 DPI.

**Bacterial inoculum.** Deer received 10⁶ CFU of a five-strain mixture of entero-hemorrhagic *E. coli* O157:H7 consisting of one venison isolate (E0139), three cattle isolates (E009, E018, 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 nonontoxicogenic strain were selected for nalidixic acid resistance. Nonontoxicogenic strains did not produce Shiga toxins and were negative by 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 10⁴ CFU/ml, and 0.8 ml of each of the five strains of *E. coli* O157:H7 was mixed in 16 ml of 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.

**Necropsy of deer.** Animals were euthanized with intravenous sodium pentobarbital, and a complete necropsy was performed. Two deer that died spontaneously were necropsied shortly after death. At necropsy, all deer were gastrointestinally 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 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 (>5 log₁₀ CFU/g) of *E. coli* O157:H7 were cultured were selected and treated in an automated immunohistostaining 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 un inoculated deer. Tissues were deparaffinized in xylene for 15 min, rehydrated through graded alcohols, rinsed in diluted citrate buffer (Antigen Retrieval Citra 10X 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 immunohistostaining 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 peroxidase 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 (Flotexx; Lerner Laboratories, Pittsburgh, Pa.), and examined by light microscopy.

**Serologic testing of experimental deer.** ELISA was performed in 96-well polystyrene immunomass-immunounassay-immunounassay plates (GIBCO, Grand Island, N.Y.). Each well was coated with 10⁶ 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 diluted-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 phosphatase (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 and 100 μl of p-nitrophenylphosphate in 1 M 2-amino-2-methyl-1-propanol (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

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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 enumerate E. coli O157:H7 in feces.

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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 placed in Cary-Blair medium as described above and then plated and incubated on sorbitol-MacConkey agar plates (Unipath, Oxoid Division, Ogdenburg, N.Y.) containing 0.1% 4-methylumbelliferyl-β-D-glucuronide (Sigma Chemical Co.). For enrichment, samples were placed in TSB containing novobiocin and incubated with agitation. 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 fecal 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 domestic cattle 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 antiseraum 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 (GAAGAGTCGGTGATTAC G), Stx1b (AGCGAGCTGAGTTAATAA), StxBa (TTAAACCCACCAAC GCAGT), and StxBc (GCTCTGGATGCATCTGGT). 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.

Genetic fingerprints of eight E. coli O157:H7 isolates obtained at necropsy on day 26 from the GI tracts of the two deer in the contact transmission trial were determined, as were fingerprints of the 3 deer isolates and 13 cattle isolates of E. coli O157 obtained in field studies. Genomic subtyping of E. coli O157 isolates was done by PFGE of XbaI restriction endonuclease digests of genomic DNA as previously described (15). PFGE patterns of the isolates from the experimental animals were compared with those of the inoculum strains, and patterns of the field isolates from wild deer were compared with those of cattle.

**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 non-typhogenic 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 nontyphogenic E. coli did not exhibit similar symptoms.

Several deer were shedding E. coli O157:H7 bacteria until 12 and 17 DPI, respectively, and intermittently shedding low numbers of E. coli O157:H7 bacteria until 12 and 17 DPI, respectively, and at 14 DPI. Additionally, one deer (deer 10) that received nontyphogenic 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 en by enrichment culture through 25 DPI (Fig. 1).

In the contact transmission trial, shedding by the two 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 DPC, the uninoculated 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 DPC. After introduction of the uninoculated 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 (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 the abomasum, duodenum (detectable only using enrichment culture), and spiral colon of the uninoculated deer (Table 1). 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 immunohistochromatically 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 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...
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 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 also was 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.

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