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Original Research

An Outbreak of *Yersinia enterocolitica* in a Captive Colony of African Green Monkeys (*Chlorocebus aethiops sabaeus*) in the Caribbean

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Yersinia enterocolitica **is a zoonotic gram-negative pathogen that causes mesenteric lymphadenitis, terminal ileitis, acute gastroenteritis, and septicemia in domestic animals and primates. In 2012, 46 captive African green monkeys (*Chlorocebus aethiops sabaeus*) died during an outbreak of acutely fatal enteric disease over a period of 1 mo on the island of St Kitts. The affected monkeys presented with a history of mucohemorrhagic diarrhea, marked dehydration, and depression. Fifteen bacterial isolates were recovered from the spleen, liver, and lungs of affected monkeys. All isolates were identified as *Y. enterocolitica* by biochemical analysis and sequence comparison of the 16S rRNA gene. Phenotypic and genotypic analysis of the recovered isolates revealed homogeneity among the recovered bacteria, and all isolates gave a random amplified polymorphic DNA pattern resembling that given by genotype D under serotypes O:7,8. This outbreak represents the first isolation and characterization of *Y. enterocolitica* as the causative agent of fatal enteric disease in primates in the Caribbean.**

Abbreviations: RAPD, random amplified polymorphic DNA; rep-PCR, repetitive-sequence–mediated PCR.

Members of the genus *Yersinia* are well-recognized human and animal pathogens. The plague, or black death, caused by *Y. pestis*, is recognized as one of the most devastating bacterial diseases in the history of mankind. The bacterium was responsible for millions of human mortalities during multiple pandemics.[37] *Y. pestis* is a highly pathogenic clone that evolved from an ancestral *Y. pseudotuberculosis* strain 1500 to 20,000 y ago.[1] Conversely, yersiniosis (caused by *Y. enterocolitica* and less frequently by *Y. pseudotuberculosis*) is typically a self-limiting gastrointestinal disease of global concern, affecting human and animal populations.[16,28]

Y. enterocolitica is the causative agent of mesenteric lymphadenitis, terminal ileitis, acute gastroenteritis, and septicemia in domestic animals, nonhuman primates, and humans. The bacterium has a very wide host range and has been detected in more than 110 species of animals worldwide, including mammals, birds, and reptiles.[3,5,22,33] Infection with pathogenic strains of *Y. enterocolitica* occurs in all age groups, but clinical illness is more reported frequently in children and young adults, with asymptomatic infection being common in adults.[24] Latent infection by *Y. enterocolitica* occurs in free-living wild rodents, which excrete bacteria in their feces.[8,19] Contaminated food and water are common sources for the introduction of pathogens.[14]

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Y. enterocolita presents high antigenic variability. There are approximately 34 O antigen and 20 H antigen serogroups.[16] In primates, serotypes O3, O5/27, and O9 have relatively low pathogenicity, mainly causing diarrhea, but serotype O8 is highly pathogenic and may cause septicemia.[15,29] Nonhuman primates appear to be quite susceptible to infection with *Y. enterocolitica*, and many fatal cases of yersiniosis have been reported worldwide.[3,5,20,29,35]

Here we describe the first reported case of *Y. enterocolitica* causing acute morbidity and mortality in captive African green monkeys (*Chlorocebus aethiops sabaeus*) in the Caribbean.

Materials and Methods

Case history. During May through June 2012, an outbreak of acutely fatal enteric disease in an outdoor captive colony of African green monkeys occurred on the island of St Kitts, West Indies. Clinically affected monkeys had hemorrhagic or mucohemorrhagic diarrhea and were dehydrated. A total of 46 monkeys died during the outbreak. The animals were part of a large breeding population, approximately 2000 animals, maintained by the Behavioural Science Foundation, St Kitts. No consistent changes in husbandry were performed before the outbreak. In 4 cages, an adjustment of animal numbers and sex ratios was done approximately 3 d before any sick or dead animals were found. These cages had the first animals that showed clinical signs or found dead. Water to the animals is filtered and chlorinated.

All animals in this facility live in large social groups provided with environmental enrichment. Enclosure sizes vary, and

breeding groups range from 5 to 18 animals. Breeding troops are usually one male with multiple female monkeys. Infants are weaned between 6 to 9 mo of age, when they leave their birth cage and are moved to a nursery enclosure. Juveniles move from the nursery at approximately 12 to 18 mo of age and go to juvenile group enclosures of 10 to 18 animals, depending on the size of the enclosure. The majority of juvenile enclosures are centrally located in the facility. Aged animals are in smaller groups of 4 to 5 monkeys and are near the periphery of the yard, at the most distant location from the majority of activity. The animal housing at the facility is entirely outdoors. Regional small mammals such as mice, rats, and mongoose come into contact with the primates. Primates are fed a combination of fresh fruits and vegetables and monkey chow (Harlan, Indianapolis, IN). Food enrichment was provided in the form of oats, tree leaves, and branches and grass. The animals are dewormed every 4 to 6 mo, with alternating ivermectin and fenbendazole.

Both breeding animals and offspring have biyearly physical examinations and veterinary care. Breeding monkeys do not participate in experimental studies. Growth curves and social behavioral characteristics are documented in the offspring, which undergo routine behavioral testing, typically for 2-wk periods. Maintenance and testing of this population is approved by the Animal Care Committee of the Behavioural Science Foundation, acting under the auspices of the Canadian Council on Animal Care.[7]

Five moribund monkeys were euthanized, and routine necropsies were performed on-site or at the Ross University School of Veterinary Medicine. Samples of affected tissues were fixed in 10% buffered formalin (Fisher Chemical, Fairlawn, NJ), trimmed, routinely embedded in paraffin wax (Thermo Fisher Scientific, Waltham, MA), cut at 5 µm, and stained with hematoxylin and eosin (Thermo Fisher Scientific).

Bacterial isolation and biochemical identification. During necropsy, the spleen, lymph nodes, liver, lung, and intestine were swabbed for microbiologic examination. Briefly, samples were placed in BactiSwab (Remel, Lenexa, KS) and used to inoculate modified Thayer–Martin, chocolate, tryptic soy agar supplemented with 5% sheep blood, and MacConkey agar plates (Remel). The inoculated plates were incubated for 48 h at 37 °C. Colonies from primary isolation agar plates were replated to purify the cultures on tryptic soy agar supplemented with 5% sheep blood and incubated overnight at 37 °C. Once single colonies were observed and purity of the isolate confirmed, the pure isolates were frozen at –80 °C in PBS (Fisher Chemical) containing 20% glycerol (Thermo Fisher Scientific) for later use. All recovered bacterial isolates initially were Gram-stained and tested for cytochrome oxidase and catalase activity (BD Diagnostics, Sparks, MD). Gram-negative, rod-shaped bacteria that did not produce cytochrome oxidase were chosen for identification by using the MicroID Identification System (Remel); analytical profile indexes from the kit were used for identifying species in the Enterobacteriaceae family only.

DNA extraction. All isolates recovered were used for molecular analysis. A loop of the bacterium was suspended in 500 µL sterile PBS and underwent DNA extraction according to protocols for gram-negative bacteria (DNeasy Kit, Qiagen, Valencia, CA). Extracted DNA was stored at –20 °C for future analysis.

Molecular identification. Molecular identification of the isolates was determined by amplification and sequencing of the 16S small subunit ribosomal RNA gene. PCR amplification of the universal eubacterial 16S rRNA was performed by using published protocols and primers.[10] Amplicons were purified by using the QiaQuick PCR Cleanup Kit (Qiagen) and sequenced (Davis Sequence, Davis, CA). Sequences were compared with those stored in GenBank by using a BLASTN search of the nonredundant nucleotide database of the National Center for Biotechnology Information.

Repetitive-sequence–mediated PCR (rep-PCR). Genetic fingerprinting of each isolate was accomplished by using modifications to existing protocols.[12,13,18,38] An *Escherichia coli* (ATCC no. 25952) isolate was included in the analysis as an outlier. Genomic DNA used in analysis was obtained as described earlier. Briefly, the analysis consisted of 25-µL reactions composed of 13 µL IQ Supermix (Bio-Rad, Hercules, CA), 20 pmol (ERIC I and II) or 40 pmol (BOX, ERIC II, GTG5) primers, 10 ng DNA template, and nuclease-free water (Qiagen) to volume. Amplifications were performed on a PTC 200 gradient cycler (MJ Research, Waltham, MA) with the following temperature profiles: 1 cycle at 95 °C for 10 min; 5 cycles of 95 °C for 1 min, 40 °C for 1 min, and 72 °C for 5 min; and 35 cycles of 95 °C for 1 min, 55 °C for 1 min, and 72 °C for 5 min. Aliquots (10 µL each) of each amplification reaction were electrophoresed through a 1.5% (w/v) agarose gel (Fisher Bioreagents, Fisher Chemical) containing ethidium bromide (1 µg/mL; Fisher Bioreagents, Fisher Chemical) and visualized under UV light. Band sizes were assigned by direct comparison with concurrently run Hyperladder II DNA standards (Bioline USA, Taunton, MA). Genetic fingerprints generated by rep-PCR were analyzed by using the Quantity One 1D Analysis Software version 4.6.5 (Bio-Rad Laboratories).

Random amplified polymorphic DNA (RAPD) PCR analysis and serotyping. RAPD PCR analysis was performed to serotype the recovered isolates by using a single primer (1290, 5′ GTG GAT GCG A 3′).[31]

Antimicrobial susceptibility. The minimal inhibitory concentrations of 28 antimicrobial agents to *Yersinia enterocolitica* isolates from monkeys and a quality control (*E. coli* ATCC 25922) were tested by using the CMV2AGNF Sensititre Gram Negative Plate Format (Trek Diagnostic System, Cleveland, OH), and E-Test strips (bioMérieux SA, Marcy l'Etoile, France) according to the manufacturer's instructions and published protocols from the Clinical and Laboratory Standards Institute.[9] Briefly, *E. coli* ATCC 25922 quality control was plated on tryptic soy agar supplemented with 5% sheep blood and incubated overnight at 37 °C. Inocula were prepared by suspending bacterial colonies in PBS to a 0.5 McFarland standard. For broth microdilution, the suspension was diluted 1000-fold in Mueller–Hinton broth (Remel), and 50 µL was added to each well of the Sensititre plate containing the various antibiotics. For each plate, 3 wells contained the bacterial inoculum without an antibacterial agent (positive controls), and one well contained the bacterial inoculum with an antibacterial agent to prevent bacterial growth (negative control). Broth microdilution plates were covered with an adhesive seal provided by the manufacturer and incubated overnight at 37 °C. Bacterial growth was checked visually after removal of the adhesive seal at 24 h after inoculation. For the E-Test, sterile nontoxic swabs were immersed in MacFarland solution and were used to streak the entire agar surface 3 times, with rotation of the plate 60° each time to evenly distribute the inoculum. Once the

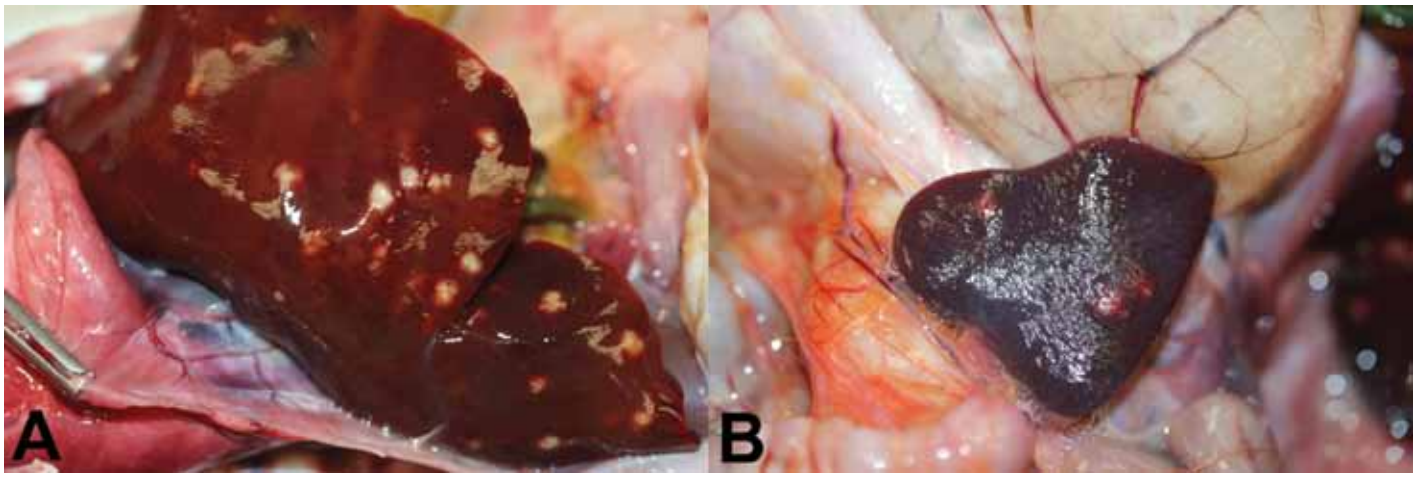

Figure 1. Gross pathologic findings in African green monkeys naturally infected with *Y. enterocolitica*. Multifocal, white or pale-yellow nodules were found throughout the (A) hepatic and (B) splenic parenchyma.

excess moisture was absorbed, E-Test gradient strips were added to the media, and the plate was incubated overnight at 37 °C. The minimal inhibitory concentration was defined as the lowest concentration exhibiting no visible growth. Each isolate was run in duplicate.

Results

Pathologic findings. Of the 5 green monkeys presented for necropsy, 3 were in poor body condition, with scant fat reserves and muscle mass. The 5 carcasses were moderately dehydrated, and there was blood-tinged watery fecal staining of the base of the tail and perineal area. Subcutaneous petechiation was present throughout the body, and mucous membranes were diffusely pale. Multifocal, variably sized (2 to 8 mm), slightly raised, off-white or pale yellow nodules were scattered throughout the hepatic and splenic parenchyma (Figure 1). On cut surface, the nodules were moderately firm and had a caseated appearance consistent with abscessation or necrosis. The stomach was usually empty and markedly dilated with gas. The mucosae of the small and large intestines were diffusely reddened. The cecum and colon of one of the monkeys contained blood-tinged mucus, and numerous thin white nematode parasites, approximately 1 cm in length, (*Trichuris* spp.) were found in the lumen of the descending colon and rectum. Mesenteric lymph nodes were moderately enlarged and slightly edematous. The rest of the internal viscera appeared grossly unremarkable.

Histologically, these monkeys had variable degrees of ulcerative, necrohemorrhagic, and suppurative enterocolitis with occasional foci of erythrophagocytosis and hemosiderosis within the enteric lamina propria; necrotizing lymphadenitis of mesenteric lymph nodes; and severe necrotizing and suppurative hepatosplenitis. Large, irregularly shaped colonies of gram-negative coccobacilli were often present within the lesions (Figure 2). Older lesions were recognized by the presence of increased numbers of lymphocytes, macrophages, and plasma cells surrounding necrotic foci.

Bacterial identification. Fifteen *Y. enterocolitica* isolates were recovered from gastrointestinal, hepatic, or splenic swabs submitted for diagnosis. The *Y. enterocolitica* isolates grew on MacConkey agar incubated at 37 °C overnight and produced small nonlactose fermenting colonies composed of gram-negative, cytochrome-oxidase–negative rods. Using a commercial biochemical kit, we identified the 15 isolates as members of the genus *Yersinia*. Molecular diagnosis confirmed that the isolates were *Y. enterocolitica*, and the closest match was *Y. enterocolitica* subsp. *paleartica* (GenBank accession no., CP002246.1).

rep-PCR and RAPD analysis of *Y. enterocolitica* isolates. The 4 different primer sets generated genetic profiles of varying complexity, with high degrees of similarity between *Yersinia* spp. isolates found in this study, all of which were significantly different from the *E. coli* outlier (Figure 3). There were no observable differences between *Yersinia* isolates, regardless of the primer set used. All isolates in the present study gave a RAPD pattern resembling that of genotype D under serotypes O:7,8.[31]

Antimicrobial susceptibility. The isolates were susceptible to tetracycline, gentamycin, chloramphenicol, amikacin, imipenem, ceftiofur, kanamycin, trimethoprim –sulfamethoxazole, ceftriaxone, ciprofloxacin, ceftazidime, piperacillin–tazobactam, aztreonam, levofloxacin, and cefepime. Isolates were resistant to sulfisoxazole, amoxicillin–clavulanic acid (2:1 ratio), ampicillin, oxacillin, amoxicillin, erythromycin, vancomycin, and clindamycin. The quality-control isolate produced results within acceptable published ranges for the antimicrobial agents.[9]

Discussion

In this case report, *Y. enterocolitica* was found as the causative agent of high mortality and bloody diarrhea in captive nonhuman primates. Yersiniosis in primates usually causes intestinal infections, with vomiting, diarrhea or constipation, weight loss, and depression.[16] Clinical disease may range from an acute, fulminant infection to a mild chronic infection.[6] Characteristic postmortem findings reported in nonhuman primates include severe enterocolitis and the presence of necrotic and suppurative foci within the liver, spleen, and mesenteric lymph nodes. Microscopic findings are characterized by the presence of multiple necrotic foci of variable size, containing cellular debris and large gram-negative colonies of coccobacilli.[6,16] The clinical, gross, and microscopic findings described in this particular outbreak are consistent with

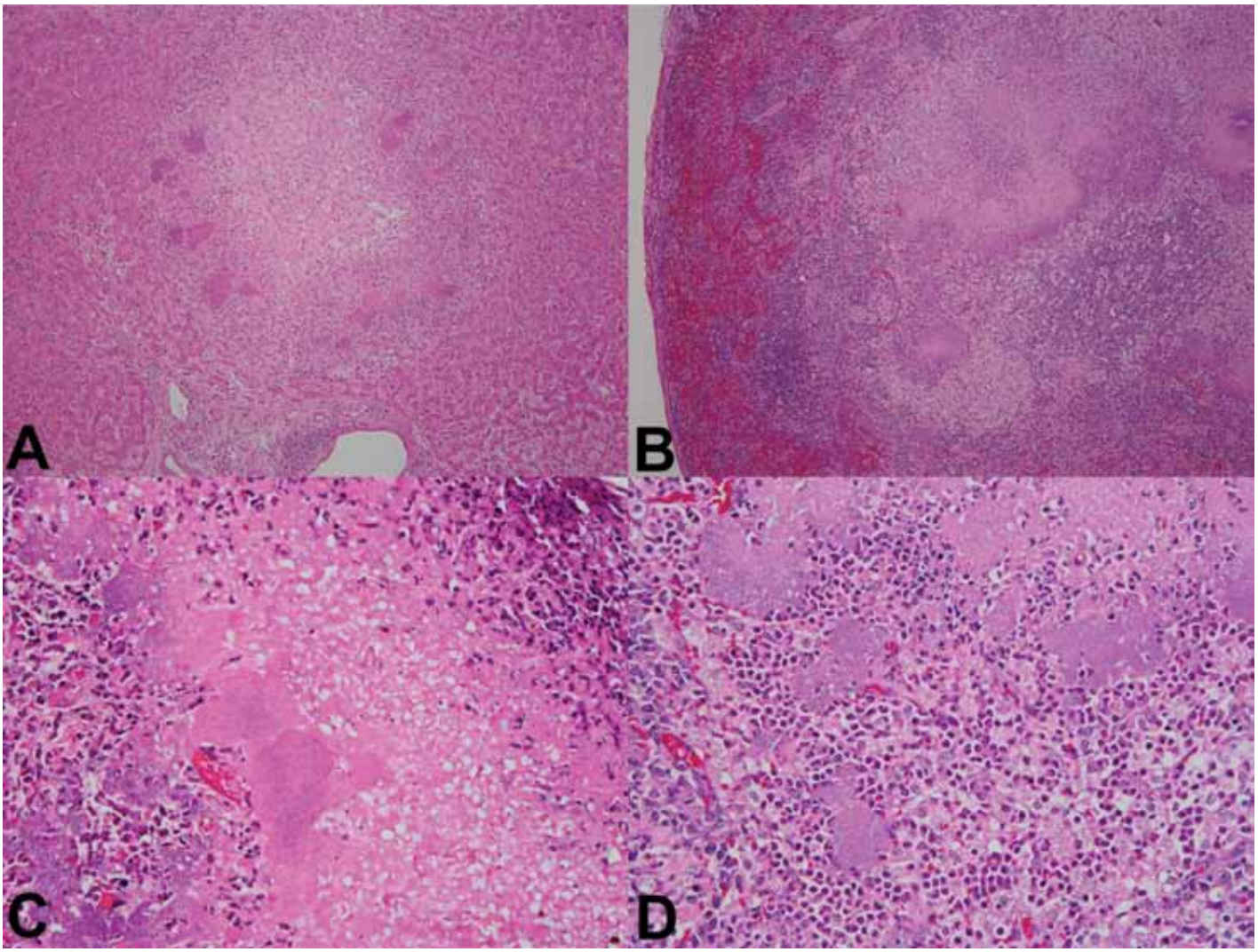

Figure 2. Histopathologic finding in liver and spleen of African green monkeys naturally infected with *Y. enterocolitica*. Multifocal necrotizing (A) hepatitis and (B) splenitis. Hematoxylin and eosin stain; magnification, 10×. Presence of large bacterial colonies within the (C) hepatic and (D) splenic parenchyma. Hematoxylin and eosin stain; magnification, 40×.

those of previously reported cases of yersiniosis in nonhuman primates.[3,5,20,29,35]

The antimicrobial susceptibility profile of the recovered isolates was similar to those obtained when testing *Y. enterocolitica* isolates from humans and animals from different locations.[32,34] Canadian isolates were susceptible to ciprofloxacin and piperacillin, and at least 98% of the strains were susceptible to trimethoprim–sulfamethoxazole, cotrimoxazole, tetracycline, chloramphenicol, cefamandole, cefotaxime, aztreonam, and 4 aminoglycosides.[32] In contrast, all strains were resistant to erythromycin, furazolidone, and clindamycin, and at least 90% of the strains were resistant to ampicillin, carbenicillin, ticarcillin, and cephalothin.[32]

In humans, enteric infections caused by *Y. enterocolitica* are usually self-limiting. However, if antibiotic therapy is indicated, the use of broad-spectrum cephalosporins, aminoglycosides, chloramphenicol, tetracyclines, and trimethoprim–sulfamethoxazole has been effective.[28] Successful treatment of humans with piperacillin–tazobactam,[26] trimethoprim–sulfamethoxazole,[25,36] ciprofloxacin,[17] and tetracycline[4,21,30] has also been reported. The isolates from our green monkeys were susceptible to these antimicrobials also, and their use might prove efficacious in the treatment of yersiniosis in nonhuman primates as well. However, pharmacokinetic, pharmacodynamics, and drug safety studies need to be performed before a treatment regimen with these drugs could be recommended for these animals. In nonhuman primates, tetracycline (20 mg/kg PO every 8 h), amikacin (5 mg/kg IM every 8 h), ceftriaxone (50 mg/kg IM daily), ciprofloxacin (10 mg/kg PO every 12 h), and gentamicin (2 to 4 mg/kg IM every 12 h) have been reported for the treatment of bacterial infections in monkeys.[2] Successful treatment of yersiniosis with tetracycline has been previously reported in animals.[16] After analysis of in vitro susceptibility, daily treatment with tetracycline (10 mg/kg IM) was recommended and applied to monkeys presenting bloody diarrhea and to those present in cages with symptomatic animals.

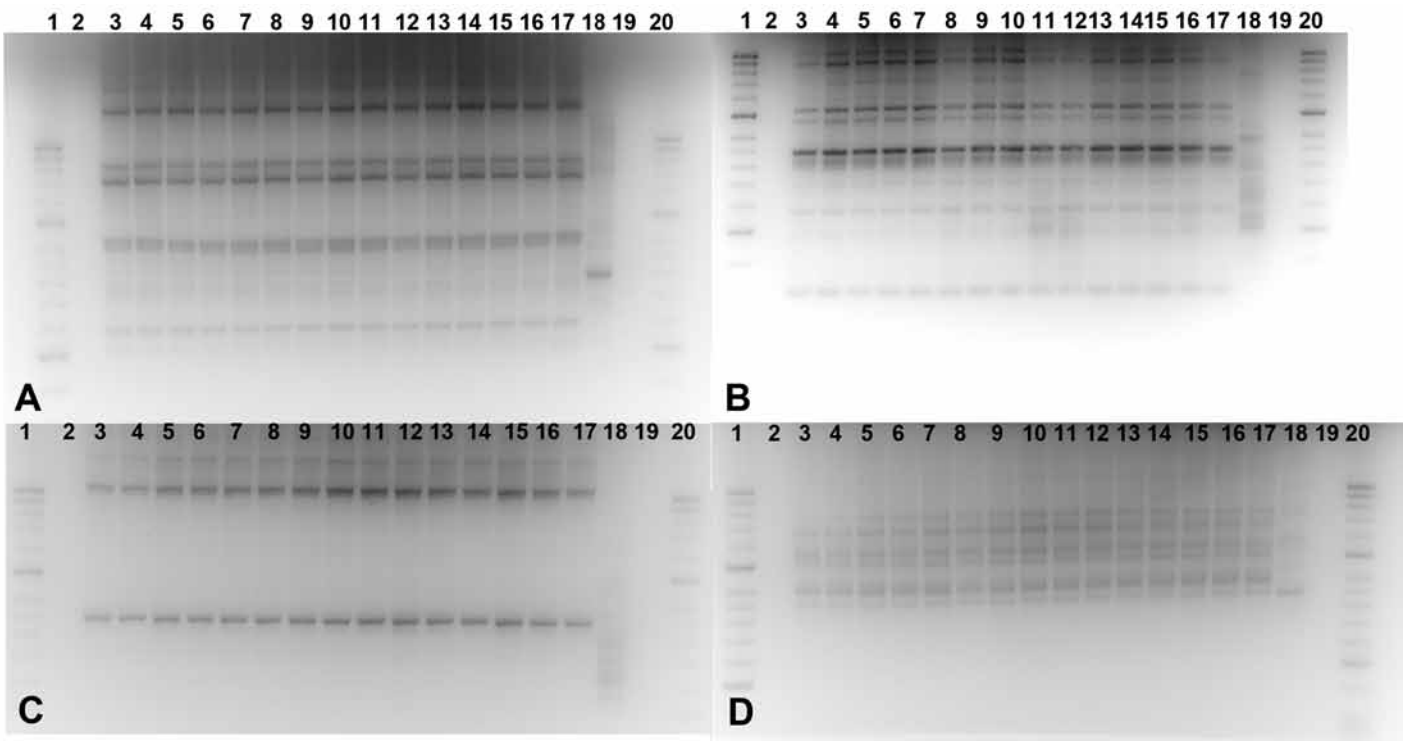

Figure 3. Electrophoretic profiles of *Y. enterocolitica* isolates recovered from an outbreak of yersiniosis in African green monkeys and reference isolates generated by using the (A) BOX, (B) ERIC II, (C) ERIC, and (D) GTG5 primer sets. Lanes 1 and 20, molecular weight ladder; lanes 2 and 19, negative controls; lanes 3 through 17, *Y. enterocolitica* isolates recovered during the outbreak; lane 18, *E. coli* reference isolate (ATCC 25922).

After the implementation of antibiotic treatment, no other mortalities were observed.

Genomic fingerprinting by rep-PCR analysis has been demonstrated as a reliable means for discriminating closely related bacterial isolates at both the species and subspecies level.[11,23,27,38] Although banding patterns for respective primer sets varied in complexity, all 4 primer sets were in agreement, suggesting that the *Y. enterocolitica* isolates recovered from this outbreak are genetically homogenous. Serotyping by RAPD PCR produced a pattern resembling that given by genotype D under serotypes O:7,8;[31] as previously discussed, serotype O8 is highly pathogenic in people and may cause septicemia and mortality events.[15,22,29] Epidemiologic studies are necessary to investigate the source of inoculum of the pathogen in this population. At present, neither the source of the bacteria nor the stressful events that may have triggered this outbreak are known. Although the water in the facility is filtered and chlorinated, high-precipitation events occurred in St Kitts several days before and during the outbreak; therefore we suspect water contamination. In addition, rats, mice, mongoose, feral cats, and wild monkeys frequently are seen close the monkey cages, so transmission from wild reservoirs is possible also. Ongoing studies will help us to identify the source of infection and to improve our understanding regarding the true prevalence of *Y. enterocolitica* in St Kitts. Moreover, the virulence of the isolate in other vertebrates, including humans, is unknown. Genotyping, serotyping, and biotyping of isolates recovered from this outbreak of yersiniosis in the Caribbean may help to elucidate the prevalence and risk of this zoonotic pathogen in local human and animal populations.

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